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# THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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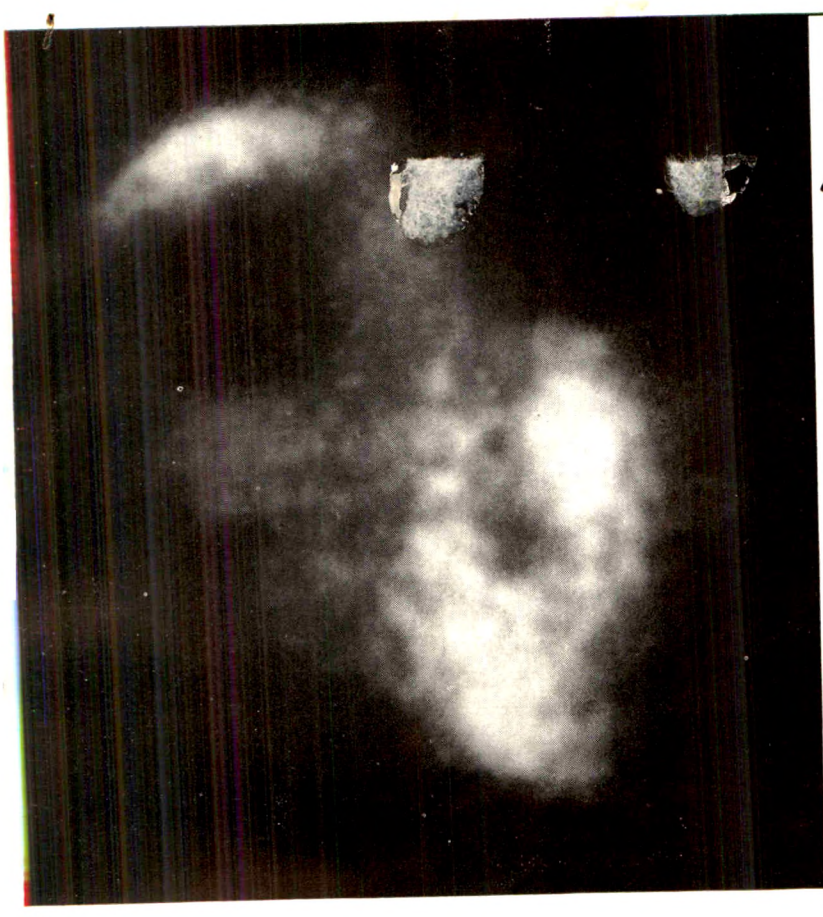
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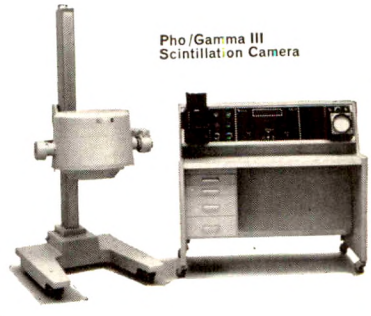
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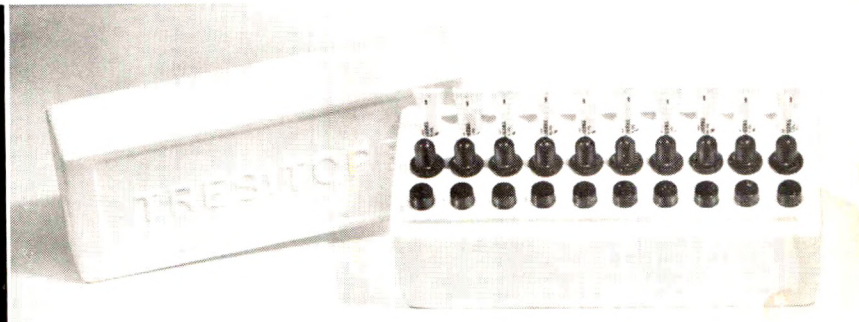
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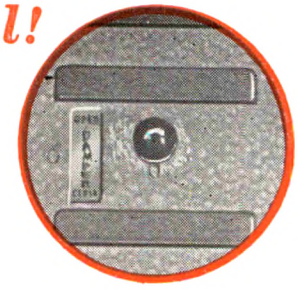
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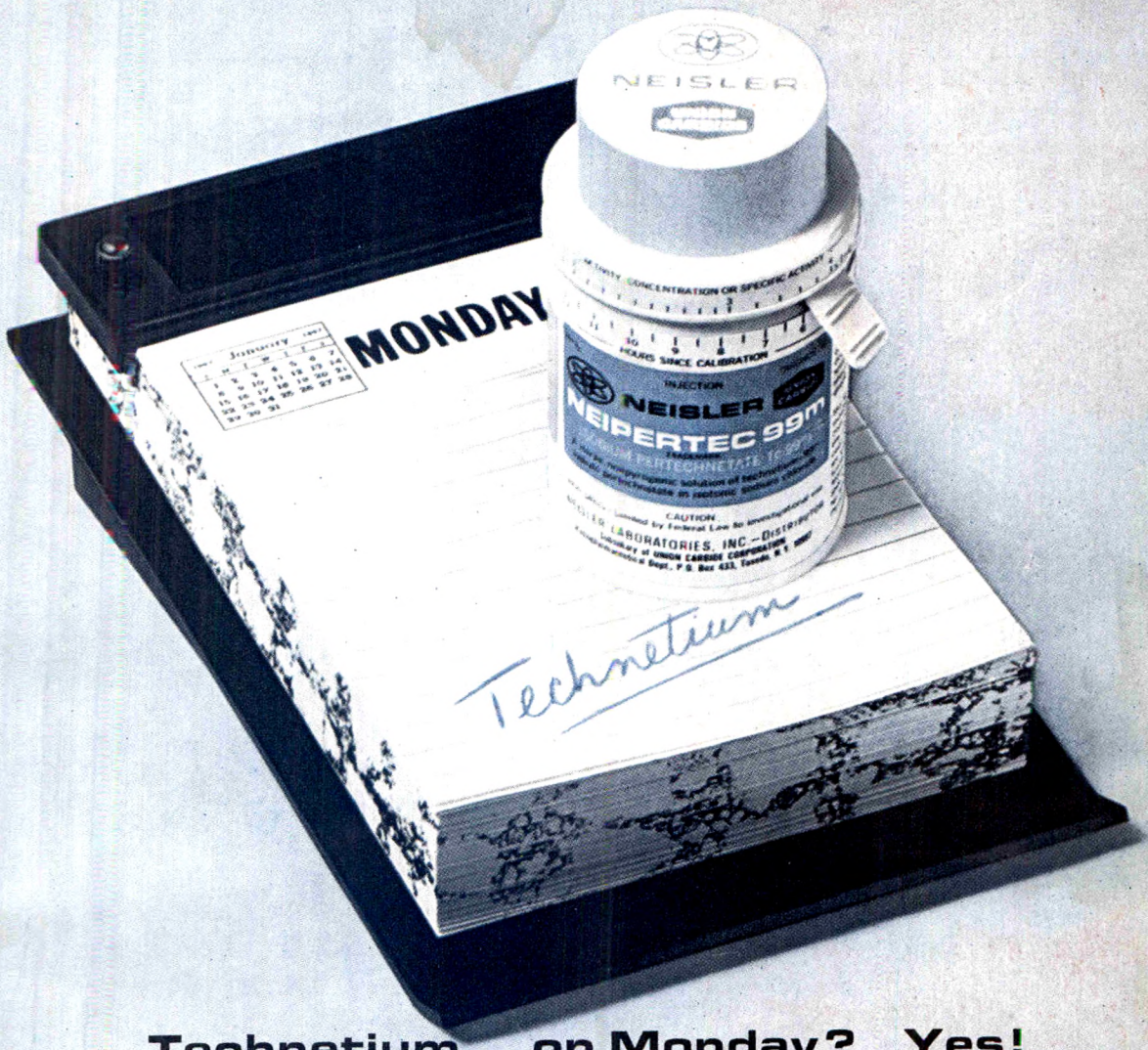
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1. J. G. McAfee, C. F. Fueger, H. S. Stern, H. N. Wagner, Jr. and T. Migita: Tc$^{99m}$ pertechnetate for brain scanning, *J. Nucl. Med.*, 5:811, 1964.

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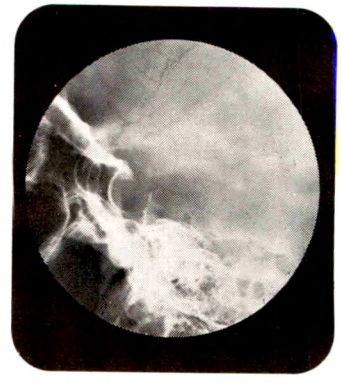
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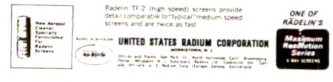



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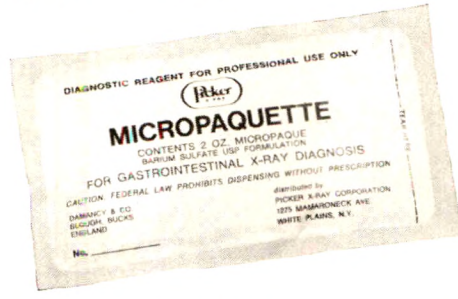
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**MICROPAQUE DISPOSABLE ENEMA KITS** contain everything you need for a single enema: a plastic enema bag, six feet of tubing with a faucet connector on one end, a rectal acorn on the other, and 10 oz. of MICROPAQUE. To use, simply fill the bag with water, knead the bag to mix the charge, and promptly administer. Discard after the examination . . . no measuring, no pouring, no clean-up, no cross-infection hazard.

# MICROPAQUE

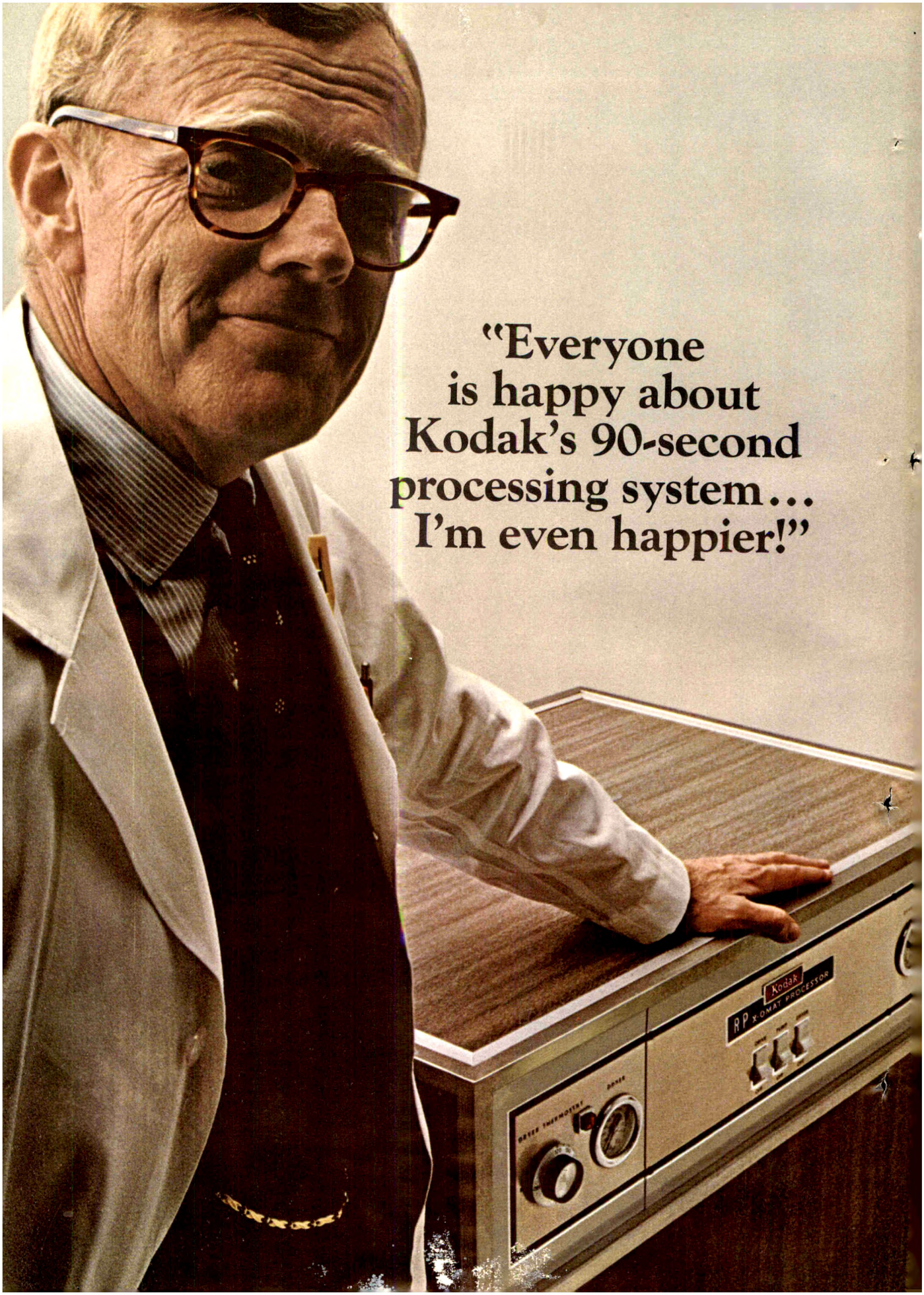
**Ask your local Picker representative**
about our special introductory offer on
Micropaquettes and Enema Kits.
Call any local Picker office, or write
Picker X-Ray Corporation, White Plains, New York.

## single-use economy

**either way,** there's no waste: you mix only as much as you need, one packet or bagful per patient. No unused surplus to squander down the drain. No possibility of contamination with dust or dirt.

**either way,** you combine the proven virtues of MICROPAQUE with new ease and accuracy in dispensing.

Picker x-ray


"Everyone
is happy about
Kodak's 90-second
processing system...
I'm even happier!"
Kodak
RP X-OMAT PROCESSOR



This is how the chief radiologist in a medical clinic feels about changing to Kodak RP X-omat processing of medical x-ray film. "It means faster, less confused service," he reports. "No more traffic snarls at the processor, more efficient use of existing facilities. Less waiting time for patients."

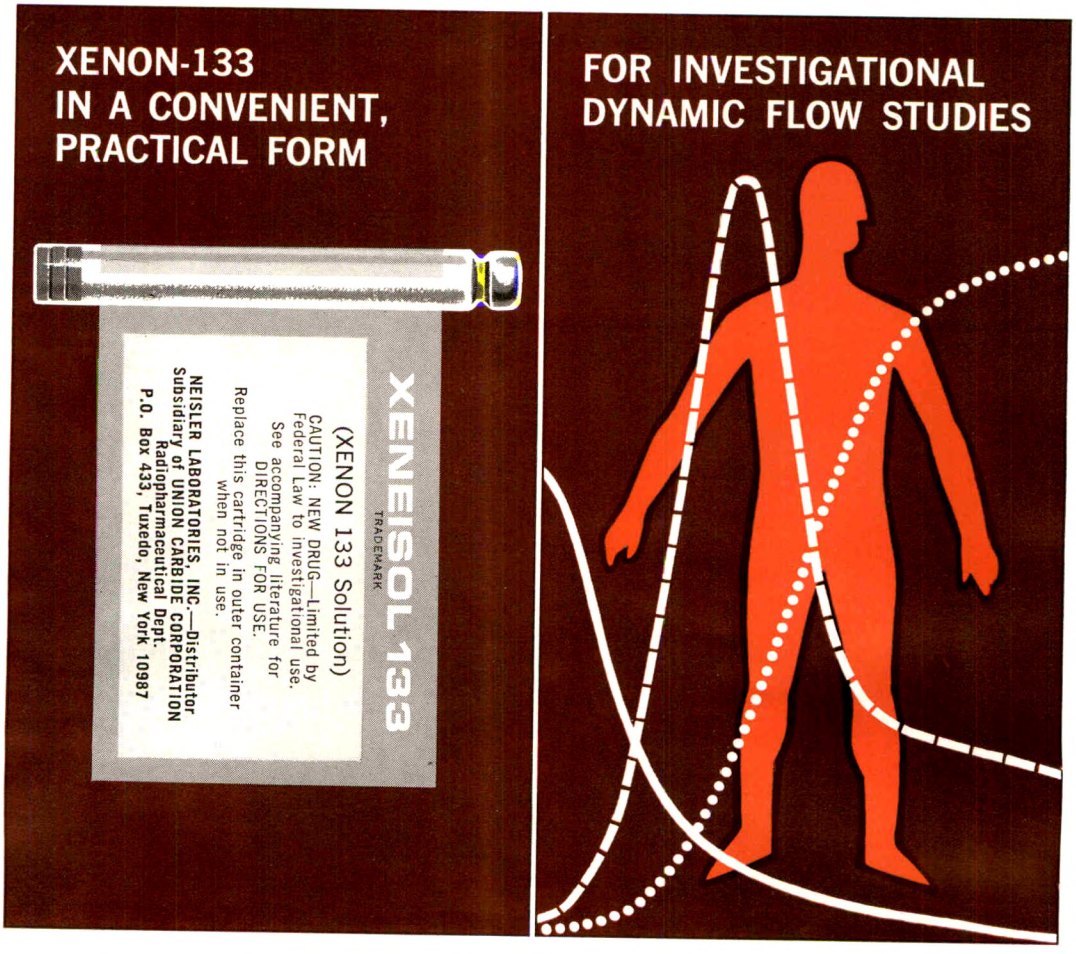
Two new films developed for rapid processing assure clean, brilliant radiographs of high diagnostic readability time after time.

Ask your Kodak Technical Sales Representative about the new Kodak RP X-omat Processor, Model M6. Or about converting most existing X-omat Processors to the 90-second cycle.

**EASTMAN KODAK COMPANY**
**Radiography Markets Division**
**Rochester, N.Y.**

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## (XENON-133 IN SODIUM CHLORIDE FOR INJECTION)

☐ **ALL OF THE GAS IN SOLUTION—**
No gas phase in the cartridge, therefore no loss of $^{133}Xe$ into a gas space. Order the amount you need...know the dose you administer.

☐ **REDUCED RADIATION RISK... CONVENIENT SHELF-LIFE—**
Biological half-life of *15 minutes or less* assures minimal radiation exposure... physical half-life of 5.27 days affords practical storage and use time.

☐ **READY FOR USE BY INJECTION—**
Supplied as sterile, pyrogen-free solution—for investigational use only.

☐ **UNIQUE COMPUTERCAP™ PACKAGING—**
Automatic computation of activity and concentration at any time after calibration.

PRECAUTIONS: Approved radiation safety precautions should be maintained at all times. Do not administer to pregnant or lactating women, or to patients under the age of 18 years, except when necessary diagnostic information cannot be obtained by other types of studies or can only be obtained at a risk greater than that of the radiation exposure caused by this drug.

SIDE EFFECTS: None reported to date; however, care should be exercised in administration.

Comprehensive literature available on request.

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**Subsidiary of UNION CARBIDE CORPORATION**
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ATOMIC ENERGY OF CANADA now offers a warranty on parts for a period of *FIVE YEARS after installation*—your assurance of the superb quality and matchless performance made possible by AECL's long experience*, specialized knowledge and established integrity.

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66.5M



# Why did this woman suffer acute abdominal pain and vomiting?

**(GAF x-ray film helped find the answer)**




*GAF would like you to have a complimentary 35mm slide of this interesting pathology, along with the final summary of this patient's history. Contact your GAF Medical X-ray Representative for a copy.*

A 74-year-old woman had acute abdominal pain, vomiting and constipation for three days prior to admission. Physical findings, which were confined to the abdomen, included muscular guarding and diffuse tenderness over the lower abdomen with some questionable rebound. There were no palpable masses; rectal examination was negative. Her white blood count was 16,000 with a normal hemoglobin. There was no previous abdominal surgery.

Initial radiographic studies of the abdomen revealed small bowel obstruction. A Cantor tube was inserted for decompression of the small bowel and barium was introduced through the tube. A small bowel study showed dilated loops of small intestine and a large radiolucent filling defect in the terminal ileum. At this time air was evident in the biliary tree. The impression, therefore, was gall stone ileus.

The radiologist's findings were confirmed in surgery.

GAF Supreme® Gafstar® x-ray film offers the most advanced emulsion on polyester base. It's the only medical x-ray film that combines maximum image quality, required contrast and speed in an x-ray film emulsion.

*A small bowel radiographic study revealed dilated loops of small intestine, a large radiolucent filling defect, and gas in the biliary tree.*

gaf **GENERAL ANILINE & FILM CORPORATION**

140 WEST 51 STREET, NEW YORK, NEW YORK 10020

# under-standable media



If the barium preparations you've used up to now have all seemed pretty much alike, then it's time to compare the contrasts with ESOPHOTRAST® (the ready-to-use esophageal cream) and BAROTRAST® (a micronized form of barium sulfate).

One of the prominent problems with most contrast media is no small matter. Their large particles have a tendency to precipitate and to flocculate out of suspension with the slightest change in pH, mucin concentration, or enzyme activity. Not so with ESOPHOTRAST and BAROTRAST, both of which are formulated to ensure an easy suspension that remains in suspension even during routine storage.

Then too, a common feature of many other barium sulfates suggests a sticky problem. The varying size of the particles reduces their adhesive properties since not enough particles come into contact with the esophageal and/or intestinal lining to provide complete coverage. With micronized ESOPHOTRAST and BAROTRAST, a maximum number of particles are available to produce a tenacious elastic coating that lasts for the time it takes to "get the picture."

ESOPHOTRAST, because of its specially formulated viscosity, provides a consistent, prolonged, residual coat that permits an excellent outline of the esophagus as well as an evaluation of cardiac size. Since it is premixed, ESOPHOTRAST is ready for instant use, which means longer visualization time and shorter preparation time. BAROTRAST, because of its natural viscosity, flows in a steady consistent column at a speed that can cut the upper G.I. series and small bowel examination time almost in half.

Both formulations are creamy, nongritty and pleasantly flavored to help your patient help you make the examination that much easier.

Considering the barium sulfates in the light of this inside information, it would seem that there is a marked contrast. And all it takes is a little understanding to get the clear picture.

# Esophotrast® · Barotrast®

BARNES-HIND BARIUM PRODUCTS

Subsidiary of Barnes-Hind Pharmaceuticals, Inc.
Sunnyvale, Calif. 94086

# Diagnostic Problem:

**When**
- **Mechanical difficulties**
  (deformity, anomaly, etc.)
- **Debilitated states**
- **Infancy**
- **Advanced age**
- **Urinary tract infection**
- **Pregnancy**
- **Extensive pelvic surgery**

**contraindicate the use of cystoscope or catheter**



# Diagnostic Remedy:

## Excretory urography with Hypaque® 50%

# 4 Diagnostic Advantages:

1. **Precisely defined urograms of kidneys, ureters, and bladder, with valuable information about their functional status.**
2. **No interference with functional activity (sometimes a problem with instrumentation).**
3. **No age limit to the procedure.**
4. **No anesthesia required.**

**Warning:** Do not use Hypaque sodium for myelography. Injection of even a small amount into the subarachnoid space may produce convulsions and result in fatality.

**Contraindications:** Advanced renal destruction associated with severe uremia, and multiple myeloma.

**Precautions:** Caution is advised in patients with hyperthyroidism, hypertension, severe cardiovascular disease, active tuberculosis, and a history of asthma or other allergy, especially to iodine. Because of the possibility of temporary suppression of urine, it is wise to allow an interval of at least 48 hours before repeating retrograde or excretory urography in patients with unilateral or bilateral reduction of normal renal function.

**Preventive Measures:** The following precautions have been recommended to help prevent reactions in intravenous urography: (1) obtain a history of personal and familial allergies (for example, bronchial asthma, hay fever, and eczema), of previous iodine studies and of sensitivity to iodine and other drugs, (2) make a preliminary sensitivity test, (3) give preliminary antihistaminic medication, and (4) have medications on hand for emergency use.

**Side Effects:** Excretory urographic agents may produce symptoms of an anaphylactic* nature in sensitive persons. Patients should be watched carefully during injection as serious reactions, including fatalities, have occurred with all commonly used mediums. Although allergic reactions generally occur quickly, occasionally they may not be manifested for 10 or even 15 minutes. Warning signs and symptoms of possible intolerance or allergy include respiratory difficulty (wheezing, dyspnea or sensation of suffocation, and tightness in the throat or chest), sneezing, itching or urticaria, nausea or vomiting, and fainting. Minor reactions, such as nausea, vomiting, excessive salivation, flushing, dizziness, urticaria, and muscular twitching may occur. Special care is advisable in persons with a history of bronchial asthma or other allergic manifestations of sensitivity, especially to iodine. Infrequently "iodism" (Salivary gland swelling) appears two days after exposure and subsides by the sixth day. In euthyroid patients following excretory urography with Hypaque, protein-bound iodine is elevated and the thyroid $I^{131}$ uptake is lowered, returning to normal after the 3rd and 4th day respectively.

**Supplied:** In sterile aqueous solution, Ampuls of 30 cc. (with 1 cc. test ampuls), boxes of 1, 10 and 25. Also, rubber stoppered vials of 20 cc. and 30 cc., boxes of 1, 10 and 25.

*Physicians should study the package insert for information on preventive measures and management of untoward reactions *before* administering Hypaque (diatrizoate sodium).

## Urograms with excellent anatomical detail

# Hypaque® 50%
(sodium)

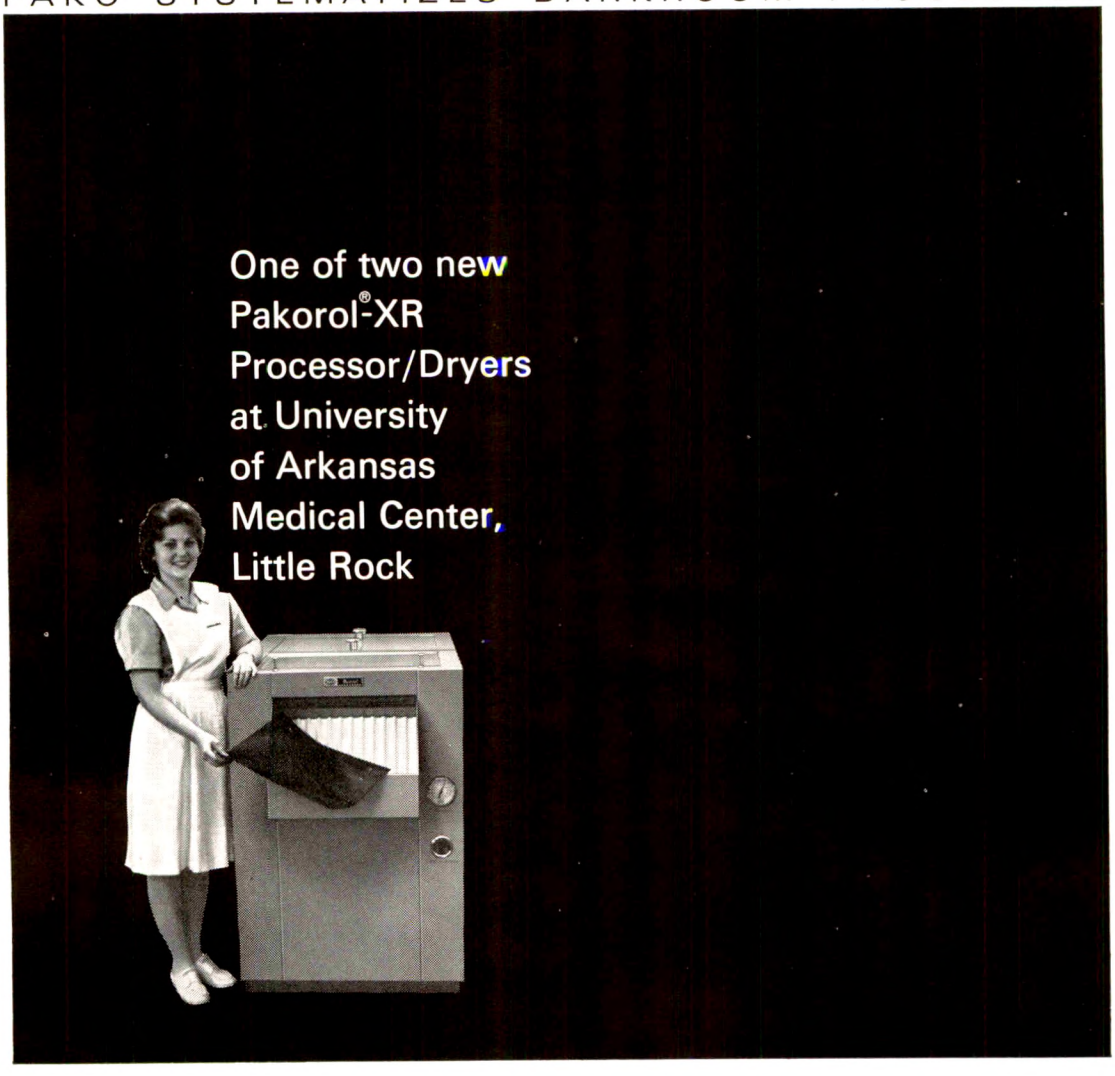
*Write for illustrated booklet containing complete information*

brand of **sodium diatrizoate**

Winthrop **PIONEERS IN PRODUCTS FOR RADIOLOGIC DIAGNOSIS**

WINTHROP LABORATORIES,
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PAKO SYSTEMATIZES DARKROOM PROCESSES




## *University Medical Center needed x-ray processors that could change with their needs*

The University of Arkansas Medical Center in Little Rock, recently installed two Pakorol-XR's. These units not only serve their needs now, but can be modified later to faster processing with new films and new chemistry.

Normal Pakorol-XR processing time is 3½-4 minutes, dry-to-dry. They can be easily converted to 7½ minute or high speed (RP) processing. Their proven roller-transport and automatic control of variables result in consistent, high quality processing.

Pakorol-XR's process a variety of films: emergency, surgery, arteriography, orthography, mammography, fluorography and ciné. Contact your Pako X-Ray Distributor for information. Or write Pako Corporation, 6300 Olson Memorial Highway, Minneapolis, Minnesota 55440.

**PAKO CORPORATION** PAKO®

PS Processing Systems for the Photographic, Graphic Arts, Motion Picture, Medical and Industrial X-Ray Industries




# Litton's Profexray Division has just introduced a new film processor with revolutionary advantage that sells at half price.



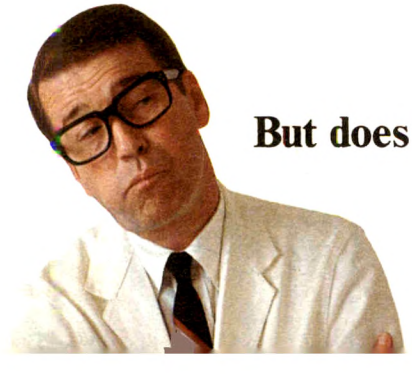












But does it work?

We're staking our reputation on this ne: A new, automatic film proc-ssor that goes for about half the rice of any other 90-second proc-ssor. And if you later decide you refer the 3½-minute cycle, you're ot stuck. We'll convert our proc-ssor for you in about an hour. At o charge. And if you change your ıind again, we'll convert it back. ıt no charge.

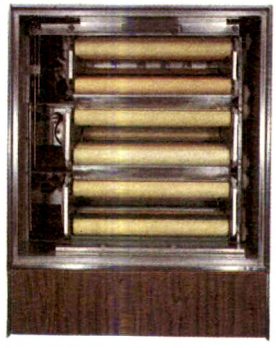
Considering the price, you might ıink we cut a few corners on our ew automatic processor.

Not so.



It's more rugged than *any* other ou can buy. Notice the gear train n the unit above. The gears are al-ıost three times the size of others. Construction is simpler, heavier, far more trouble-free. And although it's new, this processor isn't untried.

For the last 18 months Profexray has been field-testing this unit in hospitals and laboratories throughout the country. Under the toughest possible conditions, we've found several interesting facts: Our servicemen are in love with the unit because everything that might need adjustment is totally accessible from the top (see picture below).



There's no noticeable difference in operation if the unit isn't cleaned daily (chemical replenishment is a positive, cycled stream, so there are no drip-drip-drip mechanisms to clog).

The Profexray automatic processor uses about a third as much water as other units, with substantial savings in electric heating costs. It's earned — already — a remarkable service record of continuous, trouble-free operation.

In short, we know it works.

But we don't expect you to take our word for that. Instead, we invite you to give the Profexray automatic processor a 60-day trial under an unconditional money-back guarantee. It'll be the hardest-working piece of equipment in your lab.

The Profexray Division of Litton Industries guarantees it. Your local Profexray serviceman guarantees it.

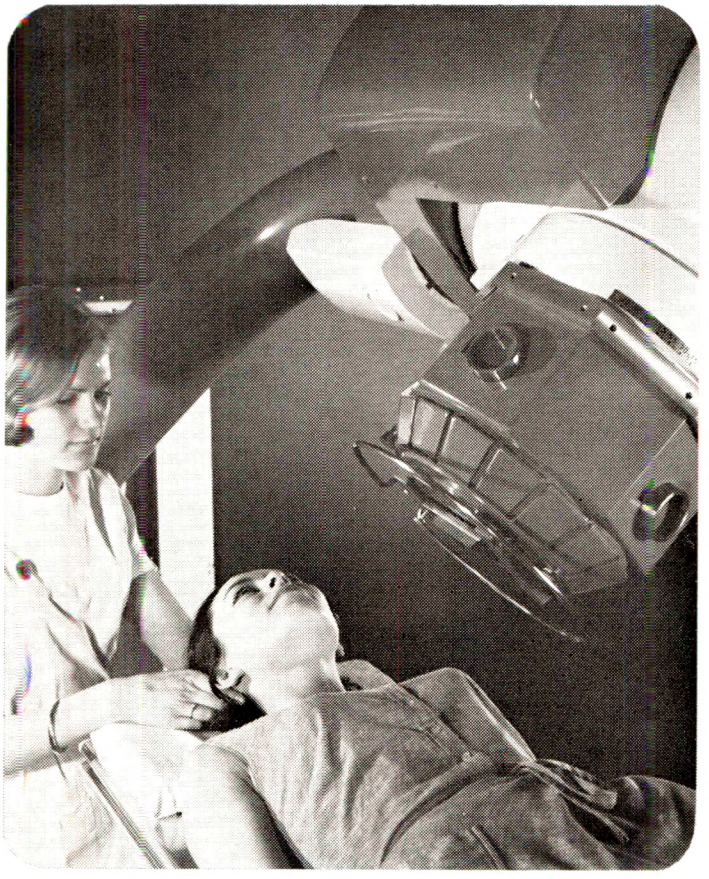
May we give you more information? Ask your local Profexray representative. Or write for a free brochure on the new Profexray automatic 90-second film processor. It's the best processor you can buy.

At any price.

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423

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P 24,407

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COBALT 60 UNIT

In assessing the applicational merits of a Cobalt 60 unit, economic considerations rank alongside those of purely therapeutic nature. The pressures bearing on a modern therapy department demand maximum utilization of all available time.
It is therefore economically desirable that the time spent on setting up should be the minimum compatible with efficiency and accuracy. Check these outstanding features built into our units to promote rapidity in setting up:

- **Direct and easy reading** of source-skin distance on patient's skin, brilliantly projected by an optical distance meter
- **Minimum time** necessary for correct positioning of patient by wide range, freely floating tabletop and motorized height adjustment
- **Quick setting,** of head angulation as result of extra high rotation speed up to 720°/min for setting-up; continuously adjustable with rotary handswitch
- **Time saving** by possibility of using two tables alternately for treatment and setting up, due to extremely simple coupling and decoupling of table from isocentre
- **Operational efficiency** and safety enhanced by logically simple lay-out of control desk.

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a choice of sixteen versions to match your individual wishes is fully described in a special brochure, obtainable from your local Philips organization or agent, as well as from:

Philips X-ray & Medical Equipment Division
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# Where exposure is important, try Cenco X-ray film

NEW!
**Ferrania N**
X-RAY FILM FOR
**AUTOMATIC PROCESSING**
NOW AVAILABLE



Important to you and your patients is the fact that Cenco-Ferrania Type N film is 4 to 6 kv faster than the medical x-ray film most widely used in the United States. You will also find Type N to be a premium-quality film that provides superior contrast, definition, and detail. Yet, it is moderately priced.

Type N is only one of several films available from your Cenco X-ray dealer. He has films for non-screen or fine tissue work, photofluorography, and for direct copies. Cenco film is available for both automatic and manual processing and is conveniently packaged for either large or small operations.

You will find your Cenco X-ray dealer uniquely attuned to your particular needs. Because meeting the requirements of radiologists is his *only* business, he gives you an extra measure of service. Why not take advantage of it?

CXR-6-321

CENCO X-RAY CO.
**Ferrania**
FINE MEDICAL AND INDUSTRIAL X-RAY PRODUCTS

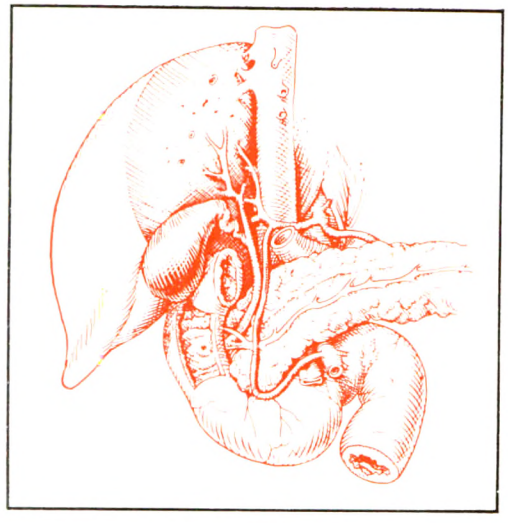
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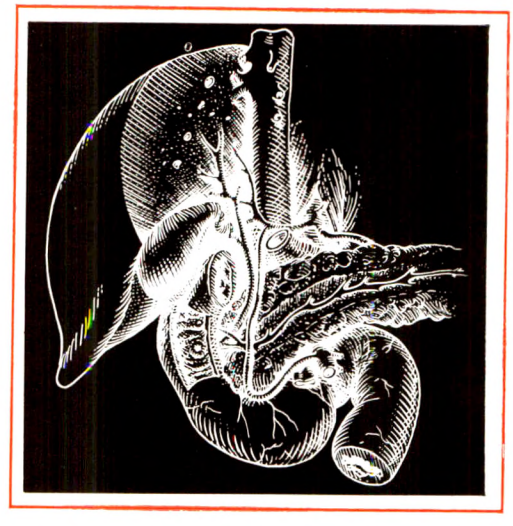
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SQUIBB

a research concept in contrast visualization

# for better definition of disease: documenting differences between oral contrast agents





no. 2 in series:

## Oragrafin®

Squibb Ipodate

## maximum concentration with better patient toleration in oral cholangiography and cholecystography

For rapid or routine studies of the gallbladder and biliary ducts, Oragrafin provides rapid, more complete absorption, for maximum concentration and excellent contrast.

**definition of maximum concentration**

**high absorption index**—"No one will dispute the statement that the diagnostic reliability of oral cholecystography depends upon the degree of absorption of the contrast medium employed."[1] Radioisotope studies with ipodate sodium show a markedly improved absorption index (approximately 70 per cent) with no increase in the toxic properties.[1]

**less bowel residue**—"Incomplete and variable absorption of a cholecystographic agent is not desirable, for the opacification of the gallbladder is then less dependent on the status of the gallbladder and more a reflection of the percentage absorption."[2] "Opaque material in the bowel was found in 46 per cent of [105 patients in] the... [iopanoic acid] group as compared with 9 per cent of those [99] of the [Oragrafin Sodium Capsules]...group."[2] In another study, 49 of 100 patients receiving iopanoic acid had residue in the gastrointestinal tract, while only 15 of 100 patients receiving Oragrafin had such residue.[3]

**excellent opacification**—"In addition to its ease of administration and safety, its principal advantages are a high yield of diagnostic films...."[4]

**definition of better toleration**

**low incidence of untoward reactions**—In contrast to findings with other cholecystographic agents, Lewitan and Garcia[5] reported in their 246 patient study: "With

ipodate calcium...there was no clinical evidence of immediate or delayed nephrotoxicity in any of the patients given it...."[5] To study possible renal toxicity, "Creatinine clearance tests were also done before and after administration of multiple doses in excess of 6 gm. in 8 cases, and showed no significant alterations."[5] However, "Multiple doses beyond 6 gm. are not recommended."[5] In their study of 120 patients, Glenn and O'Brien reported "...no reactions in this series of patients attributable to the administration of [Oragrafin]...."[4] And, McCrory reported that Oragrafin was "...used routinely in cholecystographic studies in approximately 2000 patients with excellent diagnostic films and only rare and mild reactions."[6]

**side effects compared to iopanoic acid**

side effects encountered in administration of 3 Gm. of Oragrafin Sodium Capsules and 3 Gm. iopanoic acid *

| | Oragrafin Sodium Capsules (Squibb Sodium Ipodate) | iopanoic acid |
|---|---|---|
| no. of cases | 99 | 105 |
| no. of side effects | | |
| nausea | | |
| slight | 10 | 14 |
| severe | 2 | 6 |
| vomiting | 0 | 1 |
| diarrhea | | |
| mild | 7 | 22 |
| severe | 3 | 16 |
| cramps | 5 | 15 |
| dysuria | 9 | 13 |
| total/per cent | 36 (36.3%) | 87 (82.9%) |

*Adapted from White, W. W., and Fischer, H. W.[2]

Juhl,[3] in his study of 200 patients (100 on each agent), found no significant difference between iopanoic acid and sodium ipodate in the incidence of nausea; vomiting occurred in an equal number of cases.

# definition of convenience:
# Oragrafin® Calcium Granules

## Squibb Calcium Ipodate

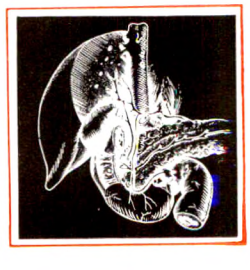
**visualization of poorly functioning gallbladder reduces the need for I.V. studies**—In a concentrating gallbladder, Oragrafin Calcium Granules (Squibb Calcium Ipodate) will show storage, concentration, delivery or stones. A patent biliary duct can generally be expected to visualize. Provided the cystic duct is patent, Oragrafin will visualize the normal gallbladder, the abnormal gallbladder containing papillomata, nonopaque stones or radiodense calculi, and gallbladders where concentrating power is diminished.

**patient convenience**—Routine cholecystography night-before procedure is easy for patients to follow; palatable granules further enhance patient acceptability. Rapid clearance of medium permits same-day administration and gallbladder and ductal films, and, if necessary, same-day re-examination; reduces need for I.V. studies.

Optimal concentration in the hepatic and biliary ducts usually occurs within 1 to 3 hours. Although the gallbladder is optimally opacified 10 hours after ingestion of the agent, diagnostically valuable information can often be obtained within 5 hours or less.

**rapid absorption permits same-day re-examination**—To determine the cause of nonopacification after routine cholecystography, most physicians require re-examination by repeating the procedure at a later date (sometimes doubling dose), or





A 2893

by administering more agent the evening of the first unsuccessful examination (again sometimes doubling dose), and repeating the study the next day. "The advantage of the calcium ipodate method is that the examination can be completed in five additional hours with a limited dose of contrast agent."[7]

**a valuable medium for peroral cholegraphy**[5]—Rapidly absorbed from the gastrointestinal tract, calcium ipodate has been reported by some investigators to be diagnostically superior to other oral cholangiographic contrast agents. With careful timing of the examination and the use of tomograms or laminograms, the frequency of good results can approximate that obtained with intravenously administered agents. According to Lewitan and Garcia,[5] the medium's relative safety makes it a valuable medium for peroral cholegraphy. Timesaving and economical Oragrafin Calcium Granules may be particularly useful in certain patients for whom I.V. radiography presents potential hazards, such as elderly patients, those with cardiovascular disease, or patients who may exhibit sensitivity to the test dose of an intravenous agent.

Unique among oral media, Oragrafin Calcium Granules permits same-day films of the gallbladder and ductal system.

dosage schedule for films of gallbladder and ductal system

| | |
|---|---|
| 8 A.M. | 2 packets of granules |
| 9 A.M. | visualization of ducts |
| 10 A.M. | optimal visualization of ducts |
| 1 P.M. | visualization of gallbladder |

**References: 1.** Sanen, F. J.: Amer. J. Roentgen. *88*:797 (Oct.) 1962. **2.** White, W. W., and Fischer, H. W.: Amer. J. Roentgen. *87*:745 (April) 1962. **3.** Juhl, J. H., *et al.*: Radiology *80*:87 (Jan.) 1963. **4.** Glenn, J. C., Jr., and O'Brien, P. S.: Southern Med. J. *56*:167 (Feb.) 1963. **5.** Lewitan, A., and Garcia, J. F.: Amer. J. Dig. Dis. *10*:219 (March) 1965. **6.** McCrory, E.: J. Tenn. Med. Ass. *58*:258 (Aug.) 1965. **7.** Crummy, A. B.: Wisconsin Med. J. *65*:84 (Feb.) 1966.

**Contraindications:** Contraindicated for persons sensitive to oral iodine compounds or for patients with combined renal and hepatic disease or severe kidney impairment. Gastrointestinal disorders, which may interfere with absorption, or liver dysfunction, which may result in inadequate biliary secretion of medium, are likely to result in unsatisfactory visualization.

**Precautions and Side Effects:** Mild and transient nausea, vomiting, or diarrhea sometimes occur; but the incidence can be reduced by using the calcium granules and restricting the dosage to 3 Gm. Transient headache, dysuria, or abdominal pains may occur.

Hypersensitivity reactions may include urticaria, serum sickness-like reactions (fever, rash, arthralgia), other skin reactions, and rarely anaphylactoid shock. They are more likely to occur in the individual with a history of allergy, asthma, hay fever, or urticaria and in the individual who is known to be hypersensitive to iodine compounds. Antihistamines and corticosteroids are used to control hypersensitivity reactions; but the occasional serious anaphylactoid reactions require the immediate use of epinephrine or phenylephrine, oxygen, and intravenous corticosteroids.

For full information see Package Insert.

**Supply:** The *calcium* salt (Oragrafin Calcium Granules) is available in single-dose foil packets providing 3 Gm. of calcium ipodate as Granules dispersed in flavored sucrose. The *sodium* salt is available in capsule form (Oragrafin Sodium Capsules) providing 0.5 Gm. sodium ipodate per capsule.

**in oral cholangiography and cholecystography**

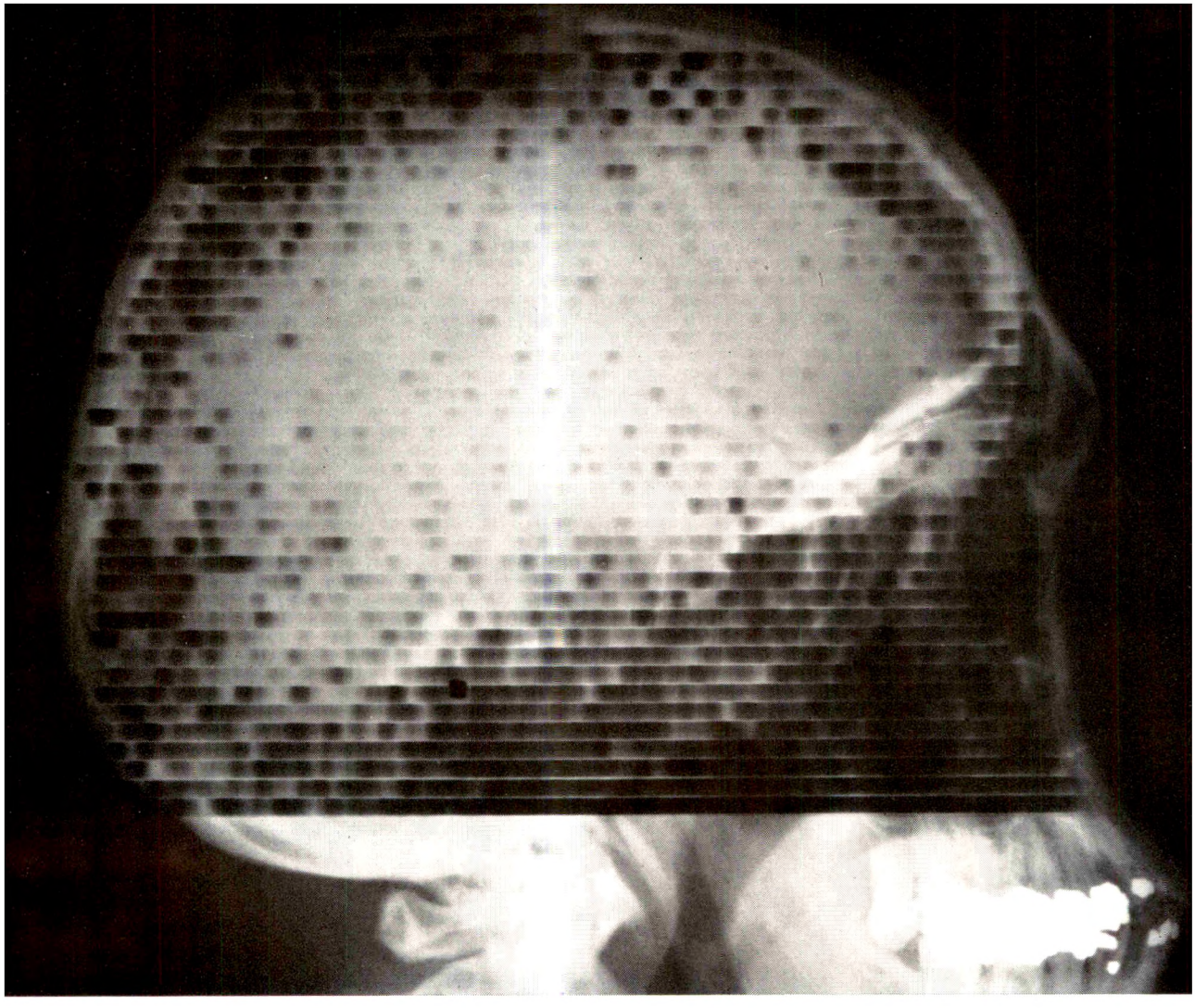
# Oragrafin®

Squibb Ipodate

**for maximum concentration with better patient toleration**

SQUIBB

'The Priceless Ingredient' in every product is the honor and integrity of its maker.



# Abbott announces Pertscan™-99m

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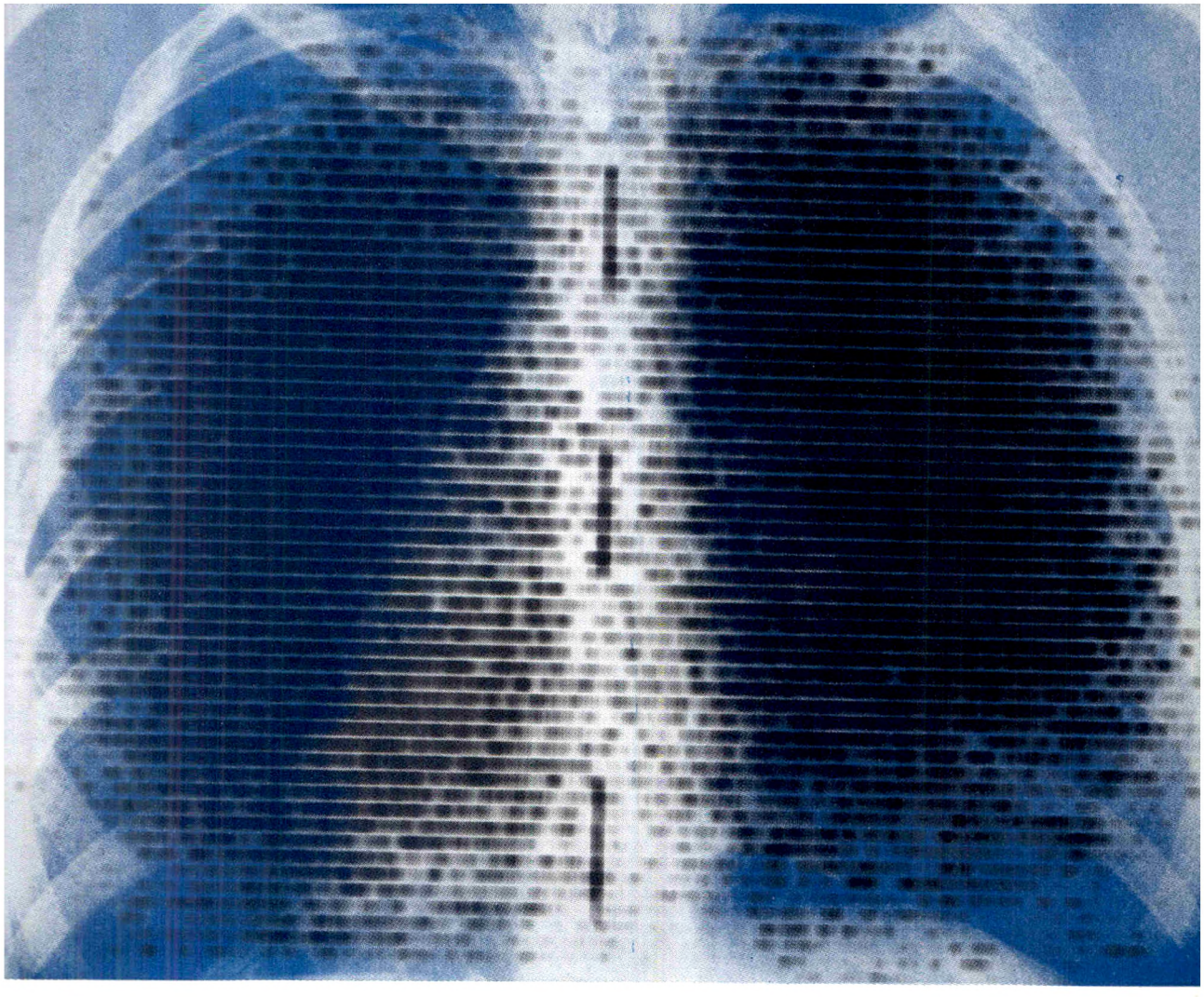
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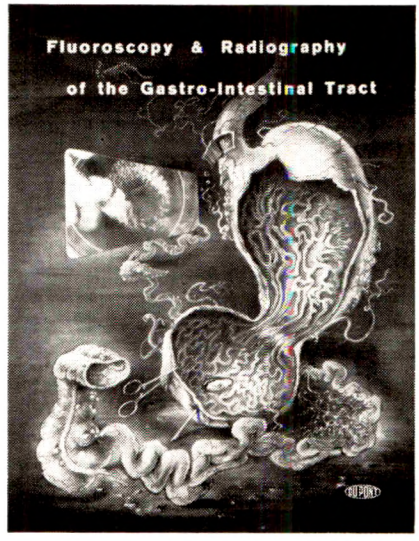
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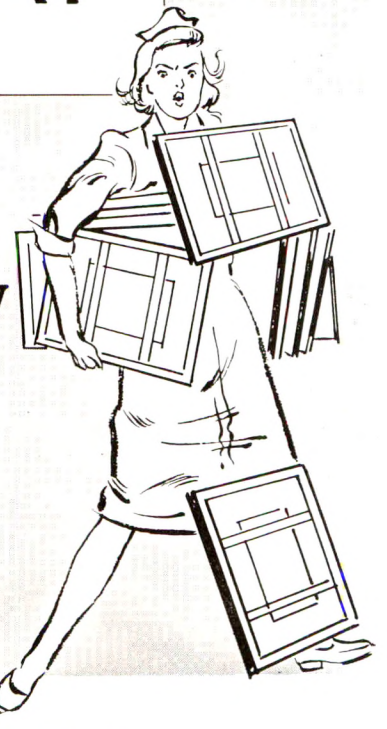
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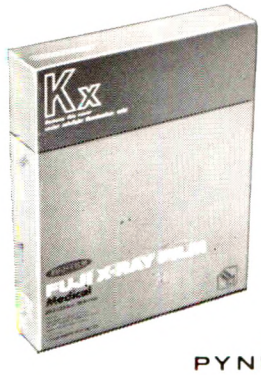
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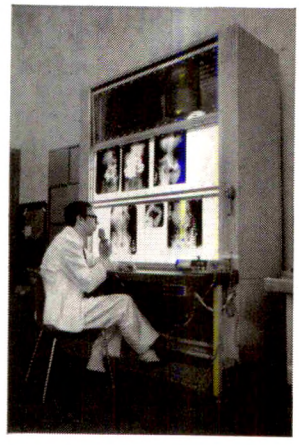
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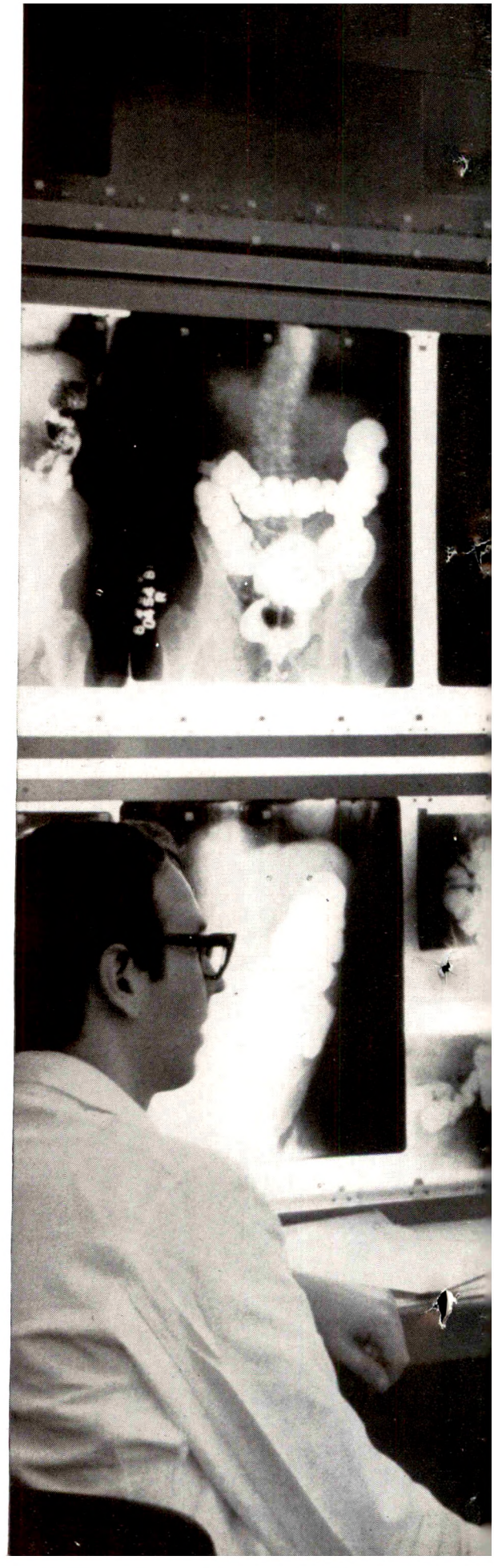
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**available with four different quantities of $Mo^{99}$**

The quantity of $Tc^{99m}$ eluted from the generator is, of course, dependent upon the quantity of $Mo^{99}$ present in the column. Technetope sterile generators contain either 50, 100, 200, or 300 millicuries of $Mo^{99}$ at the time of assay, depending on which activity has been requested. The activity obtained from subsequent elutions will depend on the time interval between elutions. (See Fig. 2.)

**Warning:** Proper radiation safety precautions should be maintained at all times. The glass column containing $Mo^{99}$ need not be removed from the lead shield at any time. The radiation field surrounding an unshielded column is quite high. Solutions of $Tc^{99m}$ withdrawn from the generator should always be adequately shielded. The early elutions from the generator are highly radioactive. For radiation protection, a lead shield for the collecting vial is included with Technetope.

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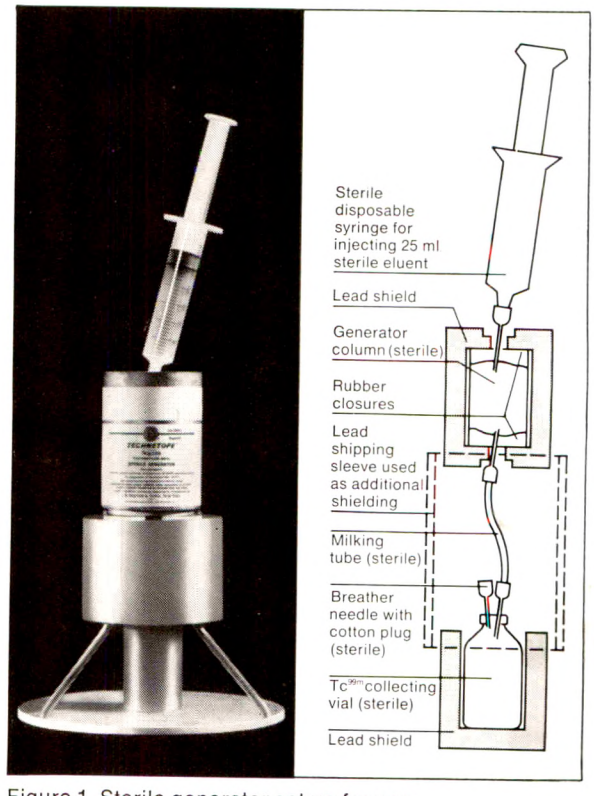



Figure 1. Sterile generator set up for use.

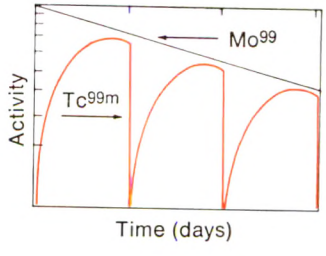



Figure 2. $Mo^{99}$ decay and $Tc^{99m}$ growth after daily elutions.

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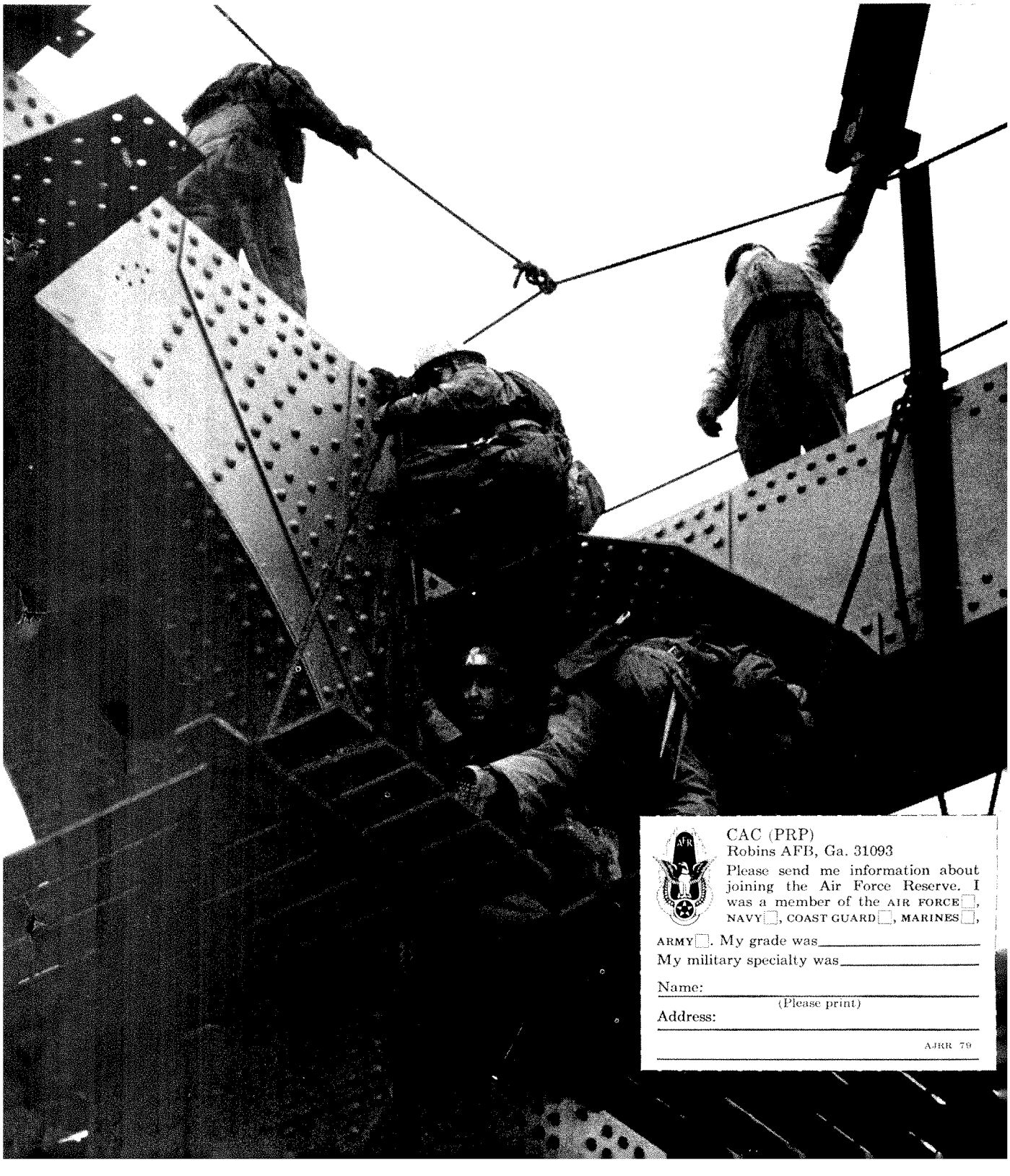
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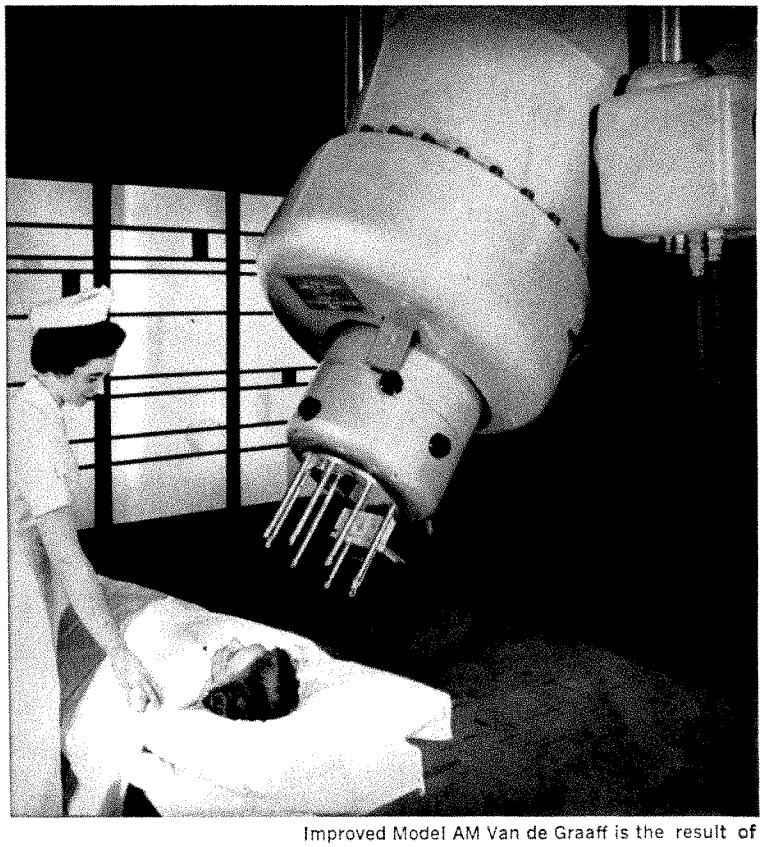
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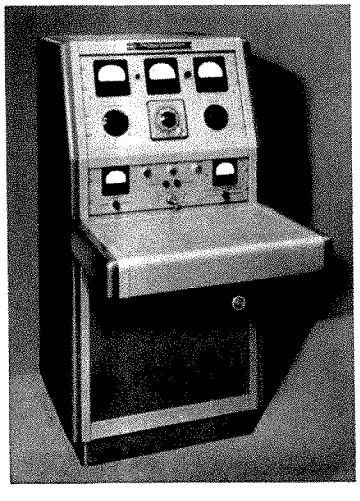


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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 101 SEPTEMBER, 1967 NO. 1

# THE VEIN OF THE LATERAL RECESS OF THE FOURTH VENTRICLE AND ITS TRIBUTARIES*

## ROENTGEN APPEARANCE AND ANATOMIC RELATIONSHIPS

*By* YUN PENG HUANG, M.D., *and* BERNARD S. WOLF, M.D.
NEW YORK, NEW YORK

THE veins of the posterior fossa can be divided into superior, anterior and posterior groups. The superior group has been previously described.[8,9] During study of the anterior group, a unique vein was found intimately related to the lateral recess of the fourth ventricle. To emphasize this relationship, particularly for roentgen diagnostic purposes, this vein has been designated as the "vein of the lateral recess of the fourth ventricle" or, for brevity, the VLR.

Twenty-three normal cerebella were dissected to demonstrate the course, constancy and anatomic relationships of the VLR. In each instance, the vein was found to be located immediately behind and below the lateral recess of the fourth ventricle (Fig. 1). This vein does not run within the lateral recess of the fourth ventricle but immediately adjacent to it in the postero-lateral fissure of the cerebellum (Fig. 1; and 2). In order to demonstrate the anatomic relationships of this vessel and its tributaries, the fourth ventricle and its recesses, the cerebellar tonsil, and the postero-lateral fissure must be described and illustrated in greater detail than available anatomic descriptions furnish (Fig. 2 through 7).

In sagittal section, the fourth ventricle appears as a triangle with a floor and tent-like roof. The roof is divided into two portions which meet at an acute angle. The junction between the two parts of the roof extends backward in the midline over the nodulus as the fastigium. Lateral to the midline, however, the fourth ventricle protrudes further posteriorly and inferiorly as a recess over the superior surface of the cerebellar tonsil on each side. This was designated by Retzius[12] as the "posterior superior recess" of the fourth ventricle. If one views the fourth ventricle from above, these recesses have the appearance of mouse ears (Fig. 4*B*). The midline notch between the two ears corresponds to the core

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From the Department of Radiology of The Mount Sinai Medical School and The Mount Sinai Hospital, New York, New York.

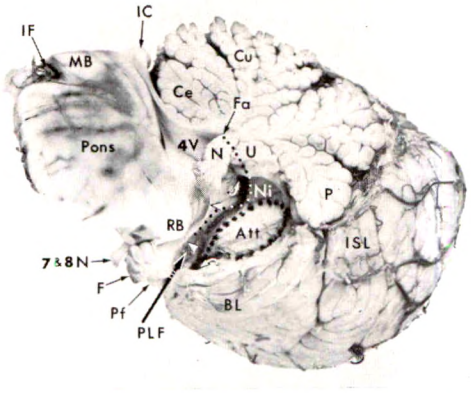



FIG. 1. Anatomic specimen showing the medial aspect of the right cerebellar hemisphere and brain stem. The specimen has been rotated to permit inspection from the inferior as well as medial aspects. The cerebellar tonsil and a portion of the uvula (U) have been removed to expose the "bird's nest" of the tonsil (nidus avis—Ni) and the ovoid area of tonsillar attachment to the white matter of the cerebellum (Att—outlined by large black dots). The medulla oblongata and lower pons have also been removed in order to expose the lateral recess of the fourth ventricle. This is seen as a channel immediately behind the restiform body (RB). In the mid-sagittal plane, the fourth ventricle (4V) is triangular with its apex or fastigium (Fa) directed posteriorly. However, its posterior superior recess (line of small dots), obscured by the nodulus (N) and the uvula, is located more laterally and extends further posteriorly. The vein of the lateral recess (black band between the two white arrows) originates deep in the nidus. It runs downward, forward and laterally between the posterior medullary velum (white arrowhead) and the tonsillar attachment, that is, in the postero-lateral fissure (PLF). Biventral lobule (BL), central lobule (Ce), culmen (Cu), flocculus (F), inferior colliculus (IC), inferior semilunar lobule (ISL), interpeduncular fossa (IF), midbrain (MB), paraflocculus (Pf), pyramid of the inferior vermis (P), 7th and 8th cranial nerves (7 & 8N).

(arbor vitae) of the white matter of the cerebellum (Fig. 1; and 5*C*).

The upper part of the roof of the fourth ventricle is formed by the anterior medullary velum in the midline and the brachium conjunctivum on each side and, more inferiorly, by white matter containing paired cerebellar nuclei. The largest nucleus is the dentate nucleus located in the superior and lateral walls of the posterior superior recess. The lateral wall of the posterior superior recess is indented by a portion of the dentate nucleus which bulges into the cavity (dentate tubercle) (Fig. 4). This dentate tubercle produces a similar indentation (dentate impression) on the antero-lateral aspect of

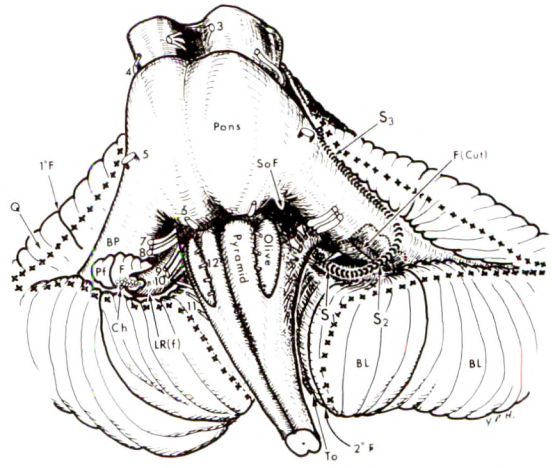



FIG. 2. Diagrammatic representation of the VLR and structures in the "cerebello-ponto-medullary (CPM) angle." On the right side (reader's left), the flocculus (F), paraflocculus (Pf), the superficial portion of the lateral recess (LRf), and the roots of the 9th, 10th and 11th nerves are retained. On the left side, the flocculus and paraflocculus are partly peeled off from their attachments and removed. Their cut surface, F (Cut), is illustrated. The lateral recess is also cut across through its deep portion, a short distance anterior to the lateral angle of the fourth ventricle. Removal of these structures from the CPM angle exposes the peduncular and floccular portions of the postero-lateral fissure. The distal (preincisural) portion of Segment 1 ($S_1$) and Segment 2 ($S_2$) of the VLR are also exposed. Segment 3 ($S_3$) of the VLR runs over the brachium pontis (BP), above the flocculus and below the overhanging quadrangular lip (Q). The CPM angle is a solid angle with its apex directed posteriorly. It is bounded medially by the olive, posteriorly by the antero-lateral aspect of the medulla oblongata, superiorly by the under aspect of the brachium pontis, and laterally by the flocculus and biventral lobule (BL). The lines of crosses indicate the lateral boundaries of the lateral pontine and lateral medullary cisterns. The pia and arachnoid are adherent along these lines. Choroid plexus (Ch), supra-olivary fossette (SoF), cerebellar tonsil (To), secondary fissure (2°F), primary fissure (1°F).

the superior pole of the cerebellar tonsil (Fig. 6).

The lower part of the roof of the fourth ventricle consists of the nodulus in the midline and the posterior medullary velum on each side (Fig. 5). More inferiorly (and anteriorly), the velum becomes continuous with the epithelial portion of the roof, *i.e.*, the tela choroidea. This in turn is attached to the lower lateral part of the floor of the fourth ventricle by the taenia choroidea or ligula. The posterior medullary velum is a thin semilunar shaped, sheet-like structure covering the superior surface of the cerebellar tonsil. It is attached posteriorly to the white matter of the cerebellum, fuses medially with the nodulus, and blends laterally and inferiorly with the floccular peduncle.[10,11,15] When the posterior medullary velum and the tela choroidea of the fourth ventricle are removed, the dome-like superior pole of the cerebellar tonsil is exposed.

The anatomy of the cerebellar tonsil[1,4,5,7,13,14] is difficult to describe because of its bizarre shape (Fig. 6). It may be said to have: (1) a superior pole which occupies the nidus avis and projects upward into the inferior part of the roof of the fourth ventricle lateral to the nodulus; (2) an inferior pole which projects downward and posteriorly into the cisterna magna forming the wall of the vallecula; and (3) a wing-like extension which projects antero-laterally and is applied to the lateral aspect of the medulla oblongata. The superior pole of the tonsil is demarcated from the wing by a notch or "incisura." This point is a useful landmark because it is located immediately behind the lateral angle of the fourth ventricle at the entrance to the lateral recess. Three surfaces of the cerebellar tonsil may be described—superior, medial and lateral. The superior surface forms the ventricular aspect of the superior pole and is continuous antero-laterally with the superior margin of the wing. As noted above, there is a rounded impression on the antero-lateral aspect of the superior surface produced by the dentate tubercle. This dentate impression is situated behind and medial to the tonsillar incisura. The lateral surface of the cerebellar tonsil faces the adjacent biventral lobule and is separated from it by the

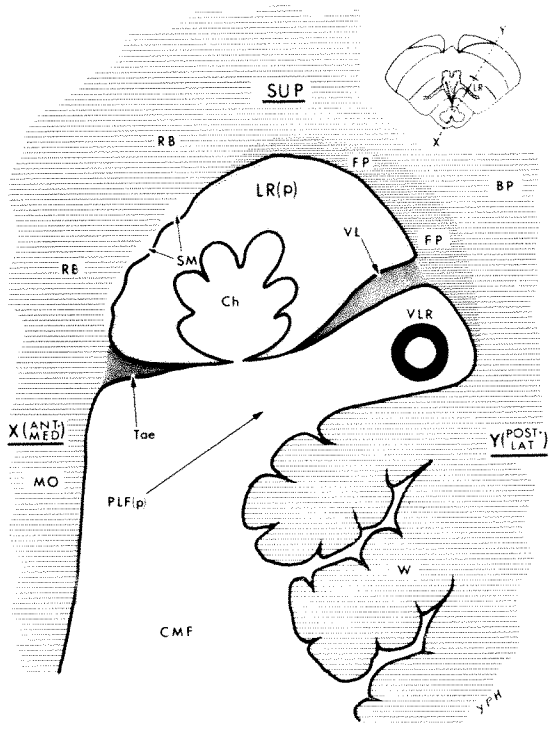



FIG. 3. Cross section through the lateral recess of the fourth ventricle and postero-lateral fissure of the cerebellum. The plane of section (XY in the insert) is at right angles to the lateral recess (LR, insert). The peduncular portion of the postero-lateral fissure, PLF(p), lies below the deep portion of the lateral recess, LR(p), and is separated from it by the velar lip (VL). This lip is an extension of the posterior medullary velum into the floor of the lateral recess. The anterior portion of the floor is formed by a similar lip-like extension of the tela choroidea designated the taenia choroidea (Tae). The choroid plexus (Ch) completes the septum between the lateral recess and the postero-lateral fissure. The VLR runs in the depth of the postero-lateral fissure on the superior aspect of the wing (W) of the cerebellar tonsil. Brachium pontis (BP), cerebello-medullary fissure (CMF), floccular peduncle (FP), medulla oblongata (MO), restiform body (RB), striae medullares (SM). X(ANT-MED), Y(POST-LAT), and SUP indicate the antero-medial, postero-lateral and superior directions of the plane of section. The relationships shown in this section are also evident in the depths of the CPM angle on the left side of Figure 2.

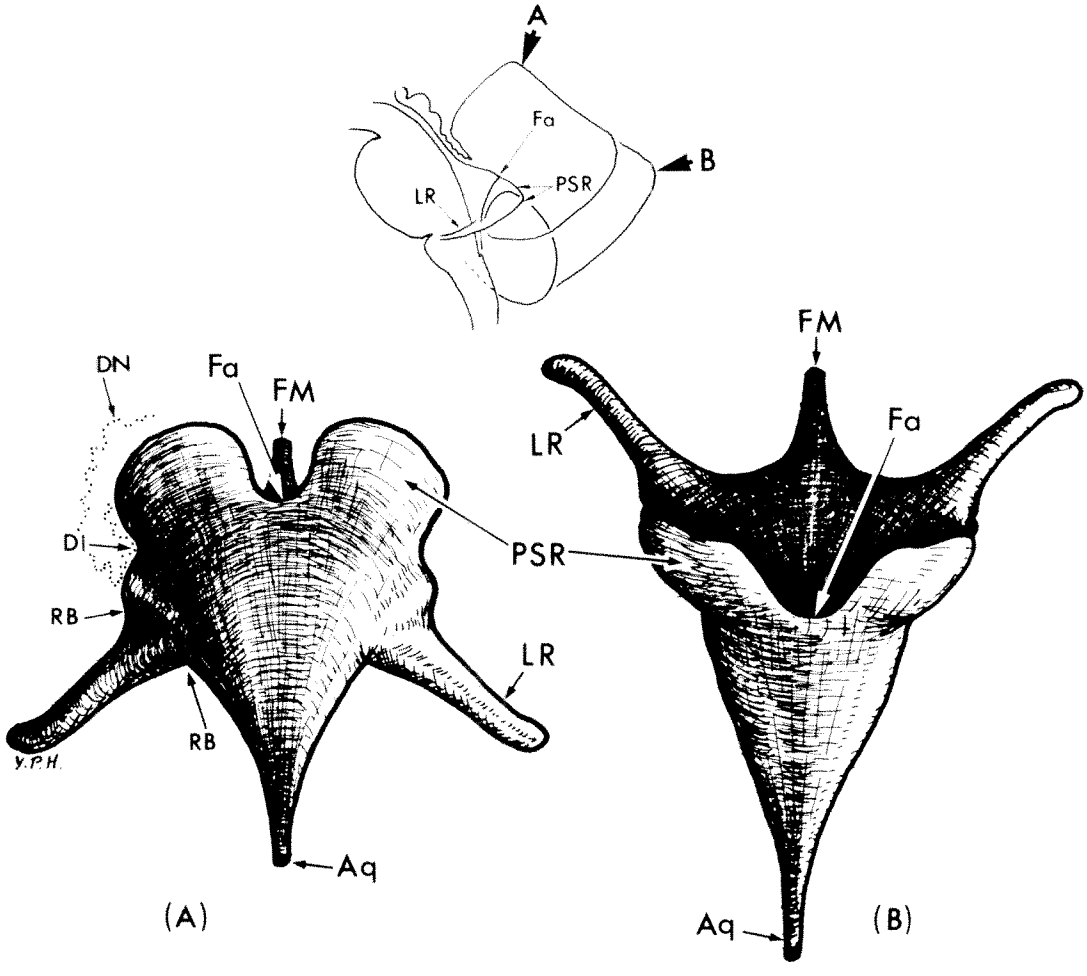



FIG. 4. (*A* and *B*) Two views of the fourth ventricle from aqueduct (Aq) to foramen of Magendie (FM) to illustrate the anatomy of the posterior superior recesses (PSR). Directions of viewing (*A*, *B*) are shown in the insert. In going from *A* to *B*, the fourth ventricle is rotated forward (as seen from above) about 90 degrees, exposing the space below each posterior superior recess. This space is occupied by the superior pole of the cerebellar tonsil. The midline notch between the two posterior superior recesses is occupied by white matter of the cerebellum representing the attachment of the nodulus and uvula to the "arbor vitae." The posterior superior recesses extend for a considerable distance behind the midline fastigium (Fa). The lateral border of the posterior superior recess is indented (dentate impression—DI) by the anterior portion of the dentate nucleus (DN) which bulges into the cavity as a small tubercle. The restiform body (RB) turns around the anterior aspect of the lateral recess (LR) to reach the roof of the ventricle a short distance below the dentate impression.

secondary fissure of the cerebellum. This fissure is quite deep and its full extent cannot be appreciated unless the tonsil and the biventral lobule are artificially separated. When this is done, it can be seen that no portion of the tonsil reaches the anterior surface of the cerebellar hemisphere; *i.e.* the anterior or medullary margin of the secondary fissure ends in the cerebello-medullary fissure. Posteriorly, the secondary fissure extends behind the tonsil as the retrotonsillar fissure and is continuous with the inferior paravermian sulcus. The medial surface of the cerebellar tonsil shows, from front to back, a concavity due to the medulla oblongata (medullary part), a flat portion separated from the same aspect of the opposite tonsil by the vallecula (vallec-

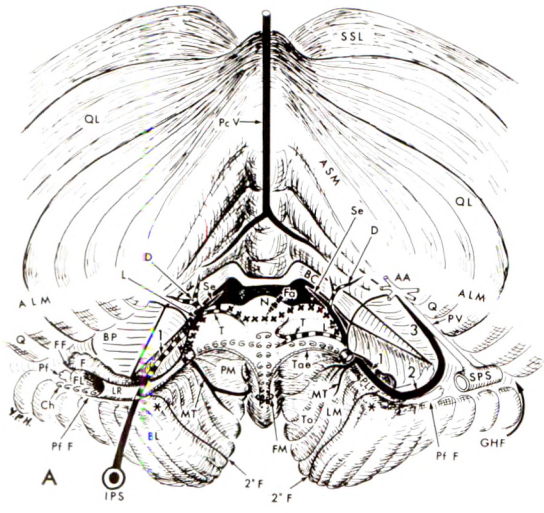



Fig. 5. The anatomic relationships of the vein of the lateral recess of the fourth ventricle. Covered portions of the veins shown in dashed fashion.

(*A*) Simulated Towne projection. The brain stem has been removed by cutting through all the cerebellar peduncles close to the cerebellum. This removes the floor of the fourth ventricle and exposes the structures forming the roof. The curved lines of the taenia choroidea (Tae) indicate the attachment of the roof of the fourth ventricle to the medulla oblongata. The line of crosses *behind* the nodulus (N) and tonsils indicates the junction between the upper and lower parts of the roof of the fourth ventricle. In the midline, this corresponds to the fastigium (Fa). On the right side, the lateral recess (LR) is shown as a complete tube extending from the lateral angle of the fourth ventricle to the foramen of Luschka (FL). The VLR is therefore obscured. On the left side, only a short segment of the lateral recess at the lateral angle of the fourth ventricle remains and more of the cerebellar peduncles has been removed to expose the VLR in the postero-lateral fissure (PLF).

Different arrangements of the transverse (T) and lateral (L) supratonsillar tributaries of the VLR are shown on the two sides. On the left, these vessels unite on the antero-lateral aspect of the superior pole of the tonsil to form the first segment (1) of the vein. On the right, the point of union is more posterior. Small subependymal (Se) and dentate parenchymal (D) veins join the lateral supratonsillar vein. The second (2) and third (3) segments of the VLR are shown in typical fashion on the left. On the right, Segment 1 extends directly forward from the CPM angle (*) to enter the inferior petrosal sinus (IPS). Anterior angle (AA), the anterior lateral (ALM) and the anterior superior (ASM) margins of the cerebellum, biventral lobule (BL), brachium conjunctivum (BC), brachium pontis (BP), choroid plexus (Ch), flocculus (F), floccular portion of the postero-lateral fissure (FF), foramen of Magendie (FM), great horizontal fissure (GHF), lateral medullary (LM) and posterior medullary (PM) parts of the medial surface of the tonsil, medial tonsillar vein (MT), paraflocculus (Pf), parafloccular fissure (PfF), petrosal vein (PV), precentral cerebellar vein (PcV), quadrangular lip (Q), quadrangular lobule (QL), secondary fissure (2°F), superior petrosal sinus (SPS), superior semilunar lobule (SSL), tonsil (To).

ular part), and a posterior superior crescentic area indented by the uvula of the inferior vermis (vermian part). The medullary part can be subdivided into lateral and posterior medullary portions. The junction of these two portions lies opposite the inferior cerebellar peduncle or restiform body. The upper end of this junction corresponds to the location of the tonsillar incisura.

The cerebellar tonsil is attached to the white matter of the cerebellum over a small fusiform area between the superior and lateral surfaces. The plane of attachment is inclined laterally and inferiorly. The lateral surface of the tonsil below the attachment slopes medially and downward (Fig. 5*B*; 5*D*; and 6). The lower border of the tonsillar attachment is therefore the most lateral extension of the tonsil.

The postero-lateral fissure in early embryonic life is a simple superficial sulcus which demarcates the most primitive portion of the cerebellum, that is, the flocculonodular lobe, from the rest of the cerebellum (Fig. 7).[1,2,6] As a result of disproportionate growth of the cerebellar hemispheres, the lower part of the cerebellum rotates downward, forward and then upward. The postero-lateral fissure, especially its medial portion, becomes deeply situated and only a small portion of the fissure below the flocculus remains discernible on the anterior surface of the cerebellum. In addition, the cerebellar tissue between the nodulus and the flocculus atrophies as a result of protrusion of the superior pole of the cerebellar tonsil into the roof of the fourth ventricle. The thinned out portion of the

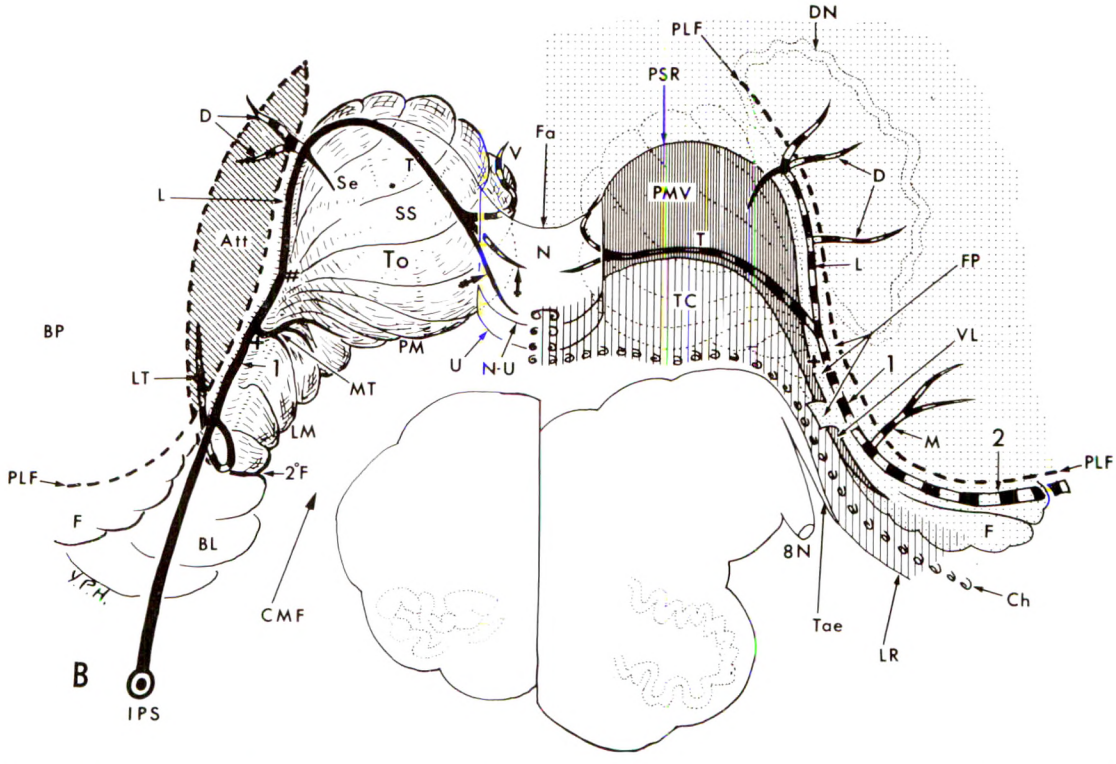



FIG. 5. (*B*) View from above. On the left side, the structures forming the lower part of the roof of the fourth ventricle, *i.e.* the posterior medullary velum (PMV, darkly shaded) and the tela choroidea (TC, vertically shaded) are intact. The extensions of these structures into the floor of the lateral recess (LR), *i.e.* the velar lip (VL) and the taenia choroidea (Tae), are also shown. The upper part of the roof of the fourth ventricle and the roof of the lateral recess have been removed. The VLR is covered by the floccular peduncle (FP) and the flocculus (F). The bundle of fibers derived from the floccular peduncle (FP, lowermost arrow) which traverses the roof of the lateral recess to enter the pons is transected. The posterior medullary velum is continuous with the nodulus (N) medially, the white matter of the cerebellar hemisphere posteriorly and laterally, and the tela choroidea anteriorly. Its velar lip fuses with the floccular peduncle and flocculus. On the left side, typical transverse (T) and lateral (L) supratonsillar veins are shown which unite in the region of the dentate impression (#) (see Fig. 6) to form Segment 1 of the VLR. The lateral supratonsillar vein runs parallel to the lateral border of the posterior superior recess (PSR). The first part of Segment 1 ("postincisural") runs parallel to the lateral wall of the ventricular cavity to the region of the tonsillar incisura (+) or entrance to the lateral recess. Beyond the incisura ("preincisural"), Segment 1 continues in relation to the lateral margin of the recess as far forward as the CPM angle. Segment 2 turns laterally below and usually posterior to the flocculus.

On the right side, the superior surface (SS) of the tonsil (To), the tonsillar wing, the postero-lateral fissure (PLF) and the tonsillar attachment (Att, shaded) are exposed. The brain stem section is at a more caudal level. A vermian tributary (V) from the inferior paravermian sulcus joins other twigs from the depth ( ‡ ) of the nodulo-uvular fissure (N-U) and from the surface ( ‡ ) of the nodulus to form a transverse supratonsillar vein (T). The variation of the VLR illustrated is the same as in Figure 5*A*. A lateral tonsillar vein (LT) is also shown running forward in the secondary fissure (2°F) below the tonsillar attachment to join Segment 1. A medial tonsillar vein (MT) enters the VLR at the incisura. Biventral lobule (BL), the brachium pontis (BP), the cerebello-medullary fissure (CMF), the choroid plexus (Ch), the dentate nucleus (DN), dentate tributaries (D), the fastigium (Fa), the inferior petrosal sinus (IPS), the lateral (LM) and posterior (PM) medullary surfaces of the tonsil, medullary tributaries (M), a subependymal vein (Se), the uvula (U), 8th nerve (8N).

flocculo-nodular lobe becomes the posterior medullary velum and its extension into the lateral recess (velar lip) (Fig. 2; 3; and 5*B*). The posterior medullary velum is therefore a bilateral structure and is not present in the midline.

As the description given above indicates, the space or cleft between the superior pole of the cerebellar tonsil and the posterior medullary velum is embryologically part of

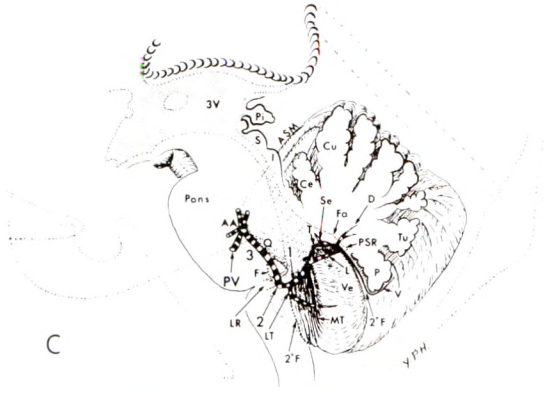



Fig. 5. (*C*) Medial aspect of the right cerebellar hemisphere. The nodulus, uvula and a portion of the pyramid (P) have been removed to expose the vermian surface (Ve) and superior pole of the tonsil. The third ventricle (3V), aqueduct, fourth ventricle and its recesses are shaded. The posterior superior recess (PSR) is located to the right of the midline fastigium (Fa). The secondary fissure (2°F) surrounds the cerebellar tonsil laterally so that only its medullary and vermian borders are evident. The anterior superior margin of the cerebellar hemisphere (ASM), as it runs forward and downward from the culmen (Cu) to the anterior angle (AA), is partly obscured by brain stem. Tributaries of the VLR include dentate (D), subependymal (Se), lateral supratonsillar (L), transverse supratonsillar (T), medial tonsillar (MT), lateral tonsillar (LT), and vermian (V) veins. The lateral supratonsillar vein runs immediately behind and below the posterior superior recess. The postincisural portion of Segment 1 extends from the region of the dentate impression (#) to the incisura (+). The preincisural portion runs below the deep portion of the lateral recess. Segment 2 is foreshortened in this view as it runs laterally below the flocculus (F). Segment 3 turns around the flocculus and runs upward and forward (and medially) to reach the anterior angle of the cerebellum. Central lobule (Ce), inferior colliculus (I), petrosal vein (PV), pineal gland (Pi), quadrangular lip (Q), superior colliculus (S), tuber (Tu) of the inferior vermis.

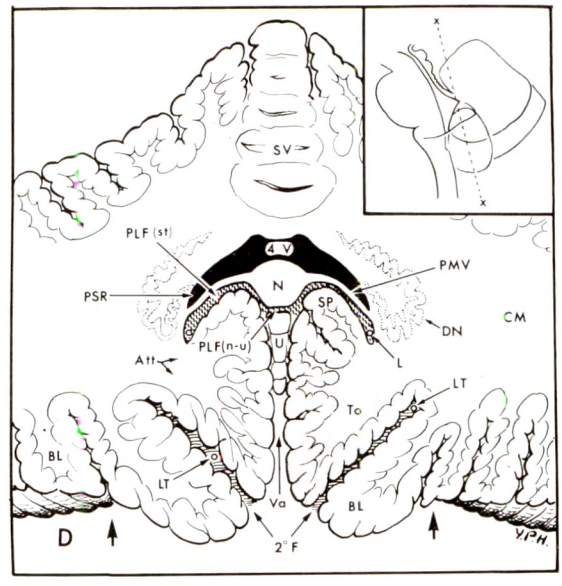



Fig. 5. (*D*) Coronal section of the cerebellum through the long axes of the cerebellar tonsils (XX in insert). The cavity of the fourth ventricle (4V) is indicated in black. The nodulo-uvular and the supratonsillar portions of the postero-lateral fissure, PLF (n–u) and PLF (st), are coarsely shaded. The superior pole (SP) of the tonsil (To) is separated from the cavity of the posterior superior recess (PSR) by this fissure and the posterior medullary velum (PMV). The attachment (Att) of the tonsil to the white matter (the corpus medullare, CM) intervenes between the lateral margin of the supratonsillar cleft and the depth of the secondary fissure (2°F). The lateral supratonsillar tributary (L) runs above the tonsillar attachment whereas the lateral tonsillar vein (LT) runs below the attachment in the secondary fissure at a variable level. The indentation (unlabeled thick arrows) on the undersurface of each biventral lobule (BL) is created by the anterior extension of the bony internal occipital crest around the foramen magnum. Dentate nucleus (DN), nodulus (N), superior vermis (SV), uvula (U), vallecula (Va).

the postero-lateral fissure. The postero-lateral fissure may therefore be said to consist of four portions: nodulo-uvular, supratonsillar, peduncular and floccular (Fig. 1; 3; 5*B*; 5*D*; and 7). The nodulo-uvular portion remains transverse in the midline but becomes quite deep during growth of the vermis. The supratonsillar portion becomes a cleft above the superior pole of the tonsil continuous with the retrotonsillar fissure posteriorly and the vallecula medially. This

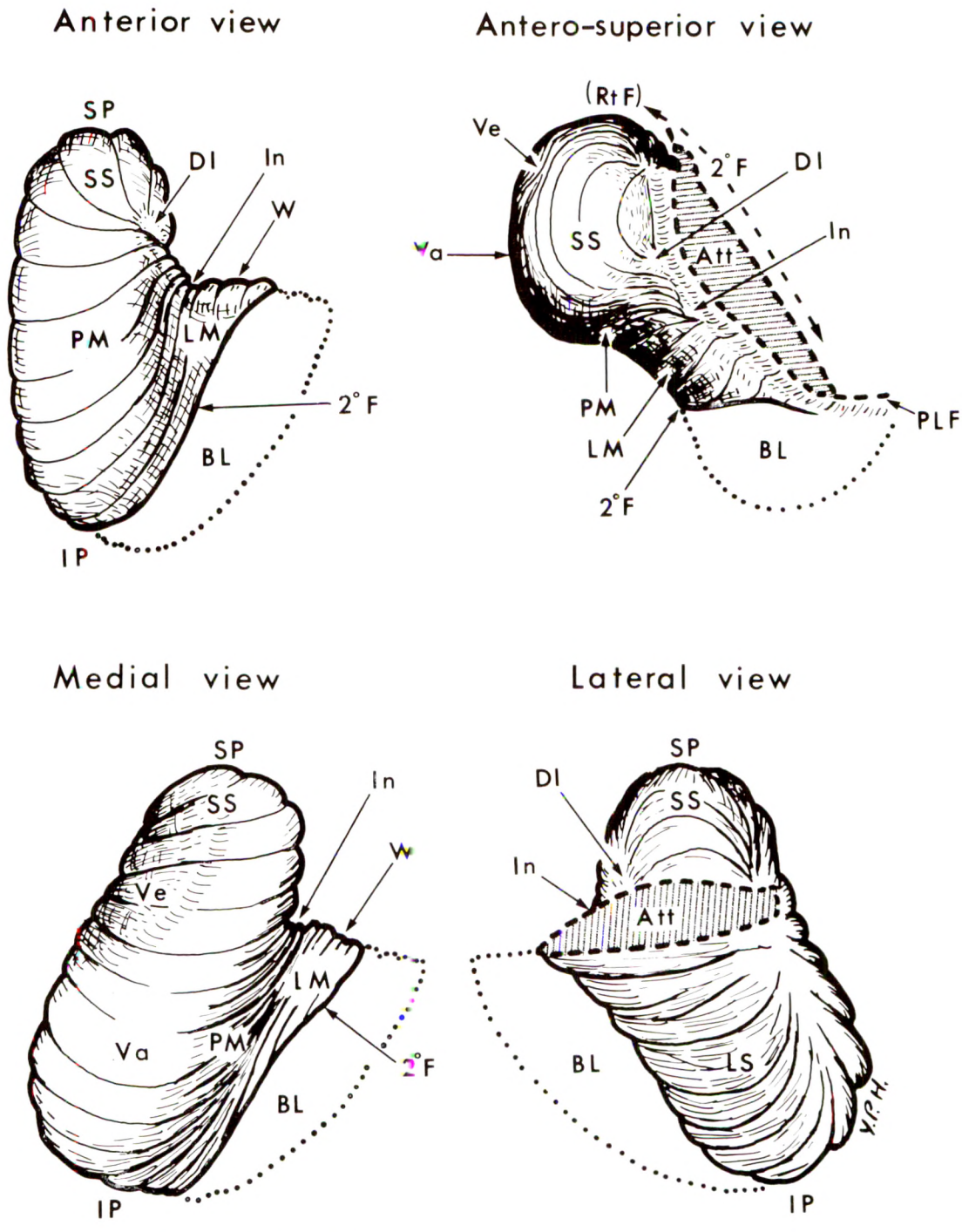



FIG. 6. Anatomy of the cerebellar tonsil; views of the left tonsil.

The cerebellar tonsil is separated from the adjacent biventral lobule (BL—only the medial portion is outlined) by the secondary fissure (2°F). The tonsil has a superior pole (SP) which projects upward into the roof of the fourth ventricle, an inferior pole (IP) which protrudes downward into the cisterna magna, and a wing (W) which projects antero-laterally and forms the lateral wall of the cerebello-medullary fissure. The tonsillar incisura (In), a notch located between the superior pole and tonsillar wing, is situated immediately below and behind the lateral angle of the fourth ventricle or entrance to the lateral recess.

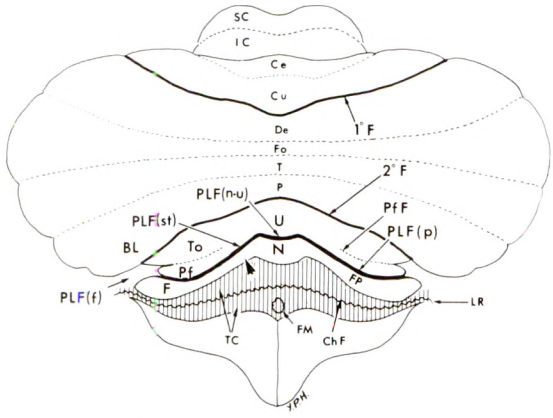



Fig. 7. Cerebellar divisions in the embryo with special reference to the development of the postero-lateral fissure.

The cerebellum originates from the dorsal aspect of the metencephalon. At about the 100 mm. stage, fissures appear on its posterior surface. The first of these is the postero-lateral fissure (PLF) which separates the flocculo-nodular lobe—nodulus (N), flocculus (F), and floccular peduncle (FP)—from the rest of the cerebellum. Shortly after, the primary fissure (1°F) appears in the most rostral portion to separate the anterior lobe from the remainder. As the cerebellar hemisphere increases in size, the flocculo-nodular lobe and the adjacent paraflocculus (Pf), tonsil (To) and uvula (U) are displaced downward, forward and then upward. The nodulus, flocculus and paraflocculus, originally situated caudally, become located above the tonsil and uvula. As the tonsil projects upward, the portion (unlabeled thick arrow) of the flocculo-nodular lobe between the floccular peduncle and the nodulus becomes thinned-out and atrophied forming the posterior medullary velum. Since the paraflocculus is small and often rudimentary, the fissure between the flocculus and the cleft is closed laterally by the white matter of the cerebellum between the attachments of the posterior medullary velum and the cerebellar tonsil (Fig. 5*D*). It communicates, however, anteriorly above the incisura with the channel-like peduncular portion of the postero-lateral fissure running along the wing of the tonsil. The boundaries of this portion are indicated in Figure 3. At the junction of the flocculus with its peduncle, in the cerebello-ponto-medullary (CPM) angle, the fissure turns laterally and runs below the flocculus (floccular portion) to fuse with the great horizontal fissure of the cerebellum (Fig. 2; and 5*A*). The postero-lateral fissure communicates with the secondary fissure anteriorly below the flocculus (Fig 5; and 6). Posteriorly, the secondary fissure also joins the postero-lateral biventral lobule may be considered a part of the postero-lateral fissure (floccular part). The postero-lateral fissure, therefore, can be divided into a nodulo-uvular portion, PLF (n-u); a supratonsillar portion, PLF (st); a peduncular portion, PLF (p); and a floccular portion, PLF (f). Central lobule (Ce), choroid fissure (ChF) of the fourth ventricle, culmen (Cu), declive (De), folium (Fo), foramen of Magendie (FM), inferior colliculus (IC), lateral recess (LR), parafloccular fissure (PfF), pyramid (P), secondary fissure (2°F), superior colliculus (SC), tela choroidea (Tc), tuber (T).

A small impression (the dentate impression—DI) on the antero-lateral aspect of the superior pole is produced by the dentate tubercle shown in Figure 4. Medially, the folia and sulci of the superior pole and of the medullary surface converge toward the incisura. The folia of the lateral surface (LS) resemble an inverted tree with a vertical trunk of exposed white matter located posteriorly. Corresponding to the neighboring anatomic structures, the medial surface of the cerebellar tonsil can be divided from front to back into lateral medullary (LM), posterior medullary (PM), vallecular (Va), and vermian (Ve) parts. Since the vermis protrudes only into the upper portion of the vallecula, the vallecular part of the medial surface extends well below the vermian part. The postero-lateral fissure is located along the superior margin of the tonsillar attachment (Att) and spreads out over the superior surface (SS). It joins the secondary fissure posteriorly in the retrotonsillar fissure (RtF). Anteriorly, the secondary fissure turns around the wing to appear in the cerebello-medullary fissure. The depth of the secondary fissure communicates with the floccular portion of the postero-lateral fissure (PLF).

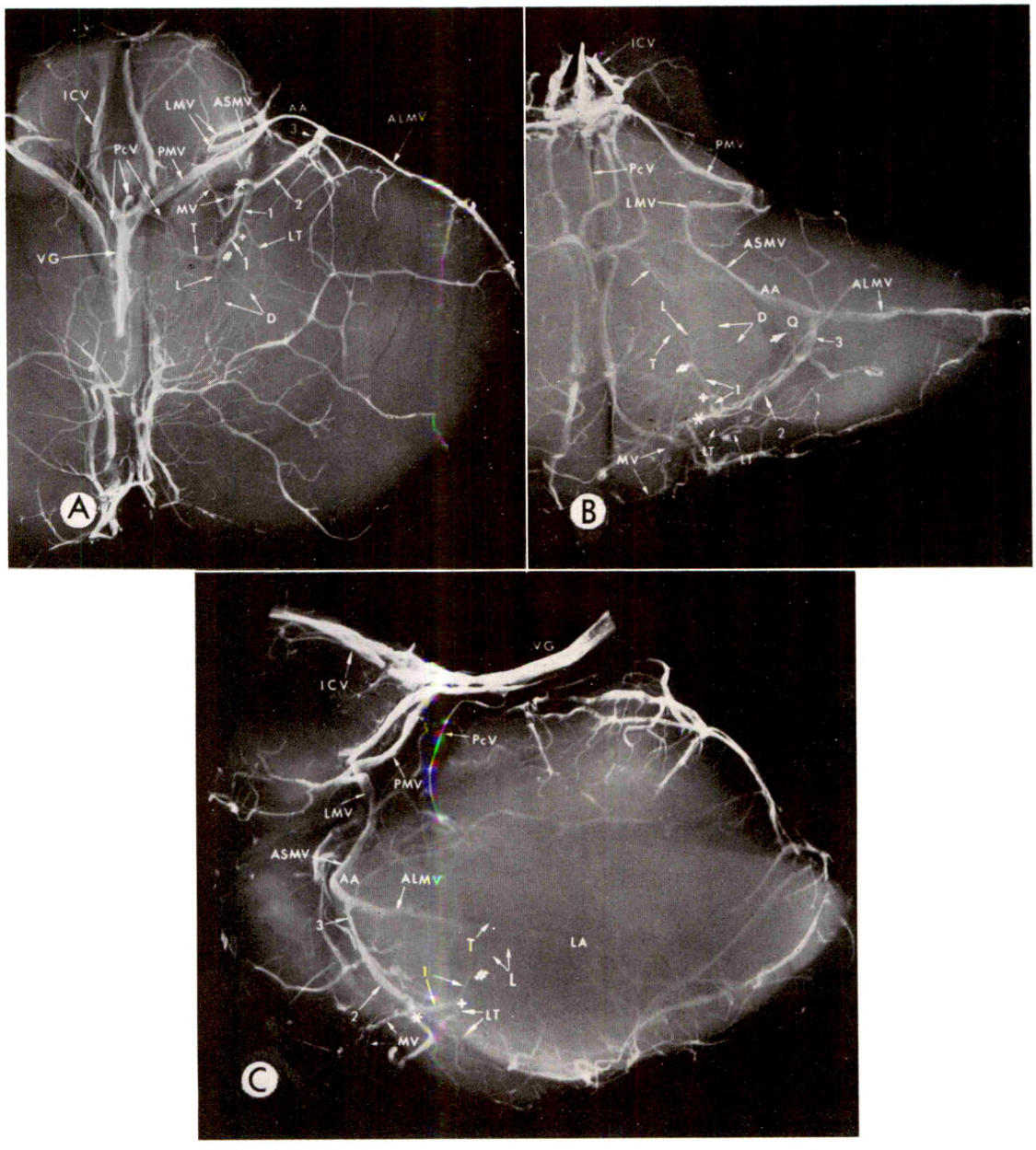



FIG. 8. Roentgenograms of an injected specimen. Colored radiopaque material was injected into the straight sinus and the dura then removed. Descriptions given below are based on subsequent dissection.

(*A*) View from above ("base" view). The brain stem and thalami are located superiorly. The first and second segments (1, 2) of the VLR are clearly seen. The third segment (3) is atypical. The first segment of the vein begins by the union of the lateral supratonsillar (L) and the transverse supratonsillar tributaries (T) adjacent to the dentate impression (#). The dentate tributaries (D) run medially and forward to drain into the lateral supratonsillar vein. An angulation in the course of the vein is present at the incisura (+). The lateral tonsillar vein (LT) opens into the first segment of the vein after turning around the anterior margin of the wing of the cerebellar tonsil. The VLR reaches the anterior aspect of the cerebellum at the CPM angle (*) and turns laterally to continue as Segment 2. The anomalous third segment (3) runs upward over the quadrangular lip to join a vein running along the anterior lateral margin of the cerebellum (ALMV). A vein from the medulla oblongata (MV) joins the VLR at the CPM angle. Anastomotic lateral mesencephalic vein (LMV), anterior angle (AA), anterior superior marginal vein (ASMV), internal cere-

fissure, behind the tonsillar attachment, in the retrotonsillar fissure (Fig. 6).

The lateral recess of the fourth ventricle extends antero-laterally from the lateral angle of the fourth ventricle to the foramen of Luschka.[3,10] It may be divided into two portions—deep (peduncular) and superficial (flocculär) (Fig. 2; 3; and 5*B*). The deep part runs parallel to the peduncular portion of the postero-lateral fissure and is separated from it by the extension (velar lip) of the posterior medullary velum (Fig. 3). The flocculär portion of the lateral recess is located in the cerebello-ponto-medullary (CPM) angle. The posterior wall of the superficial portion is formed by the flocculus while the membranous anterior wall is formed by adherent pia and ependyma. The roots of the 9th and 10th nerves are intimately attached to the anterior wall. A rather abrupt angle may be present between the two portions of the lateral recess which corresponds to a similar angle between the floccular peduncle and the flocculus (Fig. 2; and 5*B*).

The appearance of the VLR and its tributaries were studied on roentgenograms of injected specimens prior to dissection (Fig. 8, *A–C*). The VLR originates in the region of the dentate impression on the antero-lateral surface of the superior pole of the tonsil and runs forward and laterally to the incisura and then along the wing of the tonsil to the CPM angle. This most constant portion of the vein is referred to as Segment 1 or the "peduncular" segment. The first portion of this segment (postincisural) runs in relation to the lateral wall of the ventricular cavity proper. Beyond the incisura (preincisural portion), the vein is in relation to the deep part of the lateral recess (Fig. 5*B*). Segment 2 (flocculär segment) of the VLR begins in the depth of the CPM angle and crosses laterally below the flocculus in relation to the superficial portion of the lateral recess. Segment 3 (the supraflocculär segment) turns medially, upward and forward to reach the anterior angle of the cerebellum. This angle is the junction of the anterior superior and the anterior lateral margins of the cerebellar hemisphere. The exact course of Segment 3 is variable. It may run in the cleft between the brachium pontis and adjacent overhanging lip of the quadrangular lobule (quadrangular lip), or more laterally over the lip itself, or more medially over the brachium pontis near the belly of the pons. At the anterior angle, other veins (from the wing of the precentral cerebellar fissure, from the anterior lateral and anterior superior margins of the hemisphere, from the anterior aspect of the pons, from the great horizontal fissure of the cerebellum and from the superior and inferior surfaces of the hemisphere) join the VLR

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bral vein (ICV), precentral cerebellar vein and its tributaries (PcV), posterior mesencephalic vein (PMV), vein of Galen (VG).

(*B*) Antero-posterior view. The demarcation between the superior and anterior surfaces of the cerebellar hemisphere is indicated by the vein running along the anterior lateral margin of the cerebellum (ALMV). The anterior superior marginal vein (ASMV) joins this vein at the anterior angle (AA). The anastomotic lateral mesencephalic vein (LMV) connects the posterior mesencephalic vein (PMV) with the anterior superior marginal vein. The precentral cerebellar vein (PcV) and its tributaries (unlabeled small arrow) originate behind the brain stem. A small vein (unlabeled thick arrow) running along the inferior margin of the quadrangular lip (Q) joins the VLR.

(*C*) Lateral view. The anterior lateral margin extends from the anterior angle (AA) to the lateral angle (LA) of the cerebellum. The vertical portion of the anastomotic lateral mesencephalic vein (LMV) marks the lateral mesencephalic sulcus. The anomalous third segment of the vein of the lateral recess runs upward and crosses the quadrangular lip to join the anterior lateral marginal vein (ALMV). Note the angulation in the course of the first segment (1) of the VLR at the incisura (+).

to form the petrosal vein. The petrosal vein is thus a sizeable although short channel which crosses the subarachnoid and subdural space to enter the superior petrosal sinus.

In most instances, two veins located on the superior pole of the tonsil unite in the region of the dentate impression to form Segment 1 of the VLR (Fig. 5). The larger of these two vessels, the "transverse supratonsillar vein" is formed by the confluence of several small veins arising from the vermian and vallecular parts of the medial surface of the cerebellar tonsil, from the nodulo-uvular fissure, and the surface of the nodulus. The vein related to the nodulus connects the transverse supratonsillar veins of the two sides. Occasionally, small parenchymal medullary veins drain into this communicating vein or into the transverse supratonsillar vein along each border of the nodulus. The smaller supratonsillar vein, "lateral supratonsillar vein," runs on the lateral aspect of the superior pole of the tonsil parallel to the lateral margin of the posterior superior recess. Several small veins join the lateral supratonsillar vein. These arise in the white matter of the cerebellum in the region of the dentate nucleus (dentate or medullary veins) and subependymally from the roof of the fourth ventricle.

Tributaries of the VLR also arise on the medullary aspect of the cerebellar tonsil (medial tonsillar veins) and often converge toward the incisura to form a short stem before opening into the first segment of the VLR (Fig. 5*A*; 5*B*; and 5*C*). In other instances, a common stem runs on the medullary aspect of the wing below the lateral recess and joins the second segment of the VLR. A "lateral tonsillar vein" may be present which runs anteriorly in the secondary fissure to join the first or second segments of the VLR (Fig. 5*B*; 5*C*; and 5*D*). More often, however, the lateral tonsillar vein drains backward to join the posterior venous system.

The VLR and its supratonsillar tributaries can often be recognized in the course of vertebral angiography despite their small size. In order to identify these vessels, roentgenograms of excellent quality as well as satisfactory filling of the vertebral circulation are required. Stereoscopic views are helpful in distinguishing surface from deep veins. The typical appearance of the VLR and its relations to the more familiar pneumoencephalographic appearances of the fourth ventricle and basal cisterns are shown in Figure 9, *A–C*. In the Towne projection, Segment 1 begins about 12 mm. from the midline and runs laterally and downward, occasionally with a slight outward convexity. The length of this segment varies between 10 and 16 mm. The length depends on the exact point of union of the supratonsillar tributaries, the forward extent of the loop formed at the junction of the first and second segments, and the angle of projection. If the course of the vessel is prolonged geometrically, it "intersects" the petrous ridge at a point slightly medial to the medial margin of the internal auditory meatus. The angle which the vein makes with the sagittal plane averages about 41 degrees with a range of 30 to 48 degrees (Fig. 10 through 14). When the veins of both sides are filled, a symmetric inverted V-shaped configuration is produced (Fig. 12*D*). In the anterior-posterior or Towne projection, the lateral supratonsillar vein runs downward almost parallel to the sagittal plane about 12 mm. (9 to 14 mm.) from the midline (Fig. 9*B*; 10*A*; and 12*C*).

The appearance of Segment 1 of the VLR is quite constant. However, the course of the second and third segments is variable (Fig. 10*A*; 11*B*; and 12, *A–D*). The junction between Segment 1 and 2 is usually clearly identifiable by an abrupt change in course forming an angle of about 90 degrees. In some instances, Segment 2 of the VLR, after emerging from the CPM angle, runs antero-superiorly and opens into the medial portion of the superior petrosal sinus (Fig. 13, *A* and *B*). This vessel may also run directly forward to join the

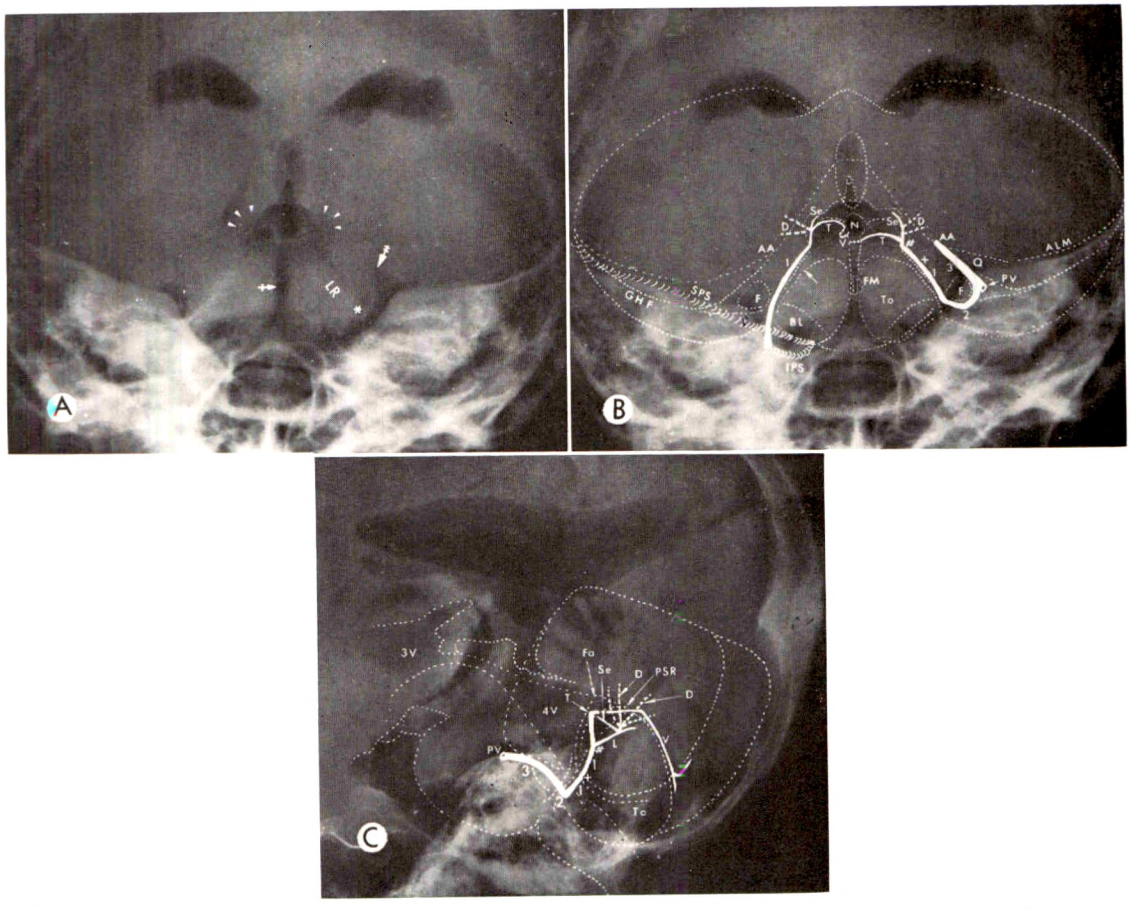



FIG. 9. Correlation of the appearance of the VLR and its tributaries with pneumoencephalographic features Labeled structures include the anterior angle (AA), anterior lateral margin (ALM), biventral lobule (BL), dentate vein (D), fastigium (Fa), flocculus (F), foramen of Magendie (FM), great horizontal fissure of the cerebellum (GHF), inferior petrosal sinus (IPS), lateral supratonsillar vein (L), nodulus (N), petrosal vein (PV), posterior superior recess (PSR), quadrangular lip (Q), subependymal vein (Se), superior petrosal sinus (SPS), transverse supratonsillar vein (T), tonsil (To), vermian vein (V), third and fourth ventricles (3V and 4V).

(*A*) Pneumoencephalogram—Towne view. The landmarks outlined by gas include the posterior superior recesses (unlabeled small arrows), the lateral recess (LR), and the foramen of Magendie superimposed upon the vallecula ( ‡ ). The pontine cistern extends laterally to form a triangular collection of air ( ‡ ) located in front of the brachium pontis between the quadrangular lip above and the flocculus below.

(*B*) Same Towne view; the VLR is diagrammatically superimposed on the pneumoencephalogram. On the right side, the VLR is shown running directly forward to join the lower end of the inferior petrosal sinus. On the left side, the VLR with three typical segments is shown extending to the region of the anterior angle and entering the petrosal vein. (Segment 3 and the petrosal vein are superimposed.) The positions of the dentate impression (#) and the tonsillar incisura (+) are indicated. The unlabeled arrow marks the location of the lateral angle of the fourth ventricle.

(*C*) Lateral view. A typical VLR (1, 2 and 3) and tributaries are illustrated, superimposed on an air study. A prominent vermian vein (V) is shown running in the inferior paravermian sulcus and joining the transverse supratonsillar vein (T) at a sharp angle.

inferior petrosal sinus (Fig. 14, *A* and *B*). Under these conditions, the second and third segments of the VLR are absent.

In the lateral projection, Segment 1 of the VLR runs forward and downward (Fig. 10*B*; 11*A*; 13*B*; and 14*B*). The geometric continuation of the course of this segment of the vein in this projection "intersects" the *external* auditory meatus or more often a point a short distance below the meatus. The transverse supratonsillar vein, in its typical form, joins the first segment of the VLR at an obtuse angle directed inferiorly (Fig. 11*A*; 13*B*; and 14, *A* and *B*). The site of angulation corresponds to the region of the dentate impression on the tonsil. The distance between this angle and the anterior margin of the foramen magnum ranges from 30 to 41 mm. under normal circumstances. The pre- and postincisural subsegments of Segment 1 may also form an obtuse angle directed inferiorly at the tonsillar incisura (Fig. 11*A*; 13*B*; and 14*B*). In the lateral projection, the transverse supratonsillar vein loops upward or makes a distinct angle as it runs over the superior pole of the cerebellar tonsil to enter the VLR (Fig. 11*A*; 13*B*; and 14*B*).

Medial tonsillar and lateral tonsillar veins can occasionally be identified on angiography and serve as landmarks for the appropriate tonsillar surfaces (Fig. 8, *A–C*; and 15, *A* and *B*). These veins must be distinguished from Segment 1 of the VLR since they also show an oblique course anterolaterally and inferiorly. In the Towne

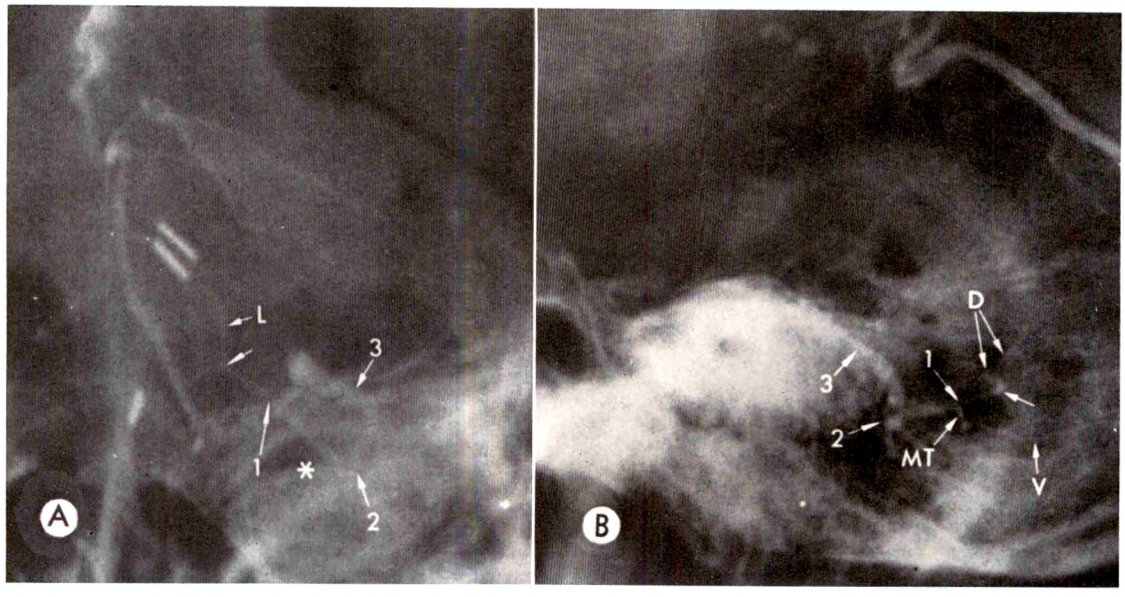



FIG. 10. Normal VLR; venous phases, vertebral angiograms. Previous left frontal craniotomy for a convexity meningioma is evident. Vessels and anatomic landmarks are labeled in this and subsequent figures as in the previous diagrams; an unlabeled thin arrow indicates the location of the dentate impression; the arrow for Segment 1 also indicates the position of the incisura of the tonsil.

(*A*) Towne view. The lateral supratonsillar vein (L) runs parallel to the midline and enters the VLR at a sharp angle at the dentate impression (unlabeled arrow). The normal distance between this vein and the midline and the normal position of Segment 1 indicate that the fourth ventricle is not enlarged. Segment 2 loops below the flocculus. In this case, this loop extends unusually far forward and downward over the upper margin of the biventral lobule. Segment 3 runs upward, medially (and anteriorly) in the cleft between the brachium pontis and quadrangular lip and then joins the petrosal vein at the anterior angle.

(*B*) Same case—lateral view. A vermian vein (V) continues on the medial aspect of the cerebellar tonsil as a medial tonsillar vein (MT) which enters the first segment of the VLR at the incisura (arrow 1). Several parenchymal or dentate veins (D) are visualized. Note the acute angulation between the first and second segments of the VLR.

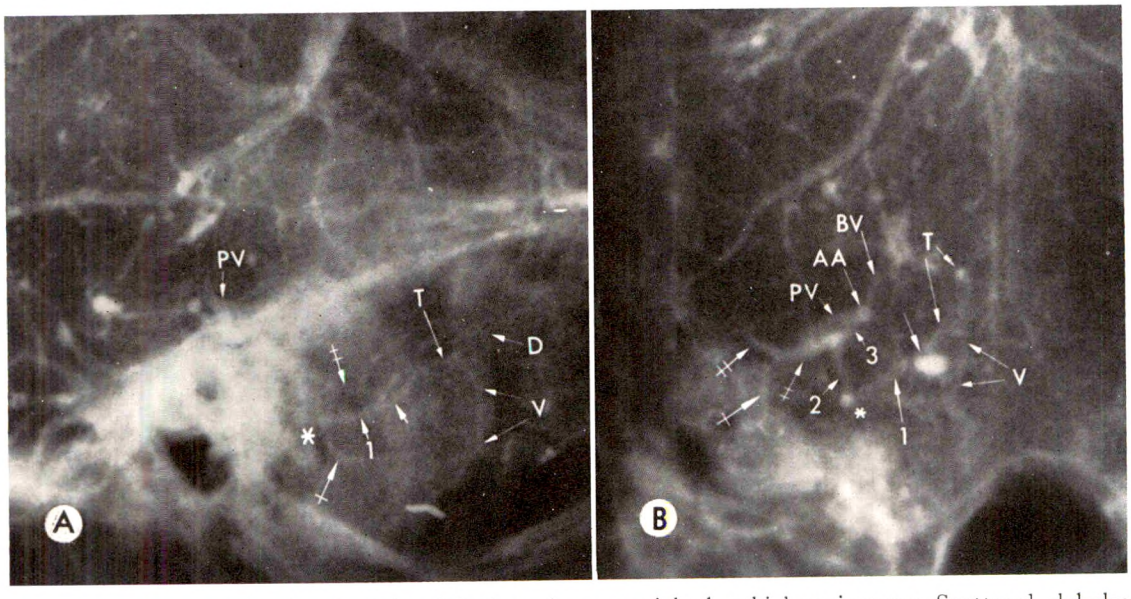



Fig. 11. Another case showing the vein of the lateral recess; right brachial angiograms. Scattered globules of radiopaque material are due to previous pantopaque studies.

(*A*) Lateral view. The VLR originates as a large vermian tributary (V) running upward in the retrotonsillar fissure and continuing as the transverse supratonsillar vein (T) into the first segment (1) of the vein. The anterior end of Segment 1 identifies the junction between the deep and superficial portions of the lateral recess in the depth of the CPM angle (*). The second and third segments of the vein are obscured by the petrous pyramids. Only a small portion of the petrosal vein (PV) is seen at the anterior angle. Superficial veins ( ‡ and ↕ ) running on the anterior and inferior aspects of the hemisphere are also evident.

(*B*) Towne view. The vermian tributary (V) indicates the position of the inferior paravermian sulcus. This vein crosses over the superior pole of the cerebellar tonsil (transverse supratonsillar vein—T) and joins the first segment (1) of the VLR. The second segment (2) shows an acute angulation as it turns upward. The third segment (3) of the vein is joined by surface veins from the great horizontal fissure ( ‡ ) and inferior aspect ( ↕ ) of the cerebellum before draining into the petrosal vein (PV) at the anterior angle (AA). A brachial vein (BV), running on the wing of the precentral fissure, also drains into the petrosal vein.

view, the points of "intersection" with the petrous ridge are, however, distinctly more lateral or medial than the extension of Segment 1 of the VLR. In the lateral view, these veins are located lower than the first segment of the VLR and their anterior portions curve smoothly upward. In the lateral view, these veins as well as the VLR may be confused with surface hemispheric veins (Fig. 11*A*). Superficial veins are, however, easily distinguished from the deeper veins in the antero-posterior projection (Fig. 11*B*). Another vein with an oblique course runs in the lateral extension of the precentral cerebellar fissure on the superior surface of the brachium conjunctivum and brachium pontis. This "brachial vein" is located distinctly more laterally and superiorly than the VLR and is usually more oblique in its course (Fig. 11*B*; 12*B*; and 12*D*). In a rare instance, medullary (or dentate) veins draining into the lateral supratonsillar vein or directly into Segment 1 of the VLR are surprisingly large and may simulate tonsillar veins. Such enlargement is associated with increased caliber of the VLR and apparently represents a normal variation in the direction of drainage of the hemispheric white matter. However, the VLR may also serve as a drainage pathway for an arteriovenous malformation and be markedly dilated (Fig. 17, *A* and *B*). It is clear also that tumors of the fourth ventricle are likely to produce characteristic dis-

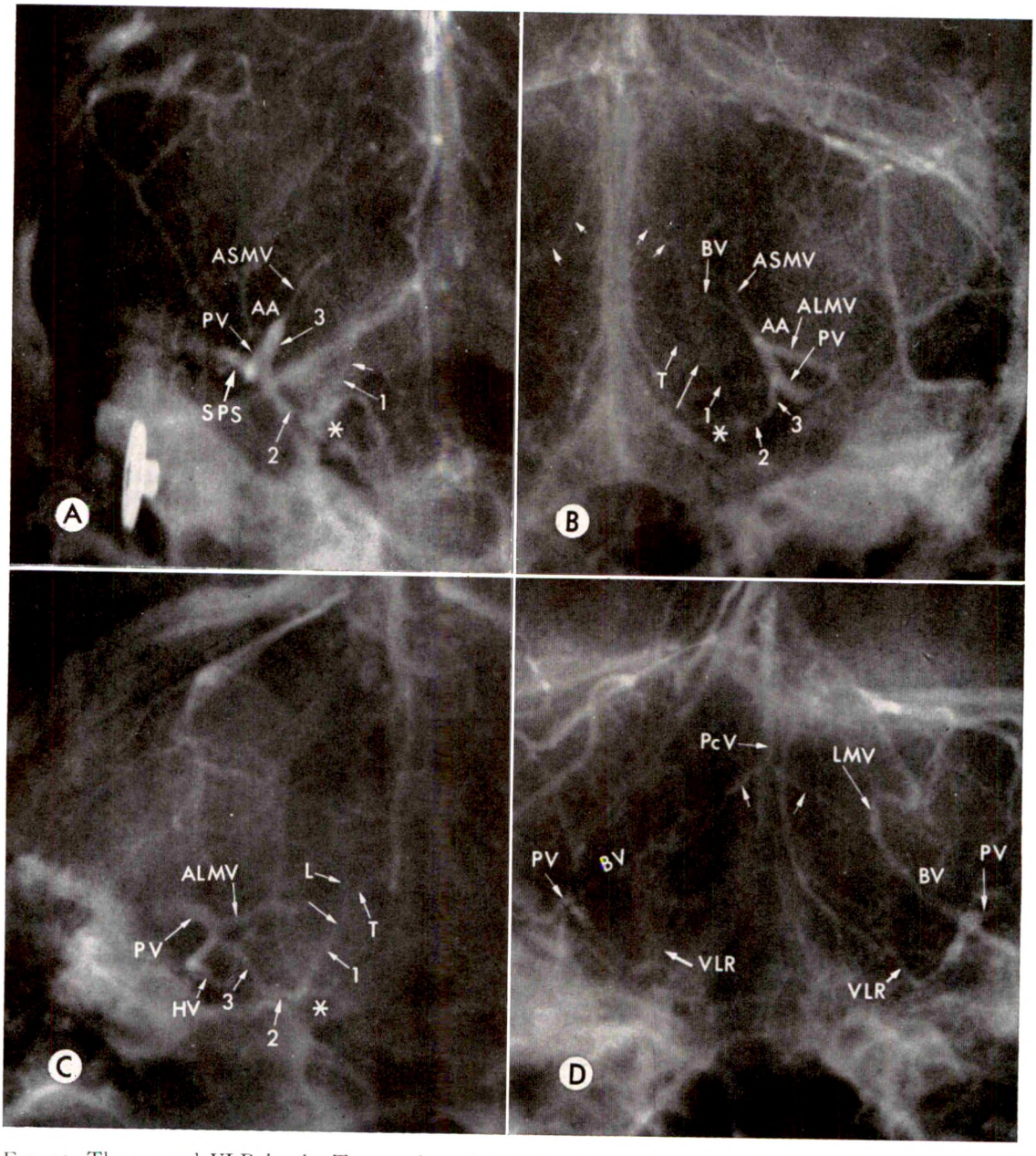



FIG. 12. The normal VLR in the Towne view; four examples.

(*A*) The three segments of the vein (1, 2, 3) are clearly seen. The distance between Segment 1 and Segment 3 indicates the thickness of the brachium pontis and restiform body.

(*B*) The transverse supratonsillar tributary (T) enters the first segment (1) at a small angle. In this case, the third segment (3) does not reach the anterior angle (AA) of the cerebellum but joins the mid-portion of the petrosal vein (PV). The anterior superior and anterior lateral marginal veins (ASMV and ALMV) unite to form the petrosal vein at the anterior angle. Short unlabeled arrows indicate symmetric brachial tributaries of the precentral vein behind the brain stem. They communicate with a brachial tributary (BV) of the petrosal vein.

(*C*) The lateral supratonsillar vein (L) runs downward parallel to the midline. It joins the first segment (1) of the VLR at an angle corresponding to the site of the dentate impression (unlabeled arrow). An anomalous third segment (3) runs laterally over the quadrangular lip to join the petrosal vein (PV). The latter is also joined by an anterior lateral marginal vein (ALMV) and a large vein from the inferior

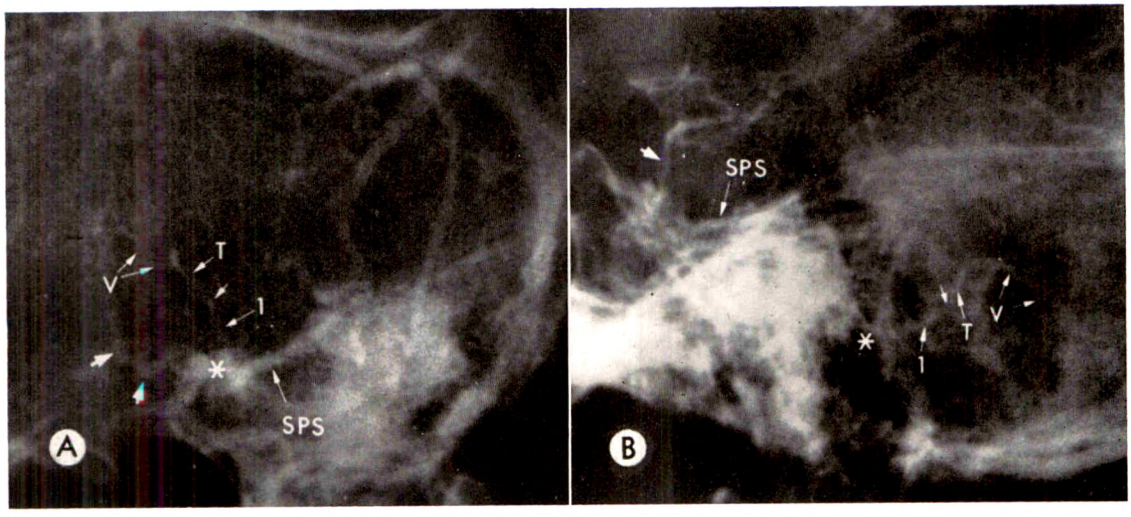



Fig. 13. Drainage of the VLR directly into the superior petrosal sinus; left brachial angiograms.

(*A*) Towne view. A vermian tributary (V) continues as the transverse supratonsillar vein (T) and enters the first segment (1) of the VLR. The vein enters the CPM angle (*) and continues forward (and upward) to join the medial portion of the superior petrosal sinus (SPS) with other tributaries from the anterior aspect of the pons (unlabeled thick arrows).

(*B*) Lateral view. Posteriorly, the vermian vein (V) outlines the inferior paravermian sulcus or retrotonsillar fissure. The direct communication between the first segment (1) and the superior petrosal sinus (SPS) is obscured by the petrous pyramid. The angulation between the transverse supratonsillar vein (T) and the first segment of the VLR identifies the location of the dentate impression (unlabeled arrow). A vein outlining the anterior aspect of the pons is also evident (unlabeled thick arrow).

placement and deformity of the VLR (Fig. 18, *A* and *B*; and 19, *A* and *B*). These changes complement other angiographic features and permit more precise localization.

## SUMMARY

1. A vein, designated the vein of the lateral recess of the fourth ventricle (VLR), is described which has unique anatomic relationships to the fourth ventricle and adjacent structures. Recognition of this vessel on angiography makes it possible to localize these structures.

2. Tributaries of the VLR can often be identified if filling is satisfactory. These include parenchymal, subependymal, medial and lateral tonsillar, and supratonsillar veins.

3. The roentgen appearances of the VLR and its tributaries are illustrated in injected specimens and *in vivo*. The anatomic

aspect of the cerebellar hemisphere (hemispheric vein—HV). The transverse supratonsillar vein (T) is faintly seen.

(*D*) Both VLR are visualized. In addition, brachial veins (BV) on each side run infero-laterally to the anterior angle to join the petrosal vein (PV). On the left side, the brachial vein connects the anastomotic lateral mesencephalic vein (LMV) with the petrosal vein. The precentral cerebellar vein (PcV) and its tributaries (unlabeled arrows) are also evident.

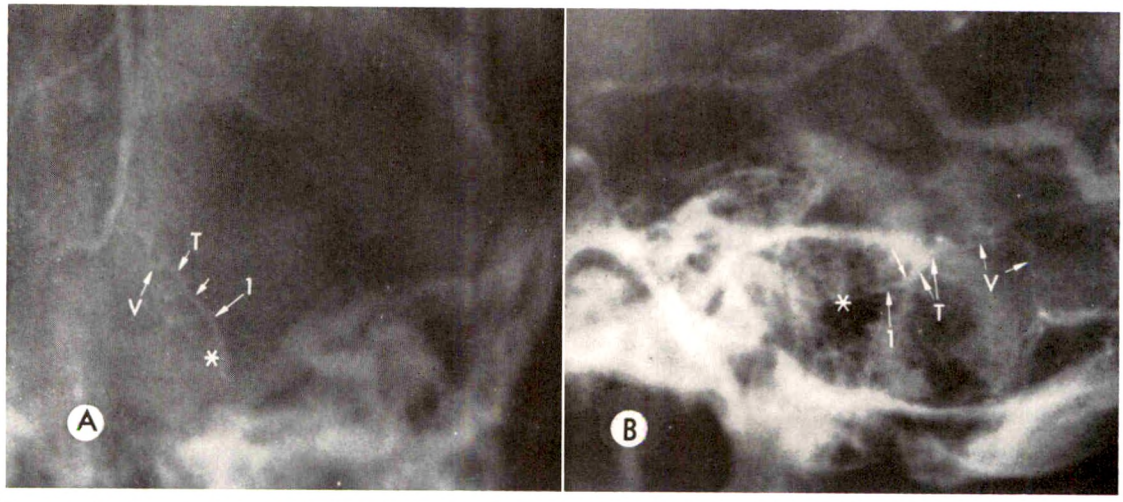



FIG. 14. Drainage of the VLR directly into the inferior petrosal sinus; right brachial angiograms.

(*A*) Towne view. Segment 1 of the VLR continues directly forward in the cistern of the CPM angel (*) to the region of the jugular foramen.

(*B*) Lateral view. The transverse supratonsillar tributary (T) is unusually prominent. Segment 1 of the VLR can be followed to the CPM angle (*) but its further course is obscured by the petrous pyramids.

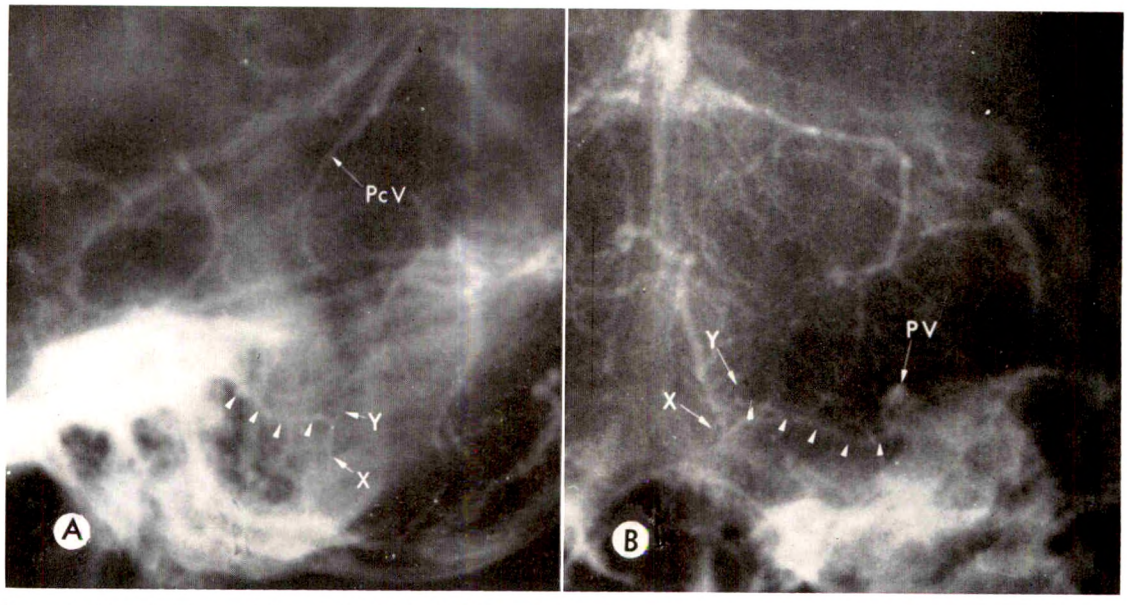



FIG. 15. Prominent medial tonsillar vein; left brachial angiograms.

(*A*) Lateral view. An unusually large medial tonsillar vein (unlabeled arrowheads) runs forward on the medial aspect of the tonsil. It originates by the union of two vertical tributaries (X, Y) running adjacent to the restiform body.

(*B*) Towne view. The medial tonsillar vein (unlabeled arrowheads) runs laterally in a more horizontal plane than a VLR. This vein also turns upward around the flocculus to join the petrosal vein (PV). The vertical tributaries (X, Y) can be faintly seen.

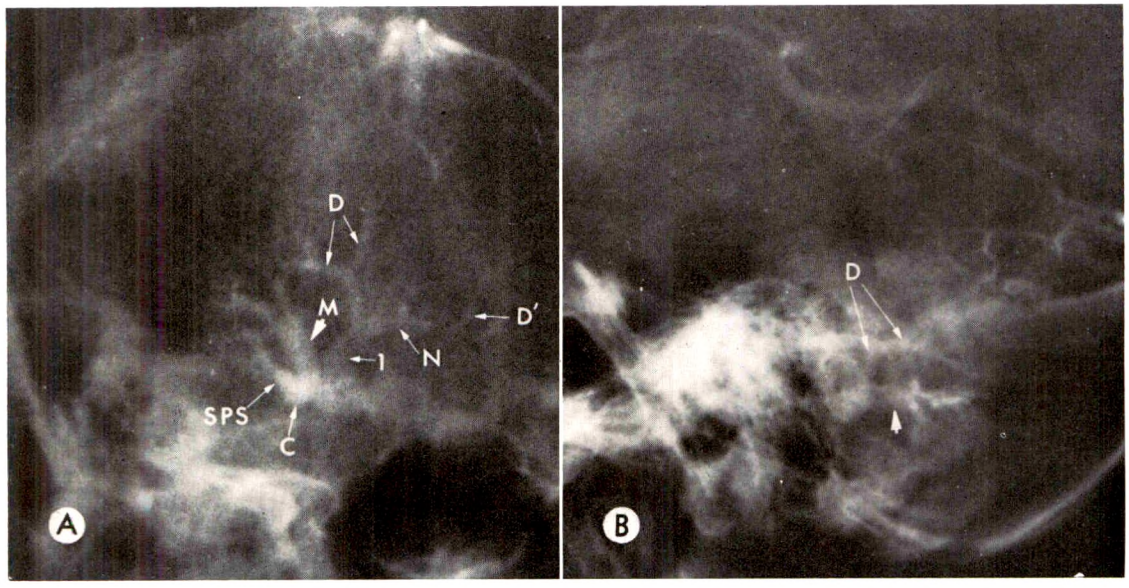



Fig. 16. Extraordinarily large dentate and other parenchymal medullary tributaries of the VLR; right brachial angiograms.

(*A*) Segment 1 is unusually large and receives the deep venous drainage from the dentate nucleus (D) and adjacent white matter. The dentate vein on the left side (D) crosses the midline nodulus (N) to join the right VLR. A huge medullary vein (M) formed by many small and large tributaries joins the distal portion of Segment 1. A short common stem (C) enters the superior petrosal sinus (SPS) directly.

(*B*) Lateral view. The large dentate vein (D) and the huge medullary vein (unlabeled arrow) can be identified. Note the marked paucity of superficial draining veins in the anterior and inferior parts of the posterior fossa. Most of the hemispheric venous drainage appears to be via the deep veins. This patient had no symptoms referable to the posterior fossa.

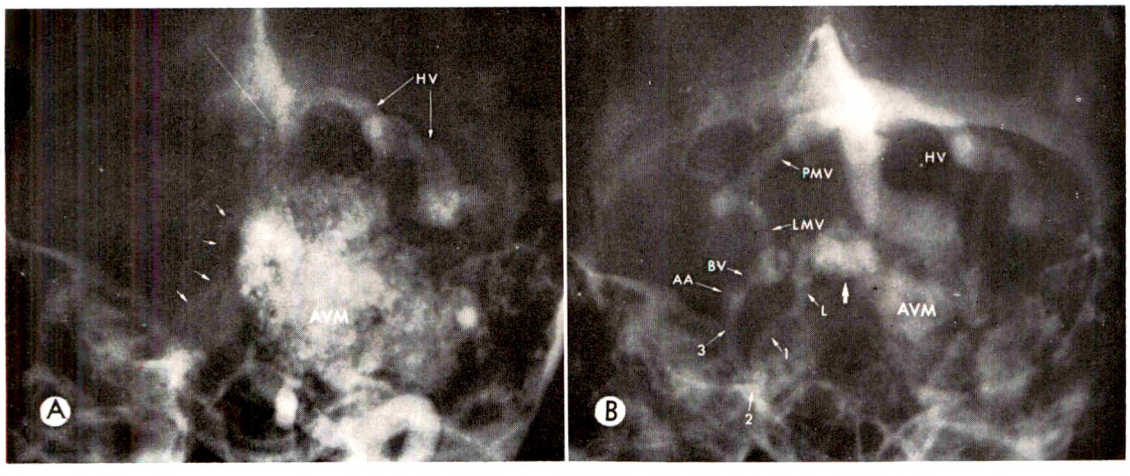



Fig. 17. Arteriovenous malformation of the left cerebellar hemisphere; left brachial angiograms.

(*A*) Towne view—arterial phase. This shows a huge network of intercommunicating dilated vessels (AVM) with premature filling of large superficial cerebellar hemispheric veins (HV). Faint opacification of a deep vein, the VLR (unlabeled arrows), to the right of the midline is also seen.

(*B*) Same examination—2 seconds later. The markedly dilated right VLR (Segments 1, 2 and 3) is now clearly visualized, receiving blood via a large midline channel (unlabeled arrow). It drains in retrograde fashion into the brachial (BV), anastomotic lateral mesencephalic (LMV) and posterior mesencephalic (PMV) veins. The normal position of the lateral supratonsillar tributary (L) and of the first segment (1) of the VLR rules out deformity and displacement of the fourth ventricle; *i.e.*, an expanding lesion can be excluded.

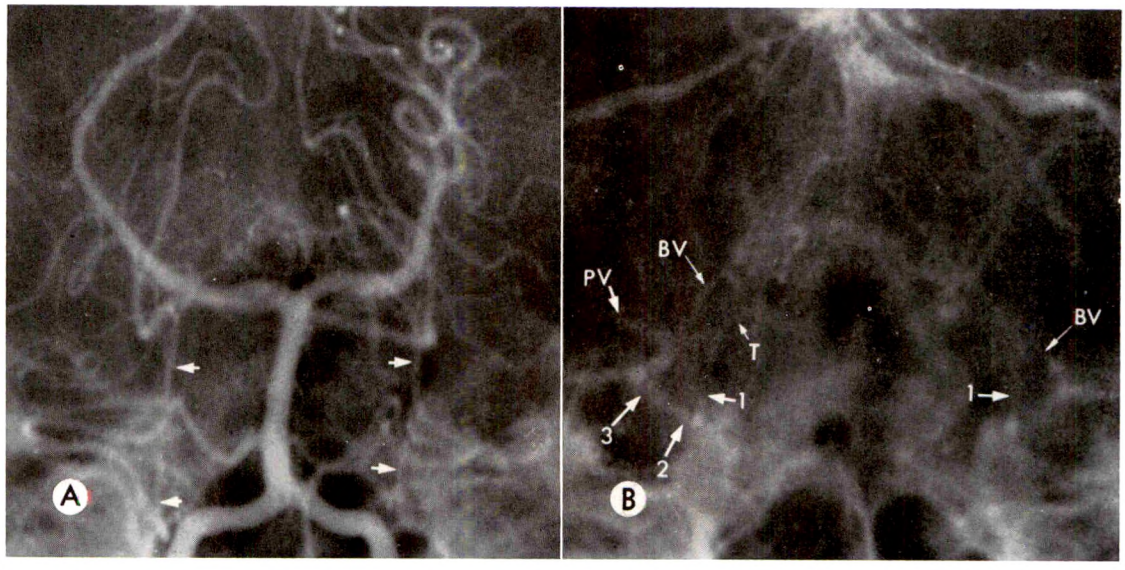



FIG. 18. Papilloma of the choroid plexus of the fourth ventricle; right brachial angiograms.

(*A*) Towne view—arterial phase. Tonsillar branches of the anterior inferior cerebellar arteries are separated and displaced laterally (arrows) indicating the presence of a midline space-occupying lesion.

(*B*) Same examination—venous phase. The first segments (1) of both VLR are symmetrically displaced laterally with increased outward convexity. This causes increased angulation between the first and second segments of the vein and diminution in the distance between the first segment of the vein and brachial tributary (BV) of the petrosal vein (PV). A faintly visualized transverse supratonsillar vein (T) on the right is also laterally displaced. These findings are characteristic of an intraventricular tumor ballooning the fourth ventricle.

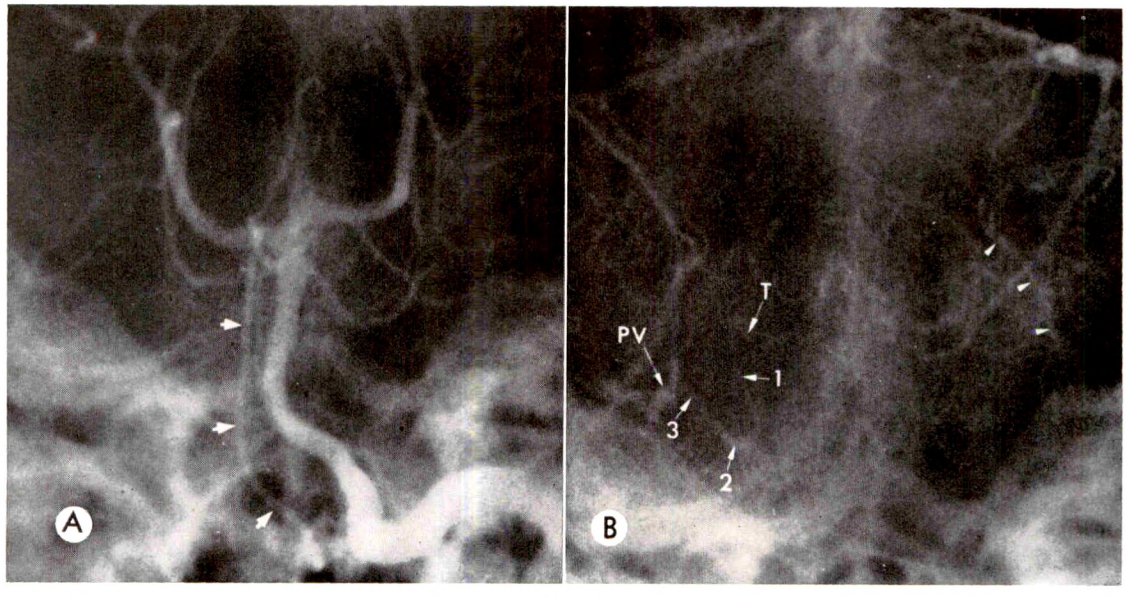



FIG. 19. Ependymoma of the fourth ventricle; left brachial angiograms.

(*A*) Towne view—arterial phase. The only positive finding in the arterial phase is a shift of the left posterior inferior cerebellar artery to the right side (arrows).

(*B*) Same examination—venous phase. The posterior portion of the first segment (1) of the VLR on the right side is displaced laterally in arcuate fashion. A brachial vein on the left side is also displaced and convex laterally. At operation, an ependymoma was found filling the fourth ventricle, extending laterally, and appearing in the cerebello-pontine angle on the left side.

relationships are described in detail and correlated with air studies.

4. The diagnostic value of the VLR is briefly demonstrated by illustrations of an arteriovenous malformation and tumors of the fourth ventricle.

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# FURTHER OBSERVATIONS ON THE MENINGEAL BRANCH OF THE LACRIMAL ARTERY*

## FOUR ADDITIONAL CASES

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IN A previous communication[4] arteriographic evidence of a meningeal branch of the lacrimal artery (a branch of the ophthalmic artery) was described by one of us in a case of pterional meningioma fed by an unusually large vessel originating from the latter artery. To our knowledge, such a finding had not been reported previously, although several publications had already appeared concerning the angiographic features of dural circulation (both in normal and pathologic conditions) or normally thin arterial branches that become visible at carotid angiography only under certain pathologic circumstances. An example of this is the Bernasconi and Cassinari artery, which can be visualized in cases of meningiomas involving the tentorium or the falx-tentorium angle.[3] A review of our total series of meningiomas of the sphenoid wing, of the pterion and of the frontal-temporal-parietal convexity failed to reveal at that time any other similar angiographic findings, thus confirming the rarity of the observation. A brief discussion of the anatomy and possible variations of the meningeal circulation, as well as of its collateral circulation, was presented.

In recent years, other similar cases have been reported[1,2,6] and the meningeal branch of the lacrimal artery has now become a well established neuroradiologic entity. We have had the opportunity of observing 4 additional cases, in which this vessel was visualized at arteriography, always under pathologic circumstances; and we would like to report them, both as a further casuistic contribution to this subject and as an expansion of our original report.

### REPORT OF CASES

CASE I. A.M., a 47 year old woman complained of a hard and tender swelling in the right temporal region, with a progressive increase in size for the past 4 years. Neurologic examination revealed only a mild bilateral papilledema. Plain skull roentgenograms showed an extensive thickening and increased density of the anterior temporal squama and of the pterion, including the outer third of the sphenoid wing. Right common carotid arteriography was performed (Fig. 1, *A* and *B*). A roentgenologic diagnosis of pterional meningioma "en plaque" with reactive hyperostosis was made and confirmed by gross operative and histologic findings. During the operation a large arterial branch apparently originating from the orbital fissure and coursing into the neoplastic mass was found and clipped off.

CASE II. C.D., a 50 year old woman complained for 6 months of left frontal headache and convulsive seizures, "grand mal" in type. On examination, she showed marked mental impairment, initial papilledema and loss of sight in the left eye (4/10). There was loss of the left corneal reflex and of the superficial sensation in the upper division of the left trigeminal nerve; and she had slight right hemiparesis. Plain skull roentgenograms demonstrated an increase of the diploic vascular channels and of the density of the left greater sphenoidal wing. A left common carotid arteriogram (Fig. 2, *A* and *B*) showed the signs of a large space-occupying lesion located at the pterion, with a pathologic stain apparently fed by a large, tortuous branch of the ophthalmic artery. The roentgenologic diagnosis was meningioma of the greater sphenoidal wing and of the pterion. At surgery, a large, richly vascularized meningioma, fed by some twigs from the middle meningeal artery but mainly

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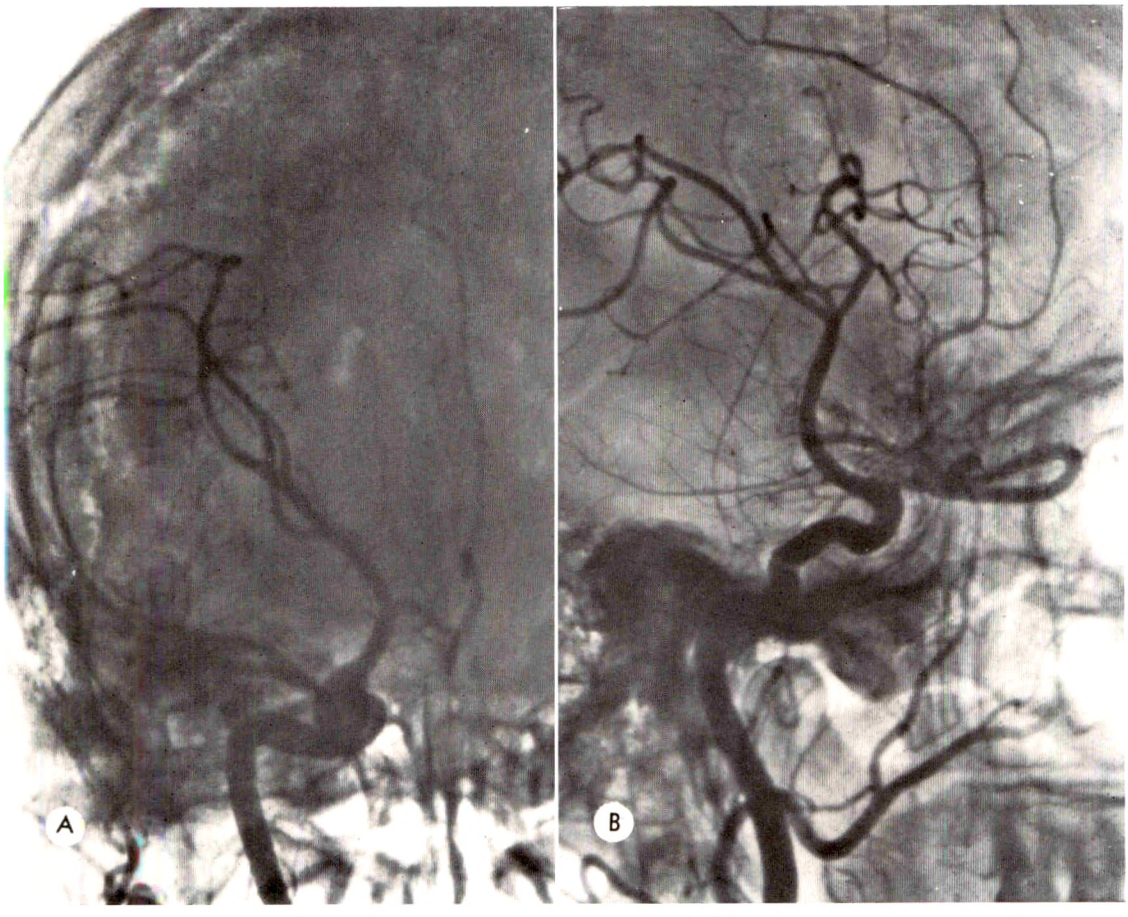



Fig. 1. Case 1. (*A* and *B*) Right common carotid arteriography. Slight shift of the anterior cerebral artery, inwards displacement of the insular vessels with reduction of the M/1–2 angle. A large and tortuous vessel can be seen running from the initial part of an enlarged ophthalmic artery, along the sphenoid ridge; entering the lateral corner of the orbital fissure and ending against the bony thickening at the pterion.

by a large artery coursing along the sphenoidal ridge, was found and completely removed.

Case III. L.Z., a 43 year old woman, was admitted with a history of progressive mental impairment and convulsive seizures of few months' duration. On examination, she showed moderate right exophthalmos and tenderness in the right frontal-temporal region; initial papilledema and a left pyramidal deficit. On plain skull roentgenograms moderate increase of density and thickening at the right pterion, with normal contour and decalcification of the sella turcica were demonstrated. Right common carotid arteriography (Fig. 3, *A* and *B*) gave evidence of a large, extracerebral space-occupying lesion, extending from the pterion anteriorly into the frontal area and posteriorly into the middle cranial fossa, with a pathologic stain fed by a large, tortuous arterial vessel originating from the ophthalmic artery and coursing along the sphenoidal ridge. At operation, a large pterional meningioma was completely removed, together with its dural attachment. The large meningeal artery seen at arteriography, entering into the neoplastic mass after a course along the sphenoidal ridge, was occluded.

Case IV. V.M., a 63 year old woman, was admitted with a complaint of exophthalmos and progressive loss of vision (up to complete blindness) on the right side. She had swelling and tenderness in the right temporal region, polyuria and undue increase in body weight. On physical examination, there was a tender, hard swelling in the right temporal and pterional region; right exophthalmos with

deviation of the eye downwards and outwards; complete blindness and primary optic atrophy in the right eye. Plain skull roentgenograms showed extensive thickening and increased density of the right sphenoidal wing and pterion with narrowing of the upper orbital fissure. Right internal carotid arteriography (Fig. 4, *A–C*) revealed a meningeal branch of the lacrimal artery feeding a pathologic stain, and displacement of the middle cerebral vessels.

The patient refused surgery and was discharged for further clinical follow-up.

## DISCUSSION

The arteriographic findings obtained in these 4 cases confirm our first description of this unusual and tortuous meningeal vessel, belonging to the territory of the ophthalmic artery circulation, and its pathologic nature. In all cases, a meningioma of the pterion with massive involvement of the dura was found. These richly vascular lesions are fed mainly by the meningeal branch of the lacrimal artery, which is constantly of larger size than its equivalent under normal conditions, which usually cannot be recognized at arteriography.

Visualization of the external carotid circulation by means of common carotid arteriography has allowed the constant demonstration of a normal middle meningeal artery and all its branches. Thus, in our opinion, the main reason for the pres-

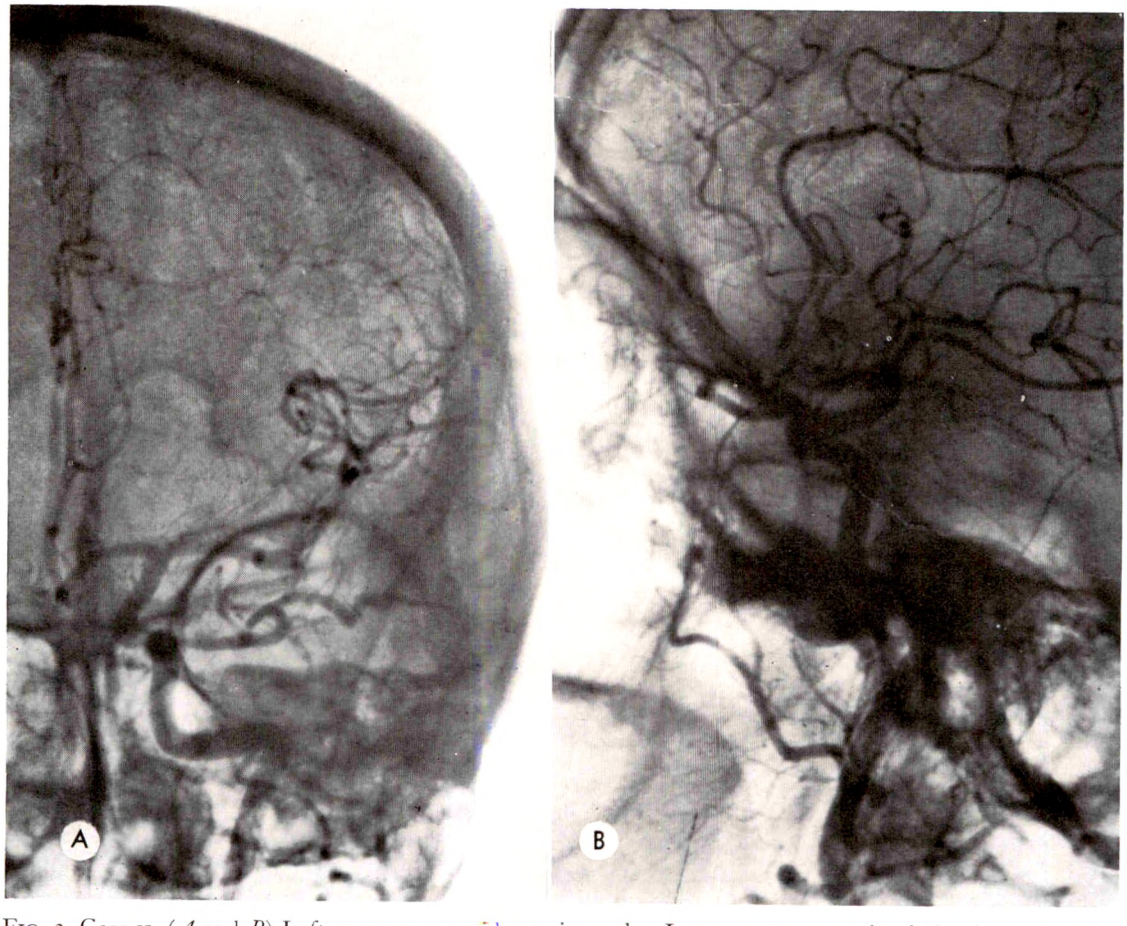



FIG. 2. Case II. (*A* and *B*) Left common carotid arteriography. Large space-occupying lesion located at the pterion, with pathologic stain fed by a large and tortuous artery originating from the ophthalmic artery. Marked upwards displacement of the M/1 tract of the middle cerebral artery and of the sylvian vessels; inwards displacement of the carotid syphon. The artery, because of the supraimposition of the petrous bone structures in the anteroposterior view (*A*), can be better identified in the lateral view (*B*).




Fig. 3. Case III. (*A* and *B*) Right common carotid arteriography. There is evidence of a large, extracerebral space-occupying lesion, extending from the pterion anteriorly into the frontal area and posteriorly into the middle cranial fossa, with a pathologic stain fed by a large, tortuous arterial vessel originating from the ophthalmic artery and running along the sphenoidal ridge.

ence and pathologic character of the artery under discussion should be ascribed to the peculiar location of the neoplasm, corresponding to the anatomic distribution of the artery, rather than to some function of the collateral circulation producing vicarious absence of the middle meningeal vessels, as it has been maintained by some authors.[1,6]

In all 4 cases reported here the origin, morphologic characters and course of this meningeal branch of the lacrimal artery were in accordance with our original description. Under these pathologic conditions, the artery is easily recognized because of its large size, tortuosity and course along the sphenoidal wing. Should it be obscured in the standard anteroposterior or lateral angiographic views because of superimposition of bony or vascular structures, it can be easily brought into evidence with an additional anteroposterior view using a more oblique direction of the roentgen-ray beam, so as to obtain a better visualization of the lesser and greater sphenoidal wings (Fig. 4, *A–C*).

From a surgical point of view, arteriographic evidence of this artery and of the pathologic stain it feeds are important because of the habit, common to some neurosurgeons, of ligating the external carotid artery prior to removal of meningiomas with a rich vascularization and a wide dural attachment.

It might be presumed that the presence of this meningeal branch of the lacrimal

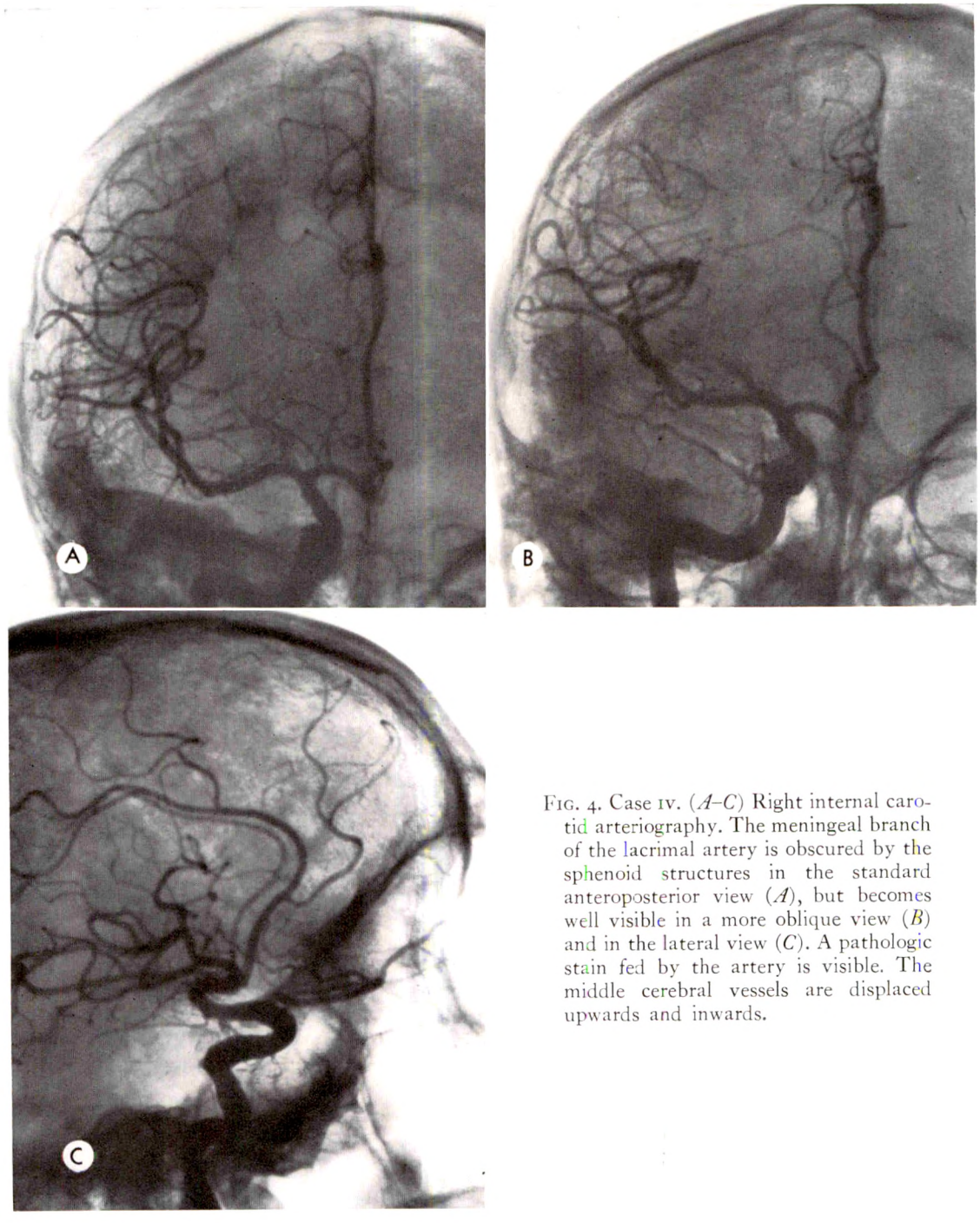



FIG. 4. Case IV. (*A–C*) Right internal carotid arteriography. The meningeal branch of the lacrimal artery is obscured by the sphenoid structures in the standard anteroposterior view (*A*), but becomes well visible in a more oblique view (*B*) and in the lateral view (*C*). A pathologic stain fed by the artery is visible. The middle cerebral vessels are displaced upwards and inwards.

artery, with all morphologic characters described herein, is suggestive of the nature of the neoplasm (*i.e.*, meningiomas); however, we believe that the artery may also be present in cases of neoplasms of a different nature, provided that an extensive involvement of the pterional dura takes place, similarly to what has already been described for the Bernasconi and Cassinari artery.[3]

SUMMARY

Four additional cases of pterional meningiomas, fed by a meningeal branch of the lacrimal artery, are reported.

The morphology, course, characters and pathologic significance of this vessel are discussed.

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# ANGIOGRAPHIC DEMONSTRATION OF DURAL BRANCHES OF THE INTERNAL CAROTID ARTERY IN SPHENOID RIDGE MENINGIOMAS*

*By* JYOJI HANDA, M.D., HARUHIKO KIKUCHI, M.D., *and* HAJIME HANDA, M.D.
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THE extracranial portion of the internal carotid artery does not usually give rise to branches in man. On entering the skull base through the carotid canal, however, the *ramus carotido- tympanicus* and the *ramus canalis pterygoideus* branch off from the internal carotid artery.[11] In the cavernous portion of the artery, several tiny branches of the internal carotid artery, which supply the cerebellar tentorium, gasserian ganglion, third and fifth cranial nerves, internal auditory apparatus, pituitary gland and the dura covering the clivus, cavernous sinus and neighbouring structures, have been described in detail in the embryologic and anatomic studies by DeLa Torre and Netsky,[4] Schnürer and Stattin[15] and others.

Although such branches of the internal carotid artery are so small that they cannot be visualized roentgenographically under usual conditions, it has been reported that the vessels sometimes become enlarged and opacified by angiography (1) as feeding vessels when there exists a pathologic process with rich vascular bed in their territory of supply, or (2) as collateral routes in cases of carotid occlusive disease.

For example, tentorial branches of the internal carotid artery, namely the *rami tentorii basalis et marginalis* of Schnürer and Stattin[15] or the *arteria tentorii* of Krayenbühl and Yaşargil,[11] become enlarged and are visualized by angiography in various disease processes such as tentorial-falcotentorial meningiomas, arteriovenous malformations and angiomas in the tentorial region, glioblastomas in the temporobasal region and others.[1–3,7–11,16–18]

In 1965, Doron and Schwartz[5] reported the cases of malignant pituitary tumors in which the abnormal feeding arteries and/or tumor stain were observed. In 1966, Rosen, Hanafee and Nahum,[14] using selective internal and external carotid arteriographies, demonstrated the contribution of the internal carotid artery to the blood supply of the nasopharyngeal angiofibroma.

When thrombosis occurs in the cervical portion of the internal carotid artery, collateral circulation is established from the contralateral internal carotid artery via the anterior cerebral-anterior communicating arteries, from the vertebral-basilar system via the posterior communicating artery, and from the external carotid artery. The ophthalmic artery is the best known of those anastomotic channels between the internal and external carotid arteries; however, there exist also the collateral routes by way of the caroticotympanic arteries.[6] Recently, Minagi and Newton[12] described a case of the hypoplastic internal carotid artery, in which the rete mirabile was angiographically demonstrated between the internal and external carotid arteries at the base of the skull.

The purpose of the authors is to report 4 cases of sphenoid ridge meningiomas in which the branches of the ganglionic and cavernous portions of the internal carotid artery were enlarged and opacified by carotid angiography.

## REPORT OF CASES

CASE 1. M.I., a 65 year old female, was admitted to our clinic with 4 years' history of proptosis and blurred vision on the right side and neuralgic pain associated with paresthesia in the right half of the face. Six months prior

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to admission, diminished vision of the left eye developed. Neurologic examination on admission disclosed bilateral blindness, proptosis, external ophthalmoplegia and adiadochokinesis on the right side.

Right carotid angiograms were compatible with a space-occupying mass in the right anterior temporal region. In the frontal projection (Fig. 1) the supraclinoid portion of the internal carotid artery was stretched; the horizontal part and knee of the middle cerebral artery were stretched, deformed and elevated; and the draping of the temporal arteries was noted. A small vessel branched off from the internal carotid artery proximal to the ophthalmic artery. The vessel coursed laterally and upward for approximately 10 mm., then turned downward and tapered off showing a beaded appearance.

In the lateral view the supraclinoid portion of the internal carotid artery was stretched; the trifurcation was elevated; the sylvian triangle was deformed and elevated, particularly in its middle one-third; and the draping of the temporal arteries was remarkable. An abnormal vessel, branching off from the ganglionic portion of the internal carotid artery, coursed posteriorly and was seen to supply a fine net of tumor vessels located posterior to the carotid siphon.

The anterior cerebral arteries were not contrast-filled either in the frontal or lateral projections.

A solid tumor, 43 gm. in weight, was removed and the patient's recovery was uneventful.

The histologic diagnosis was meningioma, apparently arising from the sphenoid ridge.

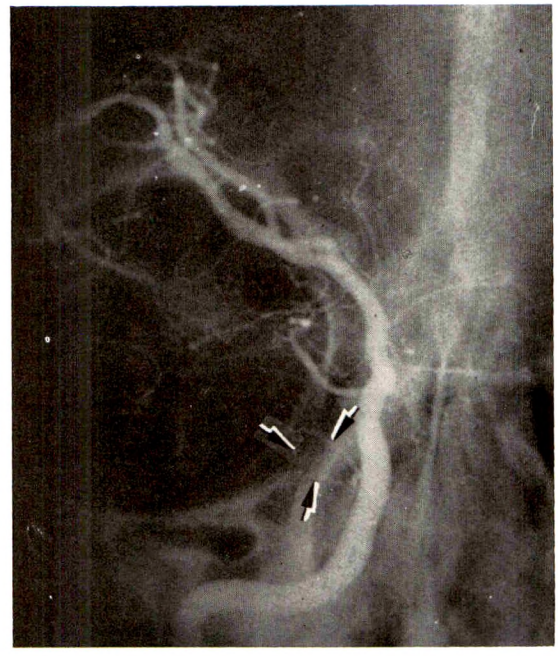


Fig. 1. Frontal projection of right carotid angiogram in Case 1. Note the opacification of the dural branches (arrows) of the internal carotid artery.

Case II. E. F., a 39 year old female was admitted to our clinic complaining of headache and progressive visual disturbances on both sides of 6 months' duration. On admission, positive neurologic findings were bilateral choked disks, loss of vision on the left side, and temporal hemianopia of the right eye.

Left carotid angiography was performed. In the frontal projection, the anterior cerebral artery, which was filled only late in the arterial phase, showed a parallel shift across the midline. The middle cerebral group was stretched and markedly elevated, and the temporal arteries showed a draping sign. A tumor stain, 45×50 mm. in diameter, superimposed in the orbita, and was supplied by an enlarged ophthalmic artery. A small abnormal artery, approximately 2 mm. in diameter, branched off from the internal carotid artery proximal to the ophthalmic artery, coursed laterally and reached the tumor stain (Fig. 2*A*).

In the lateral view, the trifurcation of the internal carotid artery was displaced anteriorly, the middle cerebral artery and the sylvian triangle were deformed and elevated. A tumor stain was disclosed in the anterior temporal region, and a faint stain remained throughout the venous phase of the angiograms.

Total removal of a meningioma (37 gm.) arising from the left sphenoid ridge was performed, and the postoperative course was uneventful.

Control postoperative angiograms (Fig. 2*B*) were essentially normal showing neither tumor stain nor shift of the anterior cerebral artery. The abnormal branch of the internal carotid artery was not contrast-filled.

Case III. F.T., a 17 year old male, had a 1 year history of headache, convulsive seizures and visual disturbances of the left eye. Six months prior to admission to our clinic right hemiparesis developed. On admission a neurologic examination disclosed bilateral choked

disks, left upper nasal quadrantanopia and right spastic hemiparesis.

The frontal projection of the left carotid angiogram disclosed stretching and medial displacement of the supraclinoid portion of the internal carotid artery (Fig. 3*A*). The pericallosal artery showed a shift across, and parallel to, the midline. The angle between the $A_1$ and the pericallosal artery was preserved. The horizontal portion of the middle cerebral artery was stretched and elevated, the knee of the middle cerebral artery was deformed and displaced supero-medially. The marked boxing, loss of normal undulations and stretching of the medial branches, and the draping of the temporal arteries were noted. A round, abnormal hypervascularization was demonstrated in the left orbita measuring 40×50 mm. in diameter, and a fine vessel, which branched off from the cavernous portion of the internal carotid artery, first coursed medially, but then turned laterally, and seemed to join the round configuration of tumor vessels.

In the lateral view (Fig. 3*B*) this vessel arose from the proximal end of the cavernous portion of the internal carotid artery. Otherwise the findings were compatible with a mass lesion in the anterior temporal region.

Craniotomy was performed, a huge tumor (60 gm.) was removed, and a cyst (30 cc.) was evacuated.

The histologic examination revealed a myxomatous meningioma.

The postoperative course was uneventful.

CASE IV. N.H., a 37 year old male was admitted to our clinic complaining of 6 months' history of visual field defects. Positive neurologic findings on admission were right homonymous hemianopia, mydriasis and sluggish light reaction of the left pupil. The fundi were normal.

Plain roentgenograms of the skull disclosed destruction of the medial part of the left sphenoid ridge.

Left internal carotid arteriography was performed. In the frontal projection the supraclinoid portion of the internal carotid artery and the $M_1$ were displaced medially; the $M_2$ was displaced medially and superiorly describing a curve concave inferolaterally; and the knee of the middle cerebral artery was markedly elevated and deformed. The anterior cerebral arteries were not filled.

A tumor stain, 75×60 mm. in diameter, was demonstrated, its center being at the medial

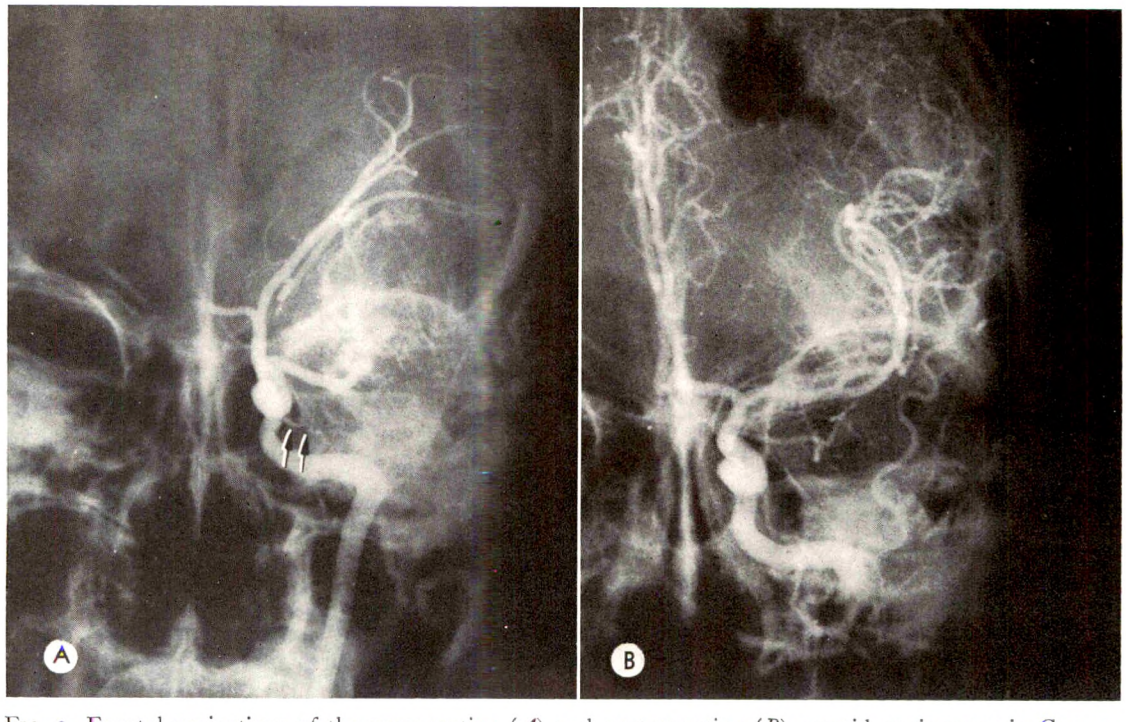



FIG. 2. Frontal projections of the preoperative (*A*) and postoperative (*B*) carotid angiograms in Case II. Note the demonstration of the dural branches (arrows) and tumor stain in *A* and their disappearance in *B*.

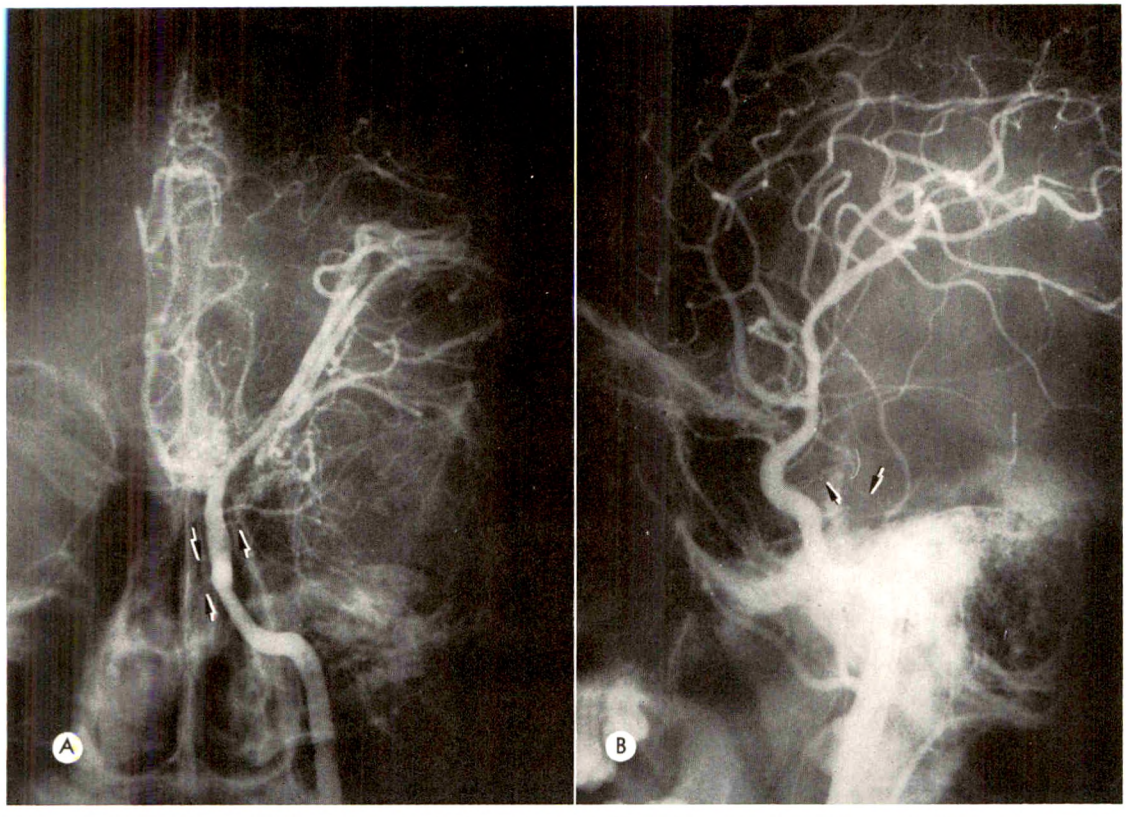



FIG. 3. Frontal (*A*) and lateral (*B*) projections of the left carotid angiogram in Case III. Note the dural rami (arrows) of the internal carotid artery.

one-third of the sphenoid ridge. The tumor cloud was homogeneous, maximum in density in the late arterial phase and remained contrast-filled throughout the venous phase.

In the lateral projection the supraclinoid part of the internal carotid artery was narrowed, and its margin irregular. The $C_1$, $M_1$ and $M_2$ were stretched and elevated, the sylvian triangle was deformed and markedly displaced superiorly. The anterior cerebral and posterior communicating arteries were not filled and the anterior choroidal artery was stretched and elevated.

The tumor stain, 70×50 mm. in diameter, was demonstrated in the anterior middle fossa, the center lying around the carotid trifurcation. The enlarged ophthalmic artery supplied the anterior part of the tumor stain, and small vessels arose from the cavernous and ganglionic portions of the internal carotid and joined the tumor stain (Fig. 4*A*).

Right carotid angiograms showed a negative shift of the anterior cerebral arteries, and the medial part of the tumor stain was contrast-filled. The tumor stain was also evident in the selective left external carotid arteriogram (Fig. 4*B*).

## COMMENTS

In Cases I through III, the clinical diagnosis of meningioma, arising from the sphenoid ridge, has been confirmed by operation and histologic examination. Although verification at surgery or by histology is lacking in Case IV, the diagnosis of sphenoid ridge meningioma is beyond doubt when one considers the characteristic roentgenologic findings. It by no means follows, however, that the angiographic demonstration of the dural branches of the internal carotid artery is pathognomonic of meningioma.

Significance of opacification of these vessels appears to be analogous to that of the artery of Bernasconi and Cassinari in tentorial meningiomas and other highly vascularized pathologic processes in the ten-

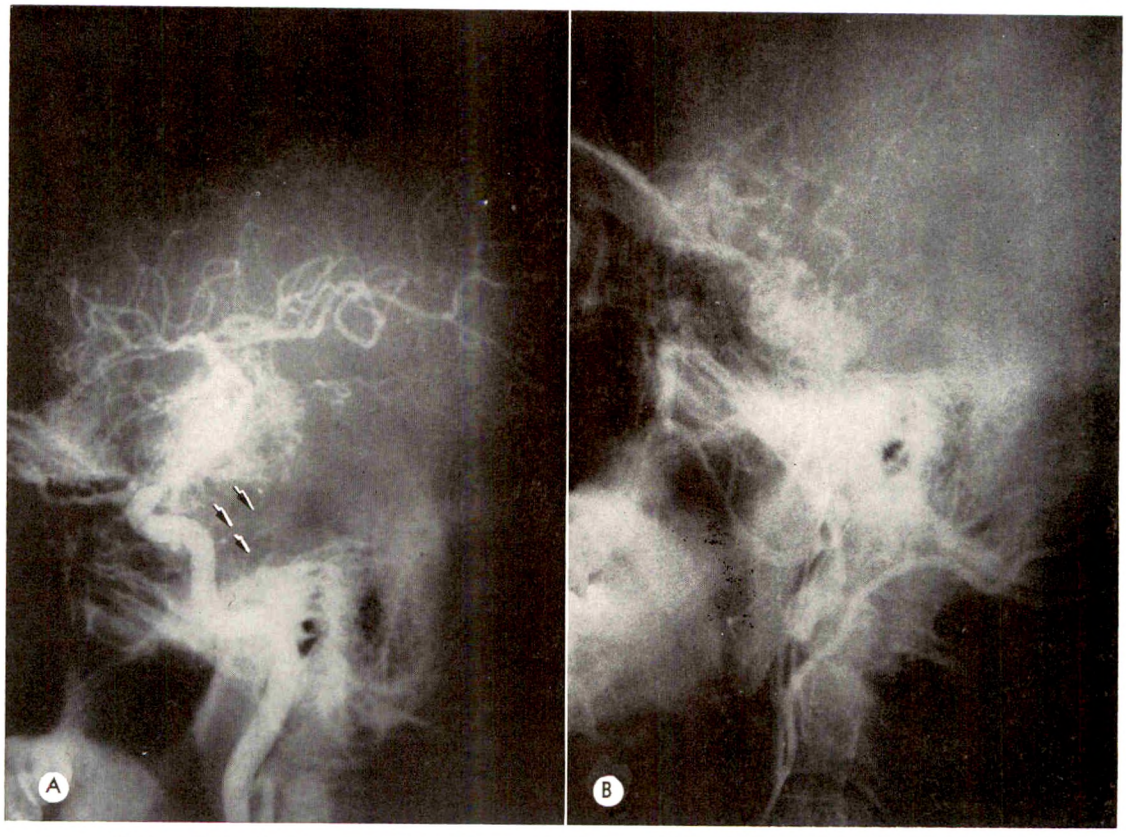



FIG. 4. Selective internal (*A*) and external (*B*) carotid arteriograms in Case IV. The meningeal rami (arrows) were contrast-filled in *A*.

torial region in the sense that the enlargement and subsequent contrast-filling of such tiny vessels are determined essentially by location and enhanced demand for blood supply of the pathologic process and not by the type of pathology of the lesion. Further detailed studies of the angiograms in patients with other kinds of tumors or vascular lesions in the same location are necessary before any conclusion regarding the diagnostic value of the visualization of these dural branches of the internal carotid artery can be drawn.

It should be noted, however, that the demonstration of dural branches in sphenoid ridge meningioma indicates the dural attachment of the tumor and that the preoperative knowledge of the location and size of the dural attachment in meningiomas greatly favors operative procedures.

SUMMARY

Four cases of meningiomas arising from the sphenoid ridge are reported. In all 4 cases enlarged dural branches of the cavernous or ganglionic portion of the internal carotid artery were contrast-filled, and in 3 cases these vessels seemed to have supplied the tumor stain.

Visualization of these vessels seems to indicate the location of the pathologic process with rich vascular supply and is not necessarily pathognomonic of the sphenoid ridge meningioma; however, the preoperative confirmation of the dural attachment of the tumor greatly favors operative procedures.

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# THE CAVERNOUS BRANCHES OF THE INTERNAL CAROTID ARTERY

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*"We only see what we look for,*
*And look for only what we know."*

THE cavernous portion of the internal carotid artery, although usually depicted without tributaries, supplies branches to the dura at the base of the skull, the cavernous sinus, the sella turcica and its contents, and the tentorium. These vessels communicate with their mates of the opposite side and with meningeal branches of the external carotid, ophthalmic, and vertebral arteries. This freely anastomosing network at the base of the skull comprises a portion of the rete mirabile, analogous to that found in animals. It is the purpose of the authors to demonstrate angiographically the cavernous branches of the carotid artery and to determine the significance of their opacification in health and disease. This presentation of 50 cases includes a variety of lesions supplied by these arteries.

Within the cavernous sinus, the internal carotid artery is situated between layers of the dura mater forming the sinus. Its entrance into the cavernous sinus is frequently demarcated by a band-like impression of the dura. The initial portion of the S-shaped cavernous carotid artery ascends toward the posterior clinoid process forming, then, a convexity upwards. The relatively horizontal portion, approximately 2 cm. in length, is terminated by a concavity upward on the medial aspect of the anterior clinoid process where the artery perforates the dura mater of the roof of the sinus. Luschka (1860), McConnell (1953),[11]

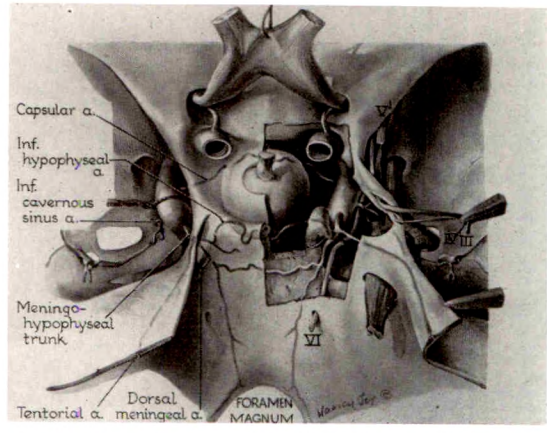



FIG. 1. Normal anatomy. (Dissection of the cavernous portion of the internal carotid artery done by Parkinson. Permission for reproduction by Dr. Dwight Parkinson and *Canad. J. Surg.*[13])

Schnürer and Stattin (1963),[15] Parkinson (1964)[13] and others presented detailed dissections of the cavernous portion of the internal carotid artery, establishing the existence of these branches in the normal. Slightly different terminology was utilized by each; this is summarized in Table 1. Parkinson's anatomic investigation of the distribution of the branches of the cavernous segment of the carotid artery in 200 cadavers forms the basis for our observations (Fig. 1).

### A. MENINGOHYPOPHYSEAL ARTERY

The meningohypophyseal artery (dorsal main stem) is the most proximal branch arising from the posterior aspect of the initial curve of the cavernous carotid and existed in 100 per cent of Parkinson's dissections. This major trunk divides shortly

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after its take off into three subdivisions of equal caliber: (1) tentorial, (2) dorsal meningeal, and (3) inferior hypophyseal.

Schnürer and Stattin[15] noted that small vessels in the neighborhood of the siphon are visible in the lateral projection of most angiographic examinations. Figure 2 demonstrates the meningohypophyseal artery in an otherwise normal study.

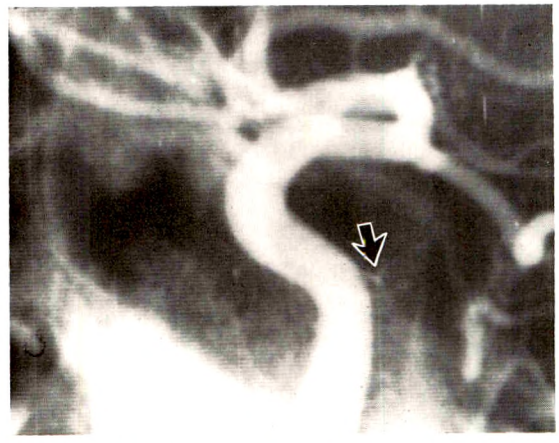


Fig. 2. Meningohypophyseal artery (↑) in an otherwise normal carotid arteriogram.

### 1. TENTORIAL ARTERY

The tentorial branch (basal tentorial branch) is directed posteriorly and laterally. In the cavernous sinus, branches are given off to the III and IV nerves, to the roof of the cavernous sinus and anastomose with the meningeal branches of the ophthalmic artery. The tentorial vessel then runs along the free margin of the tentorium supplying the tentorium, the falx, and communicating posteriorly with the tentorial branch of the opposite side.

Table I

ANATOMIC CLASSIFICATION OF CAVERNOUS BRANCHES OF CAROTID ARTERY

| Parkinson (1964)[13] | Schnürer and Stattin (1963)[15] | McConnell (1953)[11] |
|---|---|---|
| A. Meningohypophyseal Artery | A. Dorsal Main Stem | A. Inferior Hypophyseal Trunk |
| 1. Tentorial branch | 1. Basal tentorial branch | |
| a. Branch to III and IV | | |
| b. Branch to roof of cavernous sinus | | |
| c. Branch to falx and tentorium | | |
| 2. Dorsal meningeal branch | 2. Clival branches | —posterior branch |
| a. Clival branch | | |
| b. Branch to VI | | |
| 3. Inferior hypophyseal branch | | |
| a. Branch to dura at floor of sella | | —inferior hypophyseal branch to posterior hypophysis |
| b. Branch to dura of post clinoids | | |
| c. Branch to cavernous sinus | | |
| B. Inferior Cavernous Sinus Artery | B. Lateral Main Stem | —anterior branch from inferior hypophyseal trunk |
| 1. Branch to gasserian ganglion | 1. Marginal tentorial branch | |
| 2. Anastomosis with middle meningeal and accessory meningeal arteries | 2. Branch to gasserian ganglion IV, V, VI | |
| C. Capsular Arteries | 3. Anastomosis with branch of middle meningeal and branch of ophthalmic arteries | C. Capsular |
| | | 1. Anterior capsular |
| | | 2. Inferior capsular from inferomedial aspect of internal carotid artery halfway along course |

Bernasconi and Cassinari (1956) first reported the angiographic visualization of a tentorial artery supplying tentorial meningiomas. Krayenbühl and Yasargil (1957)[10] demonstrated this vessel in a patient with a subtentorial arteriovenous malformation. In 1958 Wickbom and Stattin[20] recognized that the tentorial artery originated from the cavernous portion of the carotid. Schnürer and Stattin (1963)[15] stated that the tentorial branches also have a characteristic wavy appearance, similar to other dural vessels. They have seen the tentorial artery in the "normal" ranging from 5 mm. to 35 mm. in length; if longer than 40 mm. and prominent, a pathologic lesion was considered probable. This vessel is illustrated in an otherwise normal examination (Fig. 3*A*), in patients with a tentorial meningioma (Fig. 3*B*), and a tentorial arteriovenous malformation (Fig. 3, *C* and *D*). It has also been seen with occlusive vascular disease (Fig. 10, *A–D*) in which it supplies collateral circulation and in 2 cases of invasion of the tentorium by malignant lesions, an ependymoma (Fig. 4*A*), and a glioblastoma (Fig. 4, *B* and *C*). Zimmer[22] has also observed the tentorial branch supplying a metastatic focus involving the tentorium. We have visualized an enlarged tentorial artery in a trigeminal neuroma. This has also been reported by Westberg.[19]

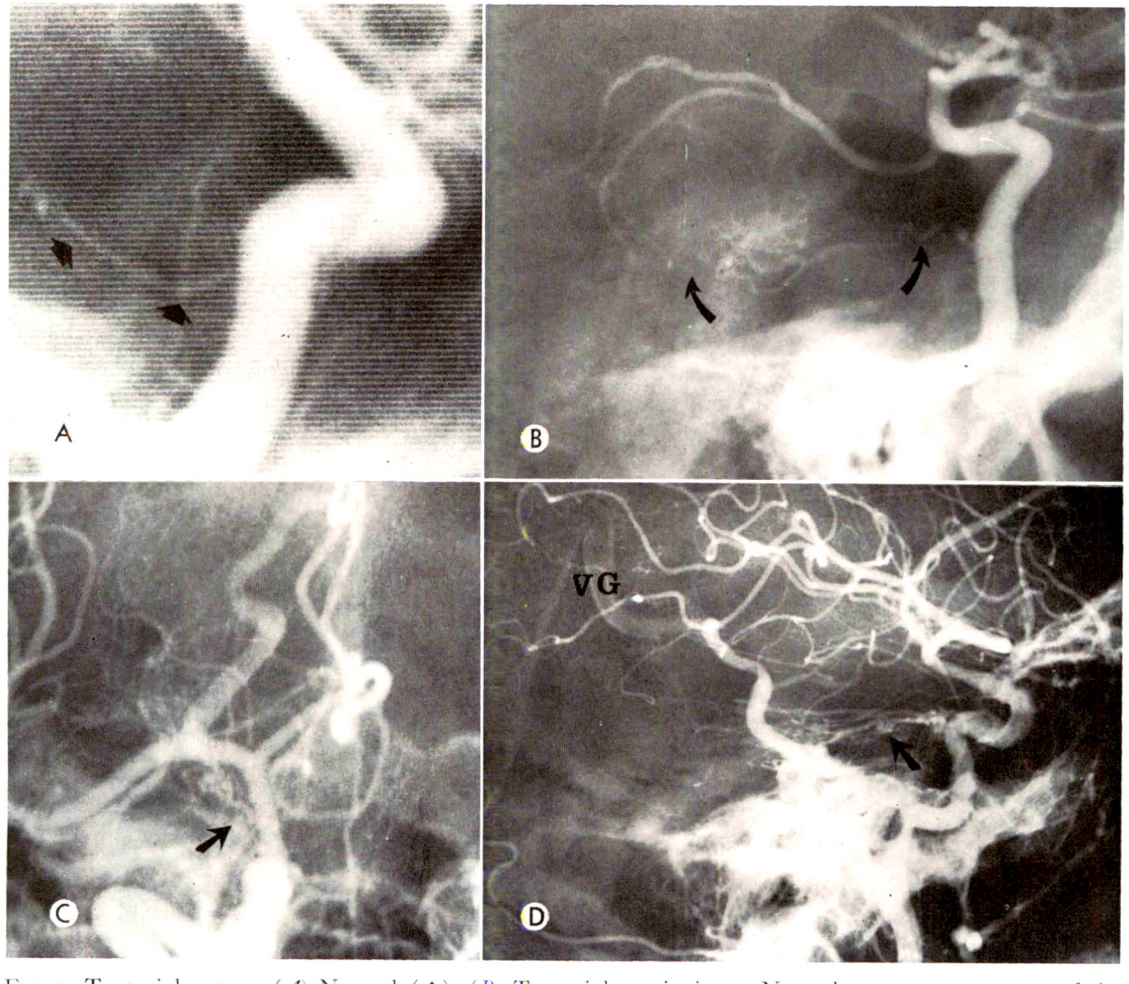



FIG. 3. Tentorial artery. (*A*) Normal (↑). (*B* Tentorial meningioma. Note the wavy appearance of the tentorial artery (↑). A tumor stain is observed. (*C* and *D*) Tentorial arteriovenous malformation. Several tentorial branches (↑) supply an arteriovenous malformation that drains eventually into the great vein of Galen (VG).

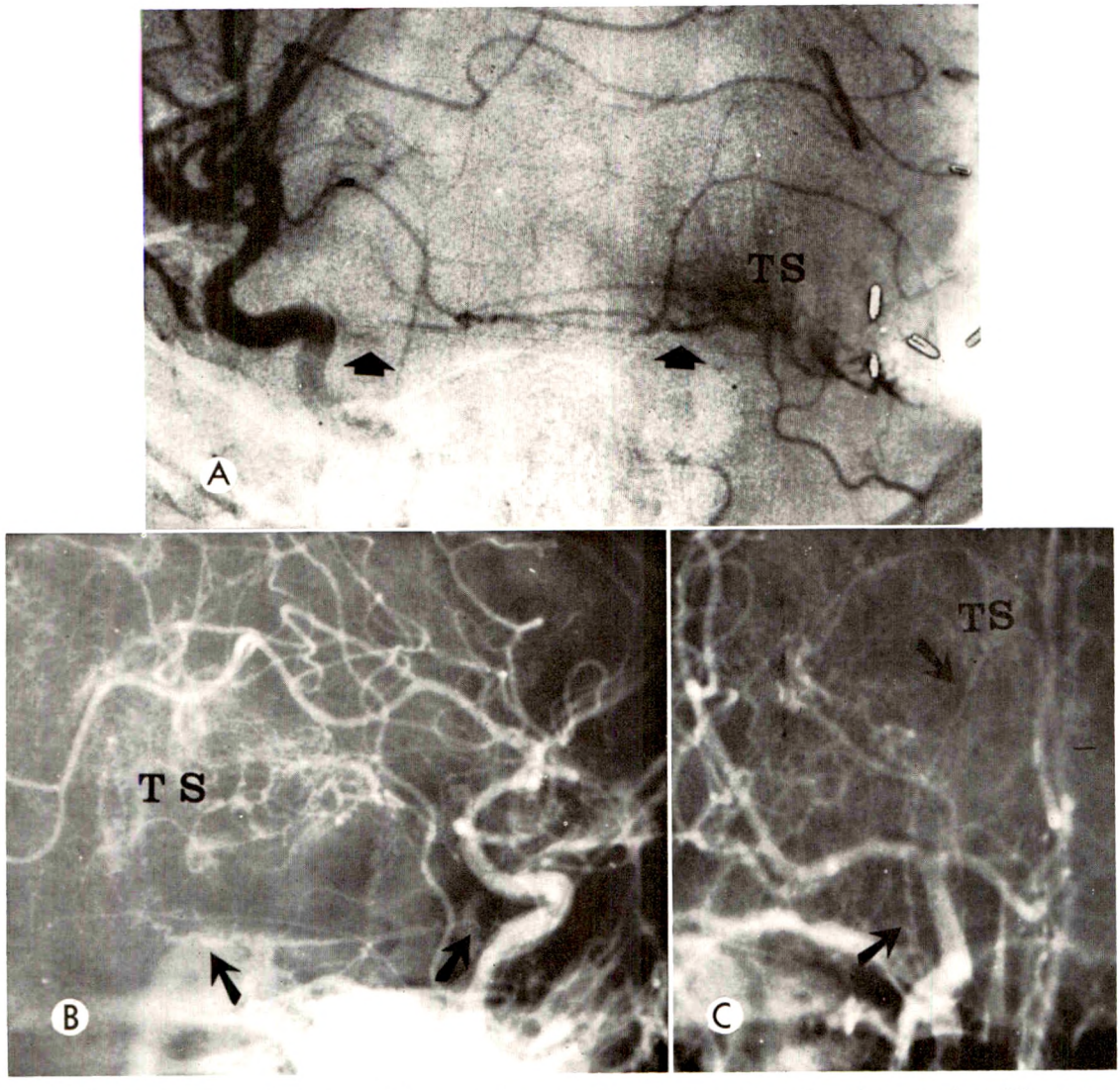



Fig. 4. Tentorial artery. Invasion of the tentorium. (*A*) Ependymoma invading the tentorium. The tumor stain (TS) is supplied by a prominent, stretched and elongated tentorial artery ( ↑ ). (*B* and *C*) Glioblastoma invading the tentorium. The tentorial vessel ( ↑ ) is enlarged and supplies a portion of the vascular stain (TS) seen.

### 2. DORSAL MENINGEAL ARTERY

The dorsal meningeal artery (clival branch) takes a posterior, inferior, medial direction through the cavernous sinus. It then passes down the dorsum and clivus to freely anastomose with the dorsal meningeal of the opposite carotid sending a branch to the VI nerve. The vessel has also been opacified in the normal, in occlusive disease of the internal carotid distal to the cavernous carotid (Fig. 12*D*) and in lesions of the clivus, *e.g.* a chordoma (Fig. 5*A*).

### 3. INFERIOR HYPOPHYSEAL ARTERY

The inferior hypophyseal artery (Luschka and McConnell) projects anteriorly and medially in the posterior sellar floor, supplying the posterior lobe of the pituitary gland and the dura of the floor of the sella, anastomosing with a similar vessel from the opposite side. Branches to the dura of the posterior clinoids and to the cavernous sinus may also be present. The inferior hypophyseal artery has been observed supplying a pituitary adenoma (Fig.

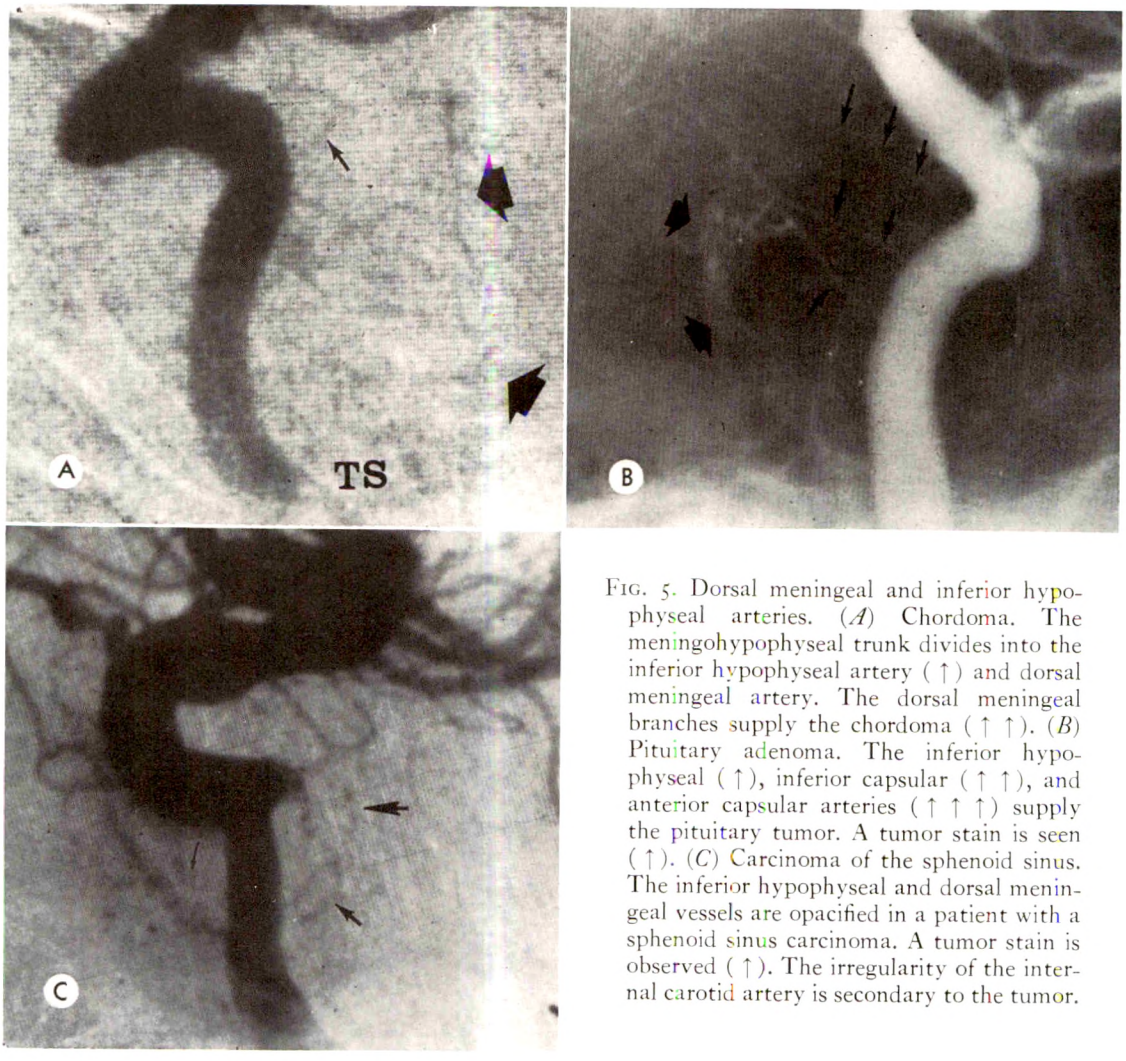



FIG. 5. Dorsal meningeal and inferior hypophyseal arteries. (*A*) Chordoma. The meningohypophyseal trunk divides into the inferior hypophyseal artery ( ↑ ) and dorsal meningeal artery. The dorsal meningeal branches supply the chordoma ( ↑ ↑ ). (*B*) Pituitary adenoma. The inferior hypophyseal ( ↑ ), inferior capsular ( ↑ ↑ ), and anterior capsular arteries ( ↑ ↑ ↑ ) supply the pituitary tumor. A tumor stain is seen ( ↑ ). (*C*) Carcinoma of the sphenoid sinus. The inferior hypophyseal and dorsal meningeal vessels are opacified in a patient with a sphenoid sinus carcinoma. A tumor stain is observed ( ↑ ). The irregularity of the internal carotid artery is secondary to the tumor.

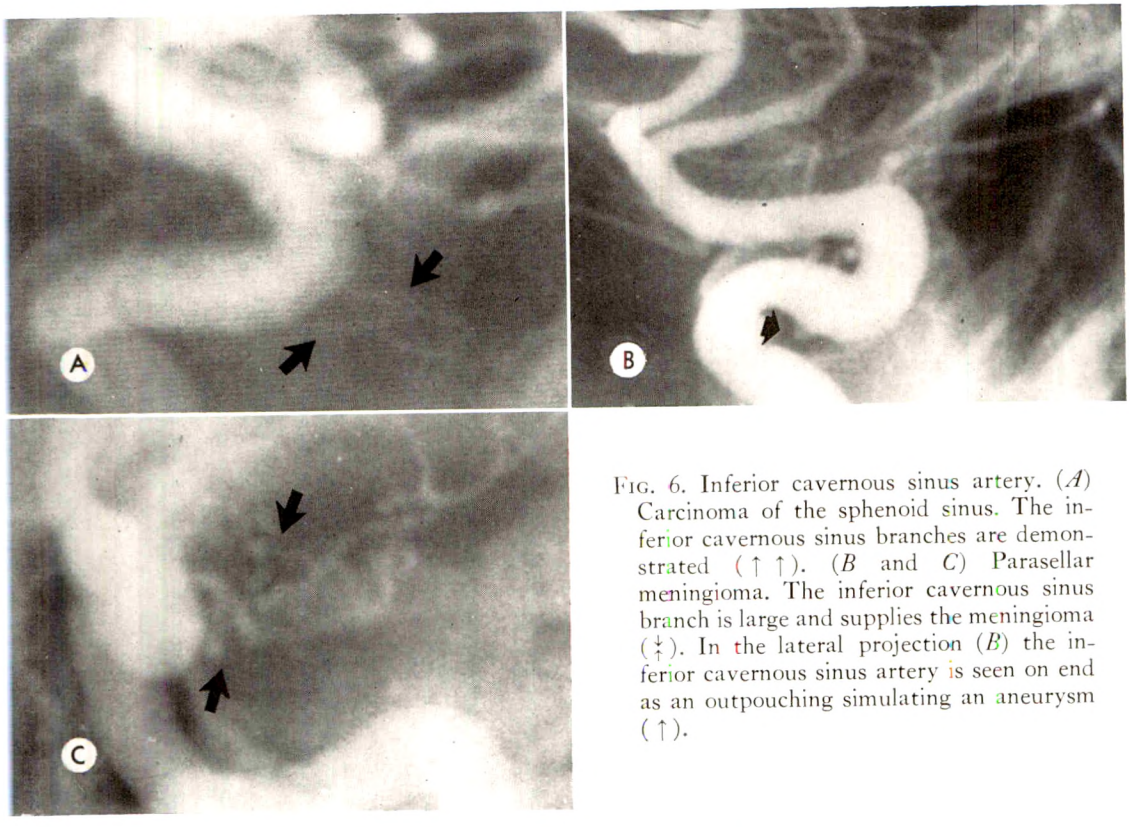



FIG. 6. Inferior cavernous sinus artery. (*A*) Carcinoma of the sphenoid sinus. The inferior cavernous sinus branches are demonstrated (↑ ↑). (*B* and *C*) Parasellar meningioma. The inferior cavernous sinus branch is large and supplies the meningioma (↓ ↑). In the lateral projection (*B*) the inferior cavernous sinus artery is seen on end as an outpouching simulating an aneurysm (↑).

5*B*). Similar cases have been reported by Ecker and Riemenschneider,[5] Kricheff and Schotland,[9] and Doron and Schwartz.[3] The inferior hypophyseal artery was also observed in a patient with a carcinoma of the sphenoid sinus (Fig. 5*C*).

### B. INFERIOR CAVERNOUS SINUS ARTERY

In 80 per cent of Parkinson's specimens there was another major trunk originating 1/2 cm. distal to the meningohypophyseal artery. The inferior cavernous sinus artery (lateral main stem) takes off from the lateral aspect of the midportion of the cavernous carotid and supplies the dura of the inferior aspect of the cavernous sinus and the sinus itself. Branches extend to the gasserian ganglion, while others anastomose with the middle meningeal arteries. Schnürer and Stattin[15] noted another branch of the inferior cavernous sinus, the marginal tentorial artery, running along the tentorial edge.

The inferior cavernous sinus artery is demonstated in 2 patients, one with a sphenoid sinus carcinoma (Fig. 6*A*) and the other with a parasellar meningioma (Fig. 6, *B* and *C*).

### C. CAPSULAR ARTERIES

The capsular arteries (McConnell),[11] one or two branches, originate further distally. The *inferior* capsular artery comes from the inferomedial aspect of the internal carotid artery, halfway along the course of the cavernous carotid. This vessel runs medially in the dura covering the inferior surface of the anterior lobe of the hypophysis. It anastomoses with branches of the inferior hypophyseal artery, giving branches to the floor of the sella.

The anterior capsular artery originates along the medial aspect of the internal carotid immediately before this vessel pierces the dura over the cavernous sinus. It runs medially in the dura at the anterior margin

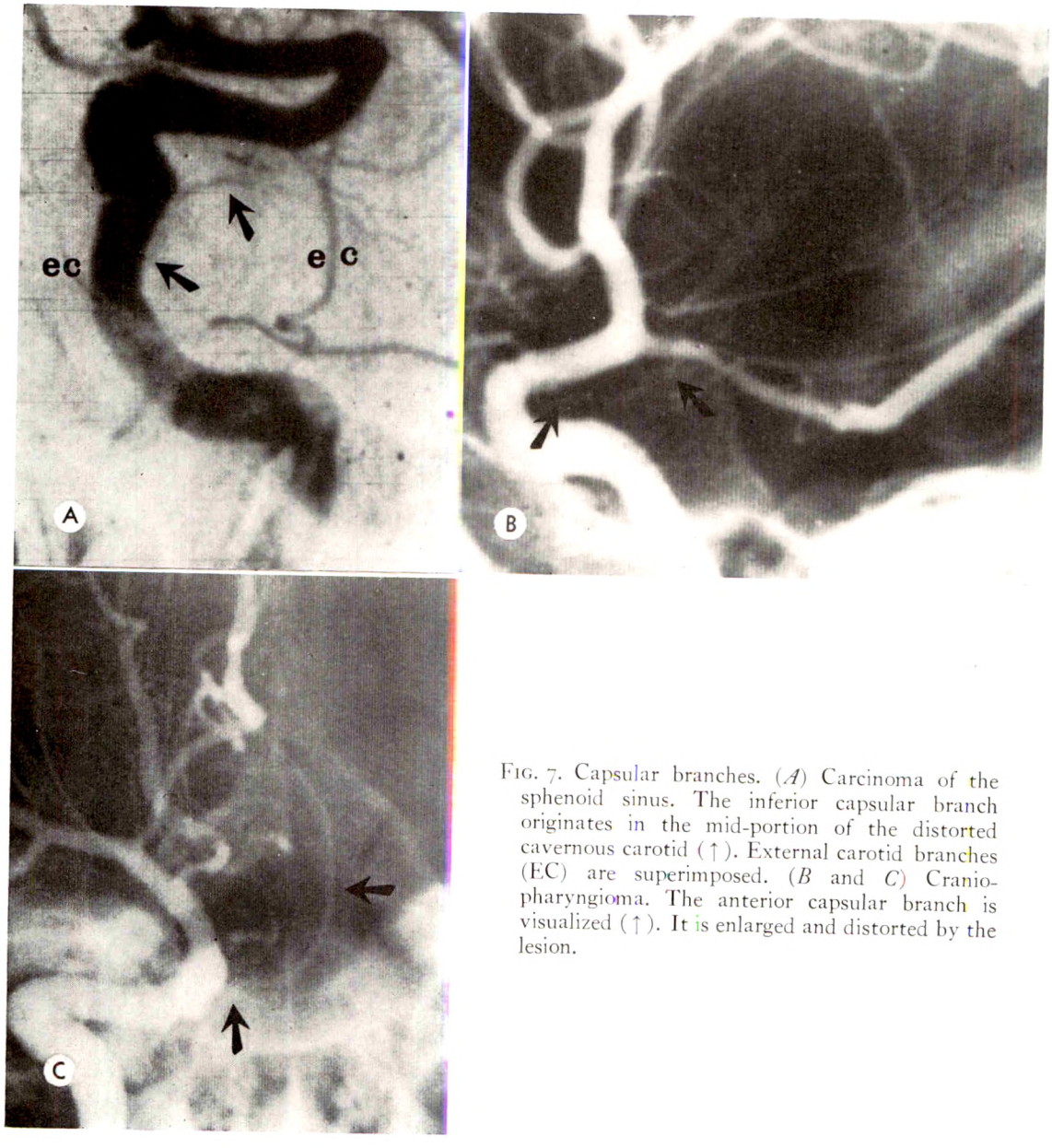



FIG. 7. Capsular branches. (*A*) Carcinoma of the sphenoid sinus. The inferior capsular branch originates in the mid-portion of the distorted cavernous carotid (↑). External carotid branches (EC) are superimposed. (*B* and *C*) Craniopharyngioma. The anterior capsular branch is visualized (↑). It is enlarged and distorted by the lesion.

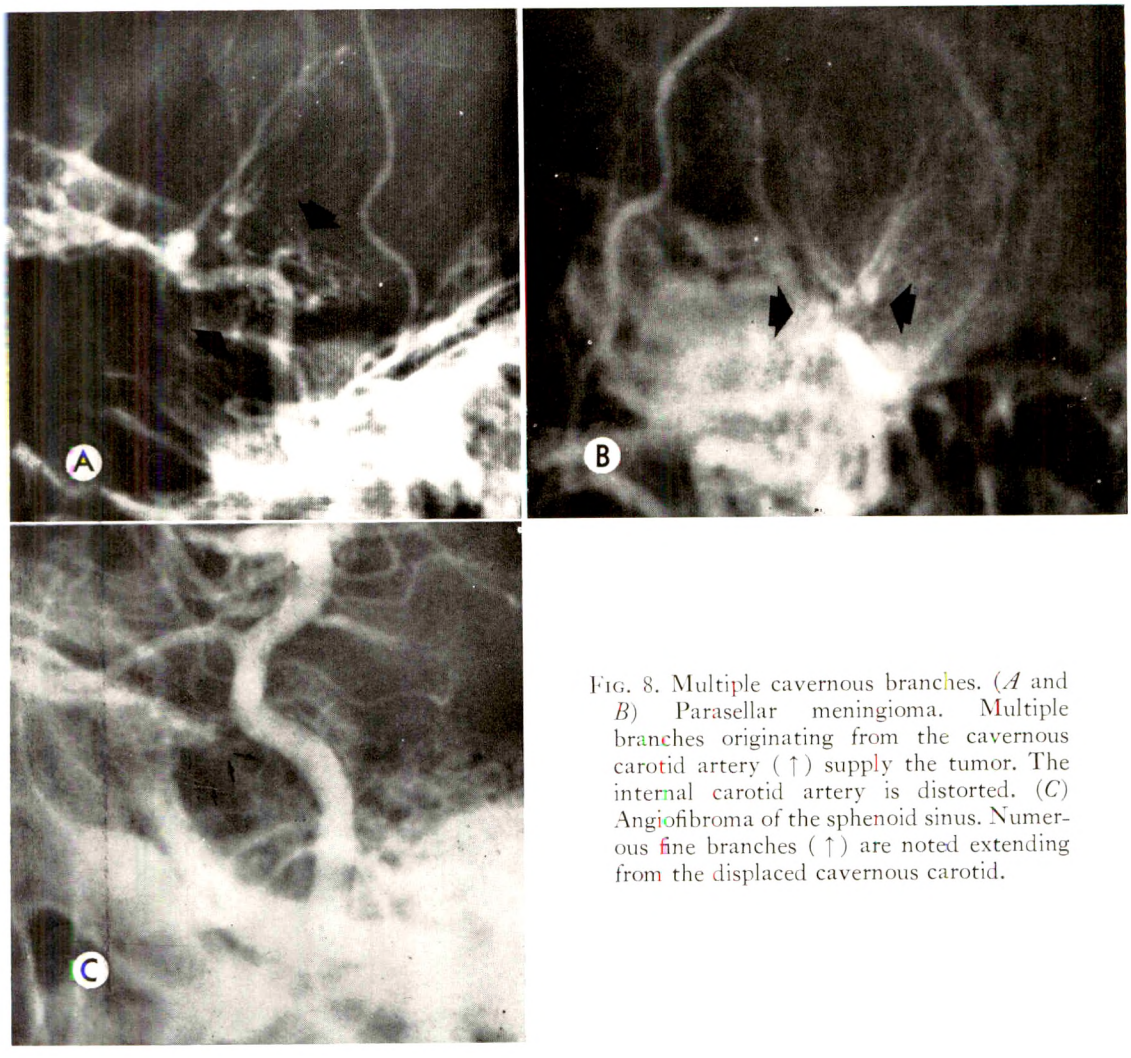



Fig. 8. Multiple cavernous branches. (*A* and *B*) Parasellar meningioma. Multiple branches originating from the cavernous carotid artery ( ↑ ) supply the tumor. The internal carotid artery is distorted. (*C*) Angiofibroma of the sphenoid sinus. Numerous fine branches ( ↑ ) are noted extending from the displaced cavernous carotid.

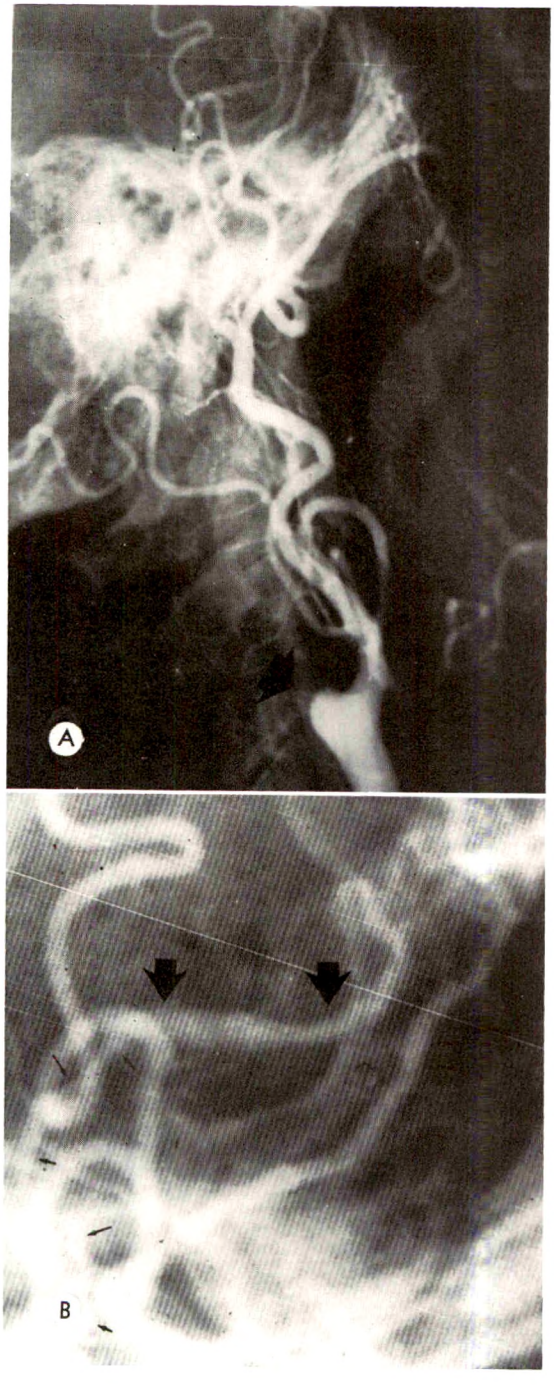



FIG. 9. Rete mirabile. Ligation of the internal carotid artery. (*A*) The internal carotid artery (↑) was ligated in the neck as treatment for a large intracranial aneurysm. (*B*) The cavernous carotid is reconstituted by branches of the external carotid artery (↑). The superficial temporal artery loop is superimposed.

of the roof of the sella, anastomosing with its fellow of the opposite side. McConnell[11] observed these two branches in half of the specimens.

These arteries were opacified in a patient with a sphenoid sinus carcinoma (the inferior capsular artery) (Fig. 7*A*), and in a craniopharyngioma (anterior capsular artery) (Fig. 7, *B* and *C*).

At times, multiple branches of the cavernous portion of the internal carotid artery may be visualized when the base of the skull is extensively involved as was observed in patients with a parasellar meningioma (Fig. 8, *A* and *B*) and an angiofibroma of the sphenoid sinus (Fig. 8*C*). In such cases, specifically named branches are difficult to delineate but rather a plethora of small vessels may be seen.

### D. RETE MIRABILE

Each of the branches of the cavernous carotid anastomoses with its mate from the opposite carotid artery, and with the meningeal branches of the external carotid, the ophthalmic, and the vertebral arteries. This basilar plexus has been termed the carotid rete or rete mirabile. These vessels are of utmost importance as collateral pathways in the event of occlusion of the internal carotid artery below the cavernous sinus. The reconstitution of the cavernous carotid artery by the external carotid artery via the cavernous branches is illustrated in a patient with a proximal internal carotid artery occlusion (Fig. 9, *A* and *B*). In still another case (Fig. 10, *A–D*), a child with multiple stenotic segments within both internal carotid arteries, the tentorial branch served as a collateral pathway reforming the posterior cerebral artery to the point of stenosis at its origin. Minagi and

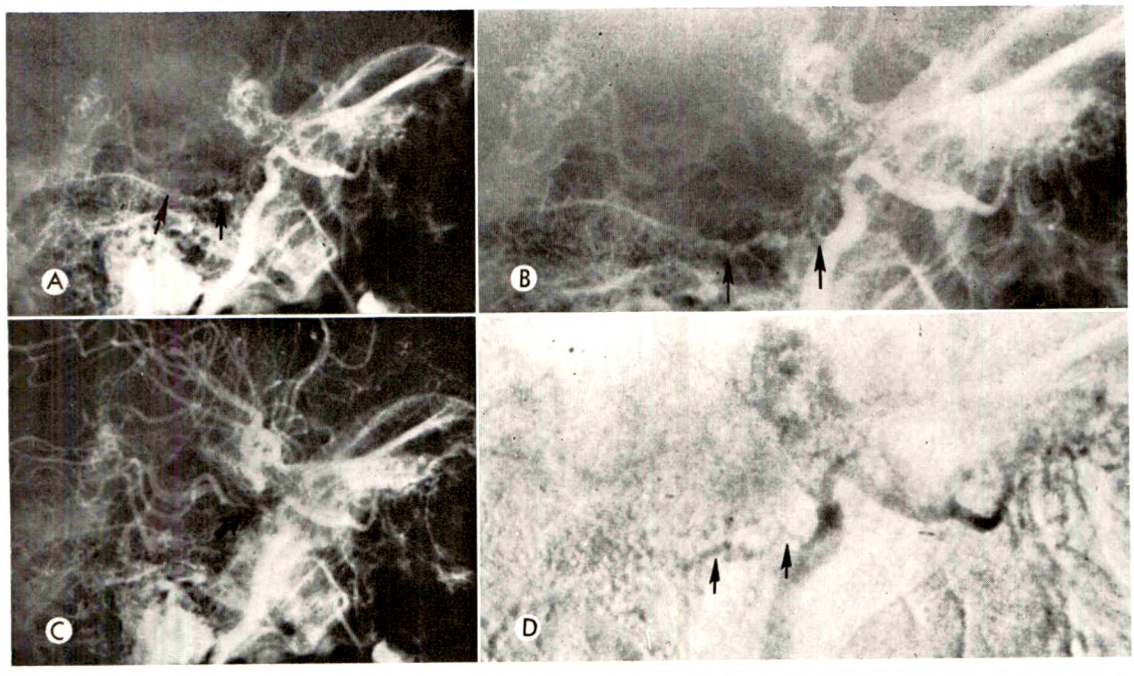



Fig. 10. Rete mirabile. A 6 year old child with microcephaly and subarachnoid bleeding who had multiple areas of stenosis with extensive collateral blood supply. (*A* and *B*) The tentorial artery ( ↑ ↑ ) serves as a collateral pathway reforming the posterior cerebral artery. There is a stenosis ( ↑ ) of the origin of the posterior communicating artery demonstrated after retrograde filling from the tentorial artery. (*C* and *D*) The tentorial vessel is visualized originating from the cavernous carotid ( ↑ ↑ ).

Newton[12] reported the presence of multiple cavernous branches anastomosing an enlarged external carotid artery with the cavernous portion of the carotid compensating for a hypoplastic cervical segment of the internal carotid artery.

The communication between the external and internal carotid arteries by way of the cavernous branches is of significance in the diagnosis and management of carotid cavernous fistulae. Selective injection of the external carotid artery demonstrated a fistula as well as the internal carotid opacification (Fig. 11, *A–C*). Hayes[7] observed similar findings following carotid artery ligation. The proper approach to the treatment of these fistulae will be dependent upon knowledge about all communicating channels.

## DISCUSSION

The ability to demonstrate carotid cavernous branches is dependent upon their size, for under normal circumstances these branches are filamentous. The cavernous branches have generally escaped recognition. Undoubtedly the utilization of high quality serial angiography, subtraction, and perhaps angiotomography[4] may aid in their demonstration.

The visualization of branches of the cavernous portion of the carotid artery does not necessarily indicate the presence of a lesion but should precipitate close scrutiny of the base of the skull and tentorium. The appearance of these branches has been observed in a variety of pathologic entities (Table II) occurring at the base. Schnürer and Stattin[15] designated a maximum normal length of 35 mm. for the tentorial artery. Length has not been found to be helpful in distinguishing the normal from the pathologic. These vessels are considered to have pathologic significance when there is enlargement of caliber, increased undulations in their course, the presence of

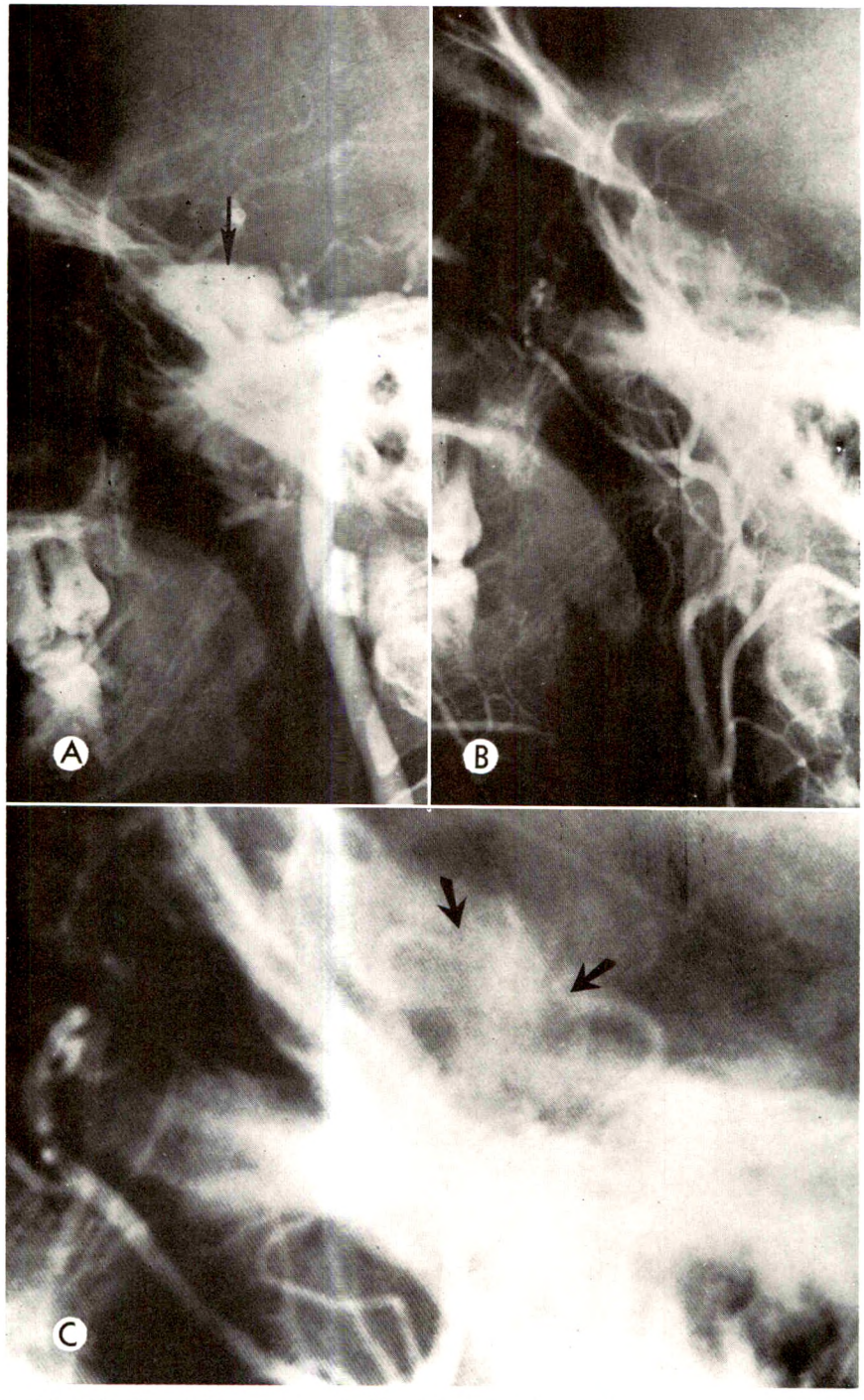



FIG. 11. Rete mirabile. Carotid cavernous fistula. (*A*) Internal carotid artery opacification demonstrates the carotid cavernous fistula ( ↑ ). (*B* and *C*) Selective external carotid artery injection at the same time demonstrates the carotid (external) communications filling the fistula ( ↑ ↑ ↑ ).

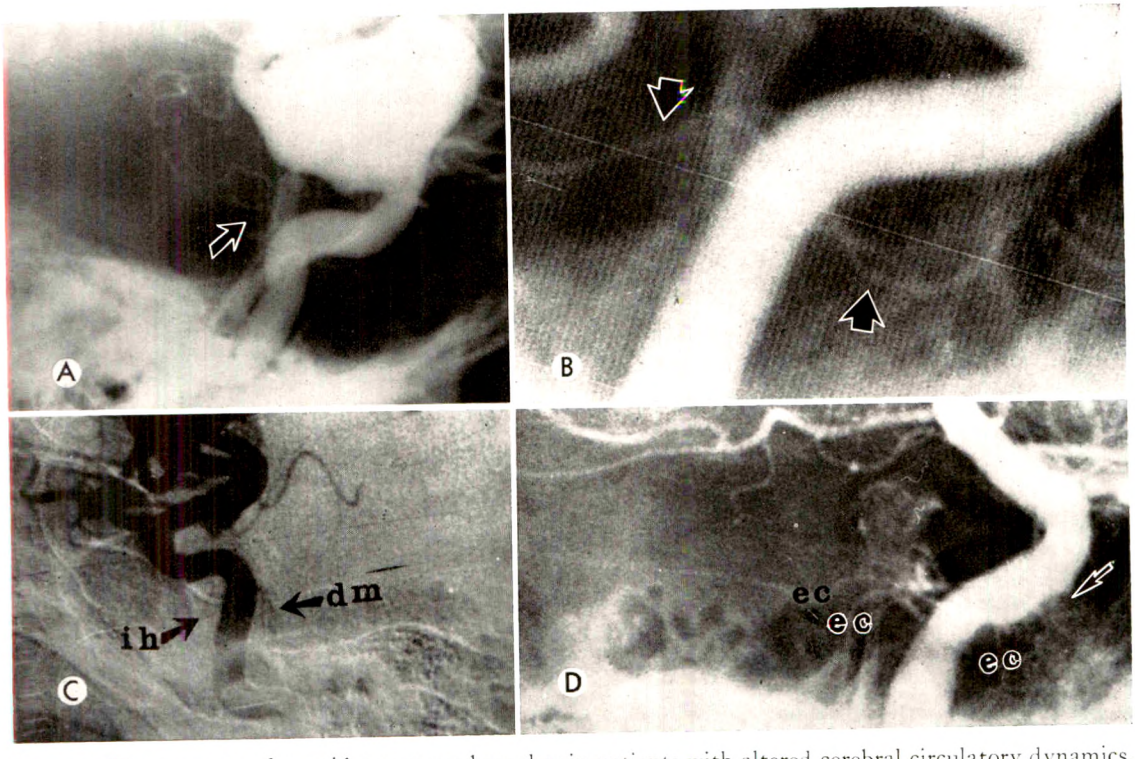



FIG. 12. The presence of carotid cavernous branches in patients with altered cerebral circulatory dynamics. (*A*) Aneurysm. Cavernous branches ( ↑ ) are opacified below the internal carotid aneurysm. (*B*) Increased intracranial pressure secondary to an intracerebral tumor. Visualization of the cavernous branches ( ↑ ) was an associated finding. (*C*) Temporal lobe glioma with increased intracranial pressure. The dorsal meningeal (←dm) and inferior hypophyseal (ih→) arteries are demonstrated. (*D*) Carotid cavernous branches are visualized but are not acting as collateral channels in a patient with partial occlusion of the middle cerebral artery. The external carotid branches are superimposed.

multiple branches and the occurrence of tumor stain. Tumor stains (Fig. 3, 4, 5, 6, and 8) have been observed in benign and malignant lesions. In our 3 pituitary tumors with stains, histologic examination revealed that all *were* benign adenomas.

Opacification of the cavernous branches may also occur with alterations in cerebral dynamics, *e.g.*, increased intracranial pressure of any etiology, the presence of an intracranial lesion at a distance (tumor and aneurysm), and cerebral vascular disease in which these branches do not appear, angiographically, to function as collateral pathways (Fig. 12, *A–D*). The carotid cavernous branches may act as the rete mirabile in animals and in some patients with proximal internal carotid artery occlusions. This type of collateral may also be demonstrated in patients with carotid cavernous fistulae. Lack of appreciation of this alternate pathway may account for the failures observed in some cases following carotid artery ligation.

## SUMMARY

Knowledge of the anatomy of the cavernous branches of the carotid artery has led to their recognition with increasing frequency during cerebral angiography. These branches have been observed in the normal, benign and malignant lesions at the base of the skull and tentorium, and in cases with altered cerebral circulatory dynamics.

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TABLE II

CONDITIONS IN WHICH CAVERNOUS BRANCHES HAVE BEEN OBSERVED

A. Normal

B. Alteration in Cerebral Circulatory Dynamics
   1. Increased intracranial pressure
   2. Lesions at a distance
      a. Tumor
      b. Aneurysm
      c. Cerebral vascular disease—not a collateral

C. Congenital and Neoplastic Lesions
   1. Tentorial arteriovenous malformations
   2. Tentorial meningiomas
   3. Tentorial invasion by intracranial neoplasm
      a. Glioblastoma
      b. Ependymoma
      c. Metastasis (Zimmer)
   4. Trigeminal neuroma (Westberg)
   5. Pituitary adenoma
   6. Craniopharyngioma
   7. Parasellar meningioma
   8. Chordoma
   9. Nasopharyngeal carcinoma
   10. Nasopharyngeal angiofibroma
   11. Sphenoid sinus carcinoma
   12. Rhabdomyosarcoma of the orbit

D. Rete Mirabile
   1. Occlusive or stenotic cerebral vascular disease
   2. Carotid-cavernous fistula

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# PERSISTENT PRIMITIVE TRIGEMINAL ARTERY DEMONSTRATED BY VERTEBRAL ARTERIOGRAPHY*

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SINCE 1950 when Sutton[12] published the first report of a case in which the diagnosis of persistent primitive trigeminal artery was made by angiography, more than 100 cases have been described. All of these cases were found by carotid arteriography and in all the blood flow was from the carotid artery into the basilar artery. Thus, Tönnis and Schiefer[13] state the following in their textbook: "In all cases the direction of the flow within the anastomosis is probably from the arteria carotis to the arteria basilaris. Likewise, according to data from the literature an opposite flow direction has so far not been observed." That the direction of flow can be reversed so that the abnormal communication fills on vertebral arteriography only, is apparent from the case reported here.

## REPORT OF A CASE

This 54 year old male had been prone to left-sided headache for approximately 2 years. On the day of admission vertigo, headache, and vomiting had developed after the patient had turned his head while driving his automobile. The acute symptoms soon subsided. On neuroroentgenologic examination, the following findings were noted.

*Left internal carotid arteriography*. Changes similar to those seen in atheromatosis were demonstrated in the carotid syphon, the lumen of the vessel being irregular and slightly narrowed. Apart from this, no other abnormalities were revealed.

*Right common carotid arteriography*. On the internal carotid artery, just above its origin from the common carotid artery, an hourglass-shaped stenosis, about 1 cm. long, was found. In the narrowest part of the stenosis, the lumen measured about 1 mm.; calcifications were present in the region of the stenosis (Fig. 1, *A* and *B*).

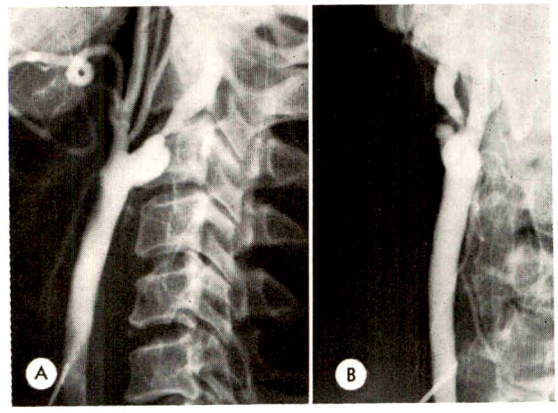



FIG. 1. (*A*) Lateral view. (*B*) Frontal view. Arteriograms showing the right common carotid artery. Just above the origin of the internal carotid artery a stenosis, about 1 cm. in length with a minimum caliber of a little more than 1 mm., is demonstrated.

In the basal part of the carotid syphon (Fig. 2, *A* and *B*), almost at the level of the dorsum sellae, a small tapering prominence of contrast material was seen dorsally. Distally, the opacification of the syphon was definitely fainter than in the proximal direction and of varying quality, so that the artery appeared somewhat wider on some roentgenograms than on others, as if contrast-free blood had entered through a persistent carotid-basilar anastomosis. The anterior cerebral artery was poorly opacified. The arterial phase lasted longer on this side than on the left: $4\frac{1}{2}$ seconds as against $2\frac{1}{2}$ seconds. On the other hand, no difference in circulation time was found, as determined by the method of Greitz.[2]

*Left vertebral arteriography* with the catheter in the left subclavian artery (Fig. 3 *A*). The left vertebral artery was absent. This was also noted on aortocervical arteriography.

*Right vertebral arteriography* with the catheter in the right subclavian artery (Fig. 3 *B*). All vessels of the vertebral region were filled with contrast medium. A wide right primitive tri-

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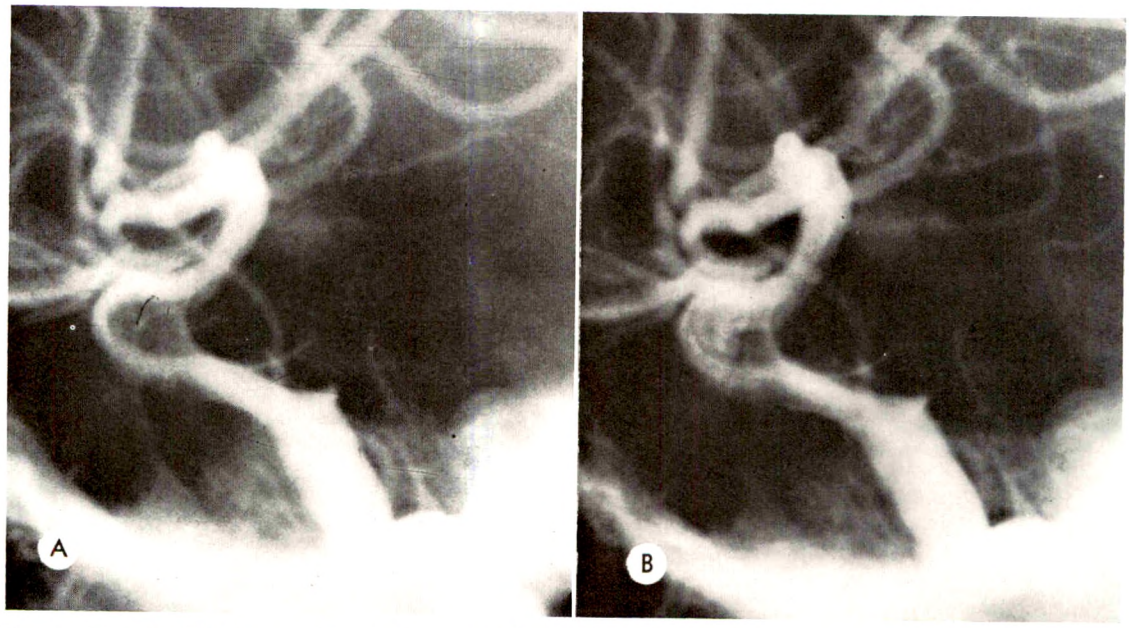



FIG. 2. (*A* and *B*) Right carotid arteriograms. Magnified portion of the syphon region. At the level of the dorsum sellae, a small conical prominence of contrast medium is seen dorsally. In the dorsal part of the syphon a streaky, somewhat varying dilution of the contrast medium is noted, as if contrast-free blood had entered.

geminal artery was demonstrated (Fig. 4, *A* and *B*). The right carotid syphon and the right middle cerebral artery were filled from this anastomosis. The site where the anastomosis joined the carotid artery corresponded to the prominence of the density of the contrast medium found in the carotid syphon on carotid arteriography.

*Pneumoencephalography*. Apart from slight cortical atrophy, no abnormalities were revealed.

## DISCUSSION

The primitive trigeminal artery is the most common of the persistent fetal carotid-basilar anastomoses. The first report of this condition appeared by Quain[8] in 1844. The embryologic conditions which have been described thoroughly by several authors,[7] and the various concepts relating to the clinical importance of this vessel and of other types of persistent fetal carotid-basilar anastomoses will not be discussed here. In this respect, the reader is referred to reports published, among others, by Schiefer and Walter,[11] Krayenbühl and Yaşargil,[4] Jackson and Garza-Mercado,[3] and Schaerer.[10]

Arteriographically demonstrated cases reported previously have shown that under normal conditions the blood flow in the primitive trigeminal artery is directed from the carotid artery into the basilar artery. Consequently, the primitive trigeminal artery contributes to the supply of blood to the vertebral system and during carotid arteriography smaller or larger portions of the arteries in the posterior cranial fossa are opacified. These conditions may vary and, consequently, Saltzman[9] distinguished between two types of the malformation. In the first type, the primitive trigeminal artery contributes to the supply of the entire vertebral system distal to the anastomosis. In the second type, the anastomosis supplies mainly the superior cerebellar artery on both sides while the posterior cerebral artery receives its blood partly through the posterior communicating artery. Combinations of the two types have also been described.

Huber[5] reported a case in which the primitive trigeminal artery filled during both carotid and vertebral arteriography. He stated that the pressures in the carotid

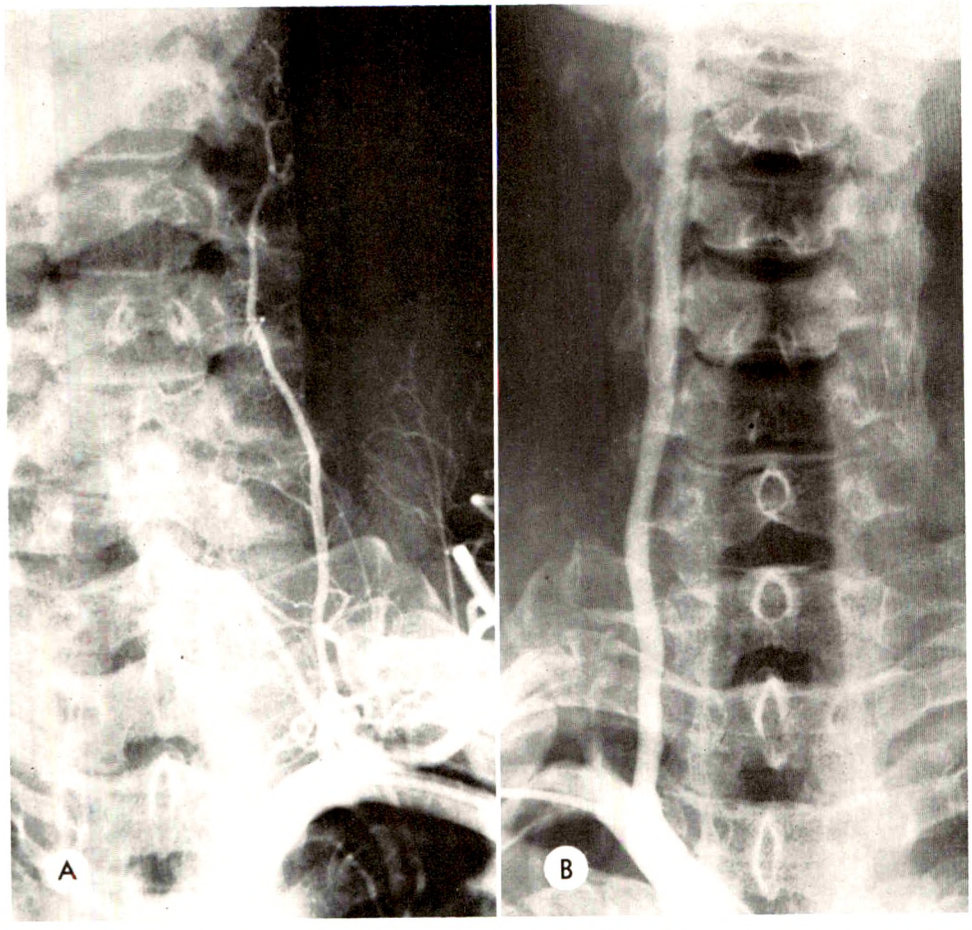



Fig. 3. (*A*) Left vertebral arteriogram with catheterization of the left subclavian artery. The vertebral artery is absent. (*B*) Right vertebral arteriogram with catheterization of the right subclavian artery. The vertebral artery is somewhat larger than normal.

artery and in the vertebral artery are identical, so that a flow in either direction may occur under certain conditions. Because of a greater kinetic energy in the carotid artery, which is normally wider than the vertebral artery, the blood flow runs from the carotid artery to the basilar artery. In one case, Nadjmi[6] showed that by compressing the carotid artery on the same side, the primitive trigeminal artery could be opacified during vertebral arteriography. He, therefore, presumed that the primitive trigeminal artery, when persistent, would opacify on vertebral arteriography almost as frequently as the posterior communicating artery. He supposed that the reason why the variation in the direction of the blood flow was found only by himself, and almost simultaneously by Huber,[5] was because vertebral arteriography is performed in relatively few cases with this anomaly.

It is universally known that the intracranial flow depends on the pressure conditions and that the vertebral system may contribute to the blood supply of the carotid system through the posterior communicating artery. If the pressure in the carotid artery is reduced in the presence of a persistent carotid-basilar anastomosis, it would be a logical consequence that the blood flow in the anastomosis should be reversed. Brice *et al.*[1] studied the effect of a stenosis on flow and pressure in the carotid artery, both in patients in whom the carotid artery was ligated and by means of model experi-

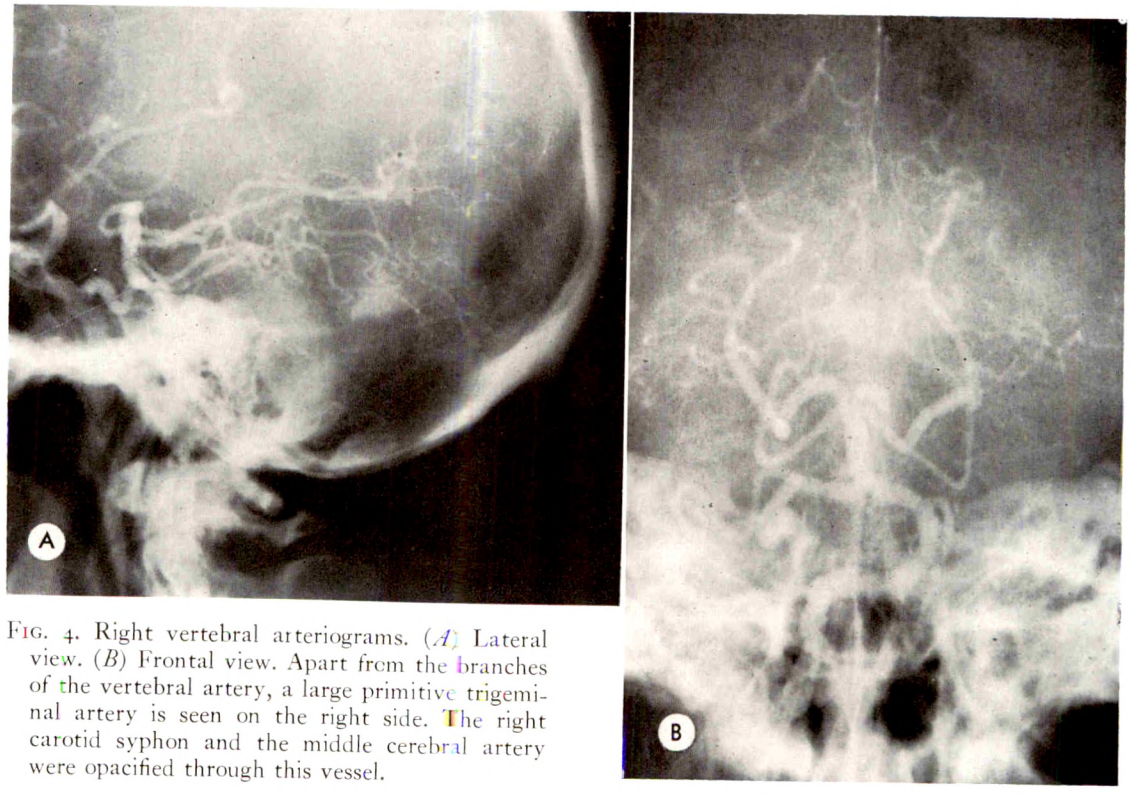



FIG. 4. Right vertebral arteriograms. (*A*) Lateral view. (*B*) Frontal view. Apart from the branches of the vertebral artery, a large primitive trigeminal artery is seen on the right side. The right carotid syphon and the middle cerebral artery were opacified through this vessel.

ments. They found that a stenosis with a cross sectional area larger than 5 sq. mm. was of no importance, irrespective of length and multiplicity of stenosis. If the diameter ranged between 2 and 5 sq. mm., the flow could be reduced, depending on the shape of the stenosis, the number of stenoses present and, to some extent, on the length of the stenosis. In cases of stenosis with a cross sectional area of less than 2 sq. mm., a significant reduction in flow and pressure ensued.

In the case reported here, the smallest diameter of the stenosis was a little more than 1 mm. and thus the cross sectional area was less than 2 sq. mm. The pressure and flow in the internal carotid artery, therefore, must have been reduced, which could explain the reverse flow in the primitive trigeminal artery. Also, the prolongation of the arterial phase during carotid arteriography indicates a decrease in pressure and flow in the right carotid artery.

Although in the present case the primitive trigeminal artery was not filled during carotid arteriography, the changes found at this examination were of such a nature as to suggest the presence of an abnormal communication between the carotid artery and the vertebral artery, and to distinguish the changes described from those seen in atherosclerosis. Hence, the distinct dilution in the carotid syphon seems to have been caused by a jet of contrast-free blood from the persistent anastomosis. The blood flow passed mainly along the upper posterior part of the carotid syphon and caused the syphon to be incompletely—and on several angiograms—differently opacified. Furthermore, part of the site of entrance of the primitive trigeminal artery was opacified in such a way that a small prominence of the density of the contrast medium was revealed dorsally on the carotid artery.

## SUMMARY

A case of persistent primitive trigeminal artery is described, in which a stenosis of the internal carotid artery was present on the same side. The persistent abnormal

fetal communication was filled on vertebral arteriography only. However, on the basis of the dilution effect seen on the carotid arteriogram, it could be conjectured that an abnormal communication existed between the carotid syphon and the vertebral artery and that the blood flow was directed from the vertebral artery to the carotid artery.

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# THE ARTERIOGRAPHIC PATTERN ACCOMPANYING CENTROSYLVIAN MASS LESIONS*

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ANGIOGRAPHY has generally been considered inferior to air encephalography in the demonstration of expanding lesions medial to the sylvian fissure. It is important to visualize mass lesions in this area because of the frequency of hemorrhage, which occurs there as a consequence of cerebrovascular disease and hypertension. As recently as 1959, Lazorthes,[1] in reviewing 52 cases of surgically treated cerebral hemorrhage stated, "if the collection of blood is deep within the parenchyma it may not displace the arteries, and in this case may pass unnoticed."

In contrast to the concept expressed in this statement, the present authors believe that most clinically apparent mass lesions in the centrosylvian portion of the brain are recognizable by a specific arteriographic pattern. During the past decade this pattern has been repeatedly noted and is now considered to be of reliable diagnostic value. The purposes of this paper are: (1) to describe this pattern; (2) to review the frequency of occurrence of its components in a series of 100 cases diagnosed angiographically; and (3) to correlate the arteriographic pattern with pathologic findings in an attempt to explain the mechanisms of the vascular displacements.

The centrosylvian region includes the portions of the cerebral hemispheres between the insula laterally and the third and lateral ventricle medially. It contains the structures of the diencephalon (mainly the thalamus), the internal capsule, and portions of the basal ganglia (the lenticular and caudate nuclei). Because of these various structures, a number of terms have been used synonymously to describe the region, *e. g.*, thalamic, capsular, nuclear. The term centrosylvian is probably to be preferred because it does not imply specific involvement of a particular neural structure.

## ARTERIOGRAPHIC PATTERN

The typical arteriographic pattern involves displacement of the middle cerebral artery in both the anteroposterior and the lateral projections. These are demonstrated diagrammatically in Figure 1, *A* and *B*, and arteriographically in Figure 2, *A* and *B*. In the anteroposterior projection, the middle cerebral artery describes a wide sweeping arc which results from downward displacement of the $M_1$ portion and lateral displacement of the $M_2$ and $M_3$ portions of the vessel. Depending upon the size of the lesion, and to a lesser extent on its position anteroposteriorly, the anterior cerebral artery may be displaced toward the opposite side. As will be noted later there is probably a correlation between the anterior cerebral displacement and the prognosis in cases of cerebral hemorrhage.

The lateral view is characterized by upward displacement of the middle cerebral components, similar to that seen in expanding lesions of the temporal lobe. This involves the anterior extent of the sylvian portion of the vessel more frequently than the posterior part. The upward displacement, as seen in the lateral projection, is apparently due to a separate mechanism from that which causes the characteristic anteroposterior configuration. Thus, these two displacements do not necessarily occur simultaneously and each may be present independently.

The typical arteriographic pattern is easily distinguishable from those produced by mass lesions occurring elsewhere in the cerebral hemispheres. Posterior frontal lesions are likely to cause downward displace-

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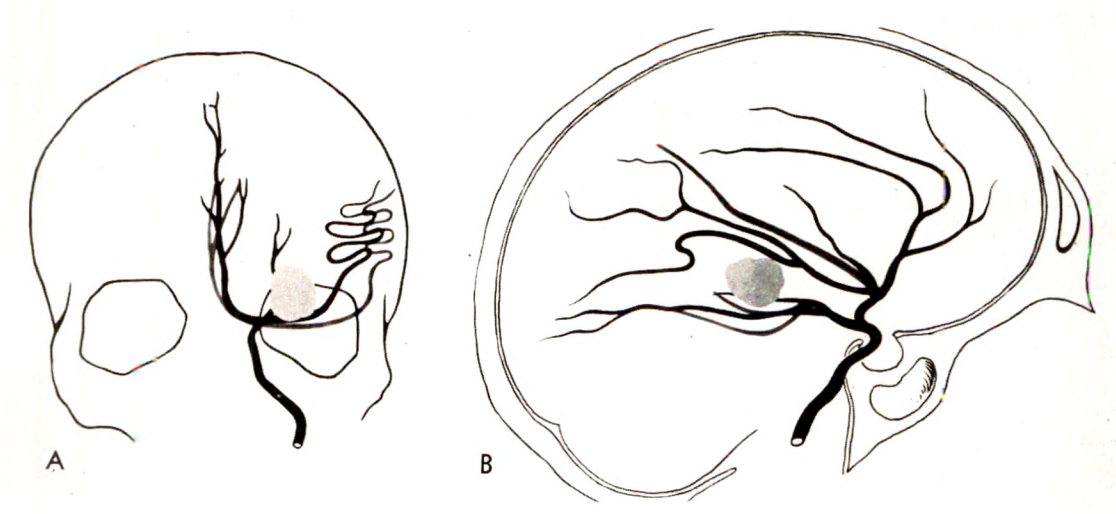



Fig. 1. (*A* and *B*) Diagrammatic representation of displacement of vessels by a centrosylvian lesion in frontal and lateral projections. The normal vascular pattern (black) is altered in a manner shown in gray.

ment of the middle cerebral complex as seen in the lateral view. Temporal lobe lesions, both anterior and posterior, may be indistinguishable from centrosylvian lesions in the lateral projection but are characterized in the anteroposterior projection by upward displacement of $M_1$ or medial displacement of the sylvian point.

## MATERIAL AND METHODS

In order to analyze the reliability of the described arteriographic pattern a series of 100 cases were reviewed. Each case had carotid arteriography performed in the frontal and lateral projections. The presence of a centrosylvian mass was determined in each case by one or more means. In 41 instances the lesion was proven at autopsy, while in 30 cases it was demonstrated by surgical intervention. In the remaining 29 cases, a clinical course characteristic of hemorrhagic infarction involving the centrosylvian structures was considered sufficient evidence to include the material in this survey.

Displacement of the middle cerebral

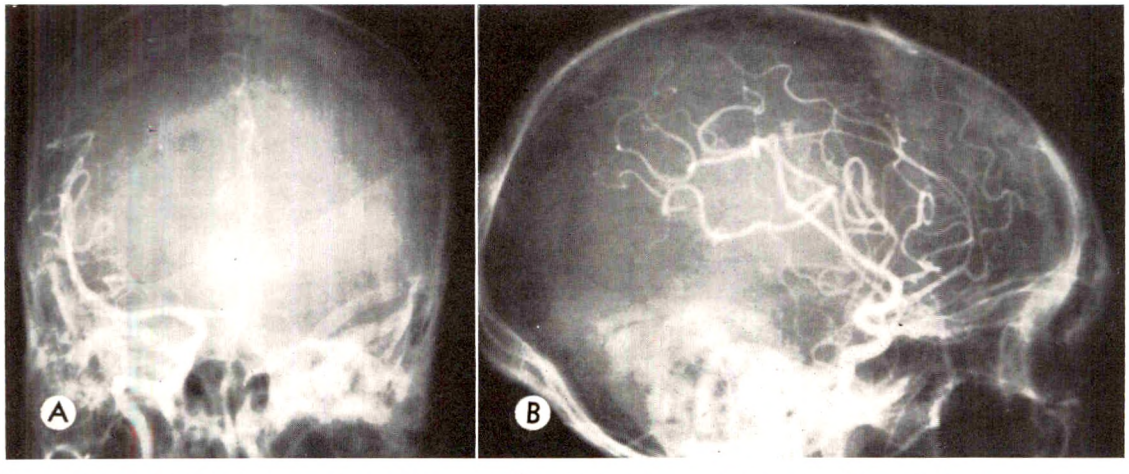



Fig. 2. (*A* and *B*) Typical arteriographic pattern caused by centrosylvian mass. The middle cerebral artery is displaced upward in lateral projection and outward in frontal projection, and the anterior cerebral artery is displaced toward the opposite side.

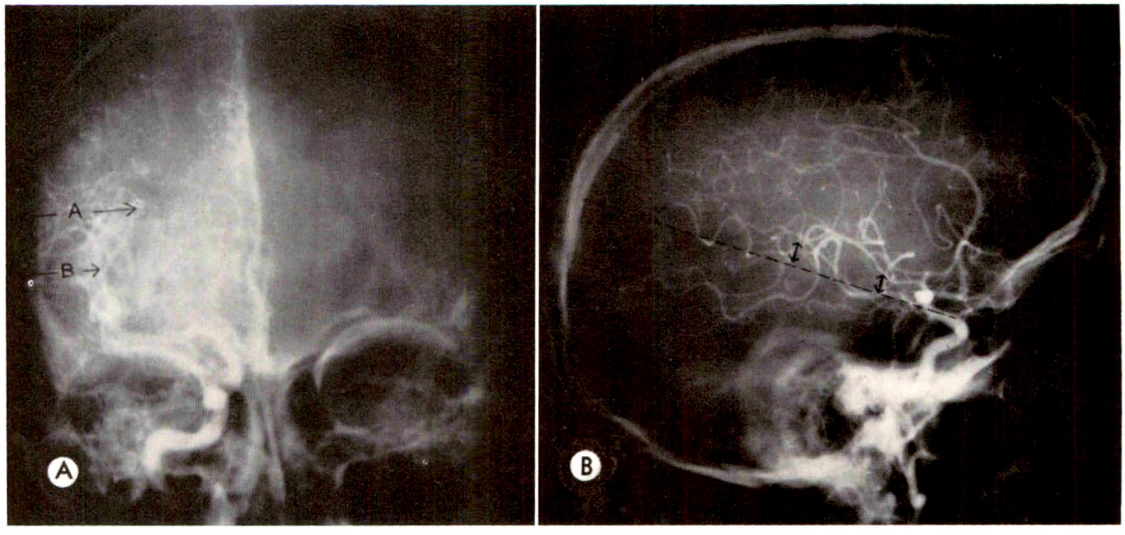



FIG. 3. (*A*) Frontal projection showing sylvian point (B) and insular point (A) and method of measurement in relation to inner skull table. (*B*) Lateral projection showing relation of clinoparietal line to middle cerebral artery.

complex was determined by measurement of the sylvian (B) and insular (A) points in the frontal projection (Fig. 3*A*) and the relationship of the main arterial channel to the clinoparietal line in the lateral view (Fig. 3*B*). As described by Taveras and Wood,[4] the angiographic sylvian point (B) represents the posterior point of the sylvian fissure and is identified in the frontal projection as the medial extremity of the last (highest) vessel emerging from the fissure to the surface of the hemisphere. The distance of this point from the inner skull table varies from 30 to 43 mm. (Fig. 3*A*). Another point, defined by Taveras and Wood,[4] is the most medial aspect of the most medial vessel outlining the lateral margin of the insula as seen in the frontal projection (Fig. 3*A*). Since this point marks the most lateral extent of the insula we have chosen to call it the *insular point* (A). It may be noted that while the sylvian point corresponds to the most posterior extremity of the sylvian fissure, the insular point is anterior to this extremity for a variable distance depending upon the displacement of the vessels within the fissure. The insular point varies normally from 20 to 30 mm. from the inner skull table.

The measured distances of the sylvian point B from the internal skull surface in the 100 cases with thalamic lesions are plotted in Figure 4. Seventy of the measurements fall below the minimum normal of 30 mm., while the remaining 30 are within the lower half of the normal range. Similarly, the measurements of the insular point

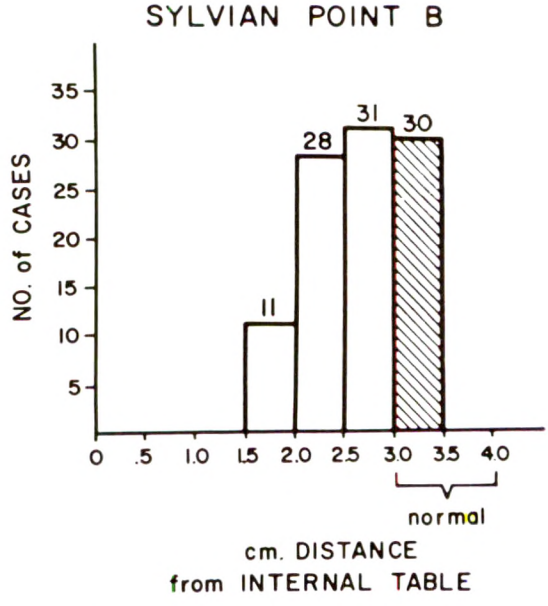



FIG. 4. Analysis of relationship of sylvian point B to inner skull table in 100 cases of centrosylvian mass lesions.

A are shown in Figure 5, and again 70 cases are below the normal limits while the remaining 30 are in the lower range of normal variation. These abnormal measurements result from lateral displacement of the insula and sylvian fissure due to the presence of an expanding mass medial to these structures.

The clinoparietal line, described by Taveras and Poser,[3] connects the anterior clinoid process to a point 2 cm. above the lambda (Fig. 3*B*). In the adult skull the lambda is frequently difficult to visualize

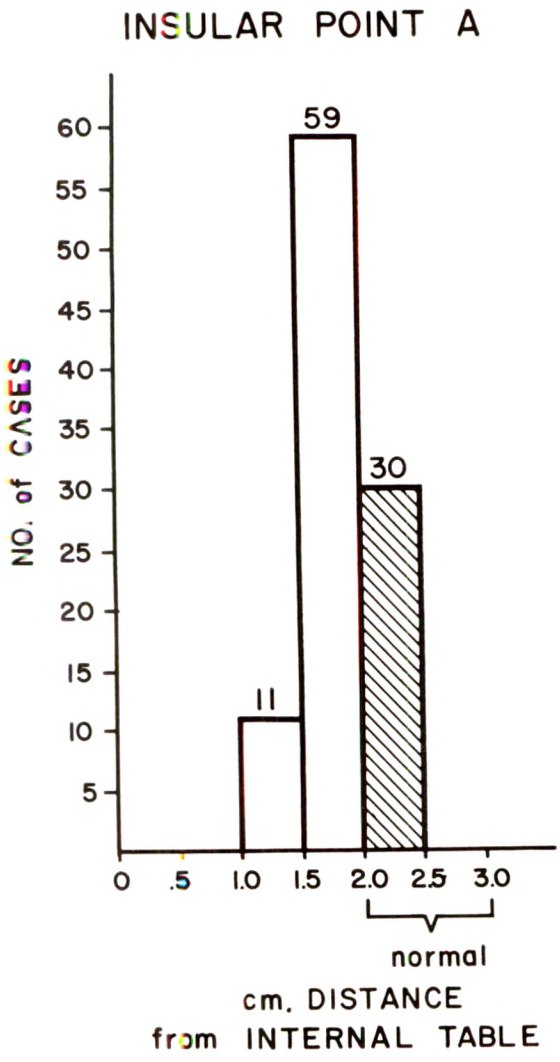



FIG. 5. Analysis of relationship of insular point A to inner skull table in 100 cases of centrosylvian mass lesions.

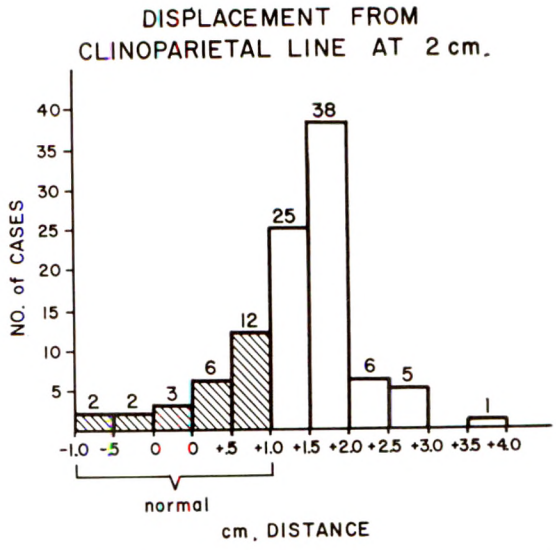



FIG. 6. Analysis of relationship of middle cerebral artery, measured 2 cm. behind the posterior clinoid process, to clinoparietal line in 100 cases of centrosylvian mass lesions.

and a point 9 cm. above the internal occipital protuberance may be used. This latter reference point was used in the present series. The clinoparietal line normally lies at, or just below, the lower major channels of the middle cerebral artery complex in a true lateral projection. Elevation of these vessels above the line implies upward displacement of the sylvian fissure and insula. In the present study measurements were made at arbitrary distances of 2.0 and 5.0 cm. behind the posterior clinoid process along the clinoparietal line. The normal variation was considered to be from 1.0 cm. below to 1.0 cm. above the line. The measurements are plotted in Figures 6 and 7. At 2.0 cm. posterior to the clinoid process, 75 cases showed upward displacement of the middle cerebral vessels while at 5.0 cm., 64 cases were elevated beyond the normal range. It may be concluded that a measurement at 2.0 cm. is slightly more reliable in demonstrating the displacement caused by centrosylvian lesions.

The arteriographic pattern which is considered diagnostic of centrosylvian lesions consists of 2 components: downward and outward displacement of the middle cere-

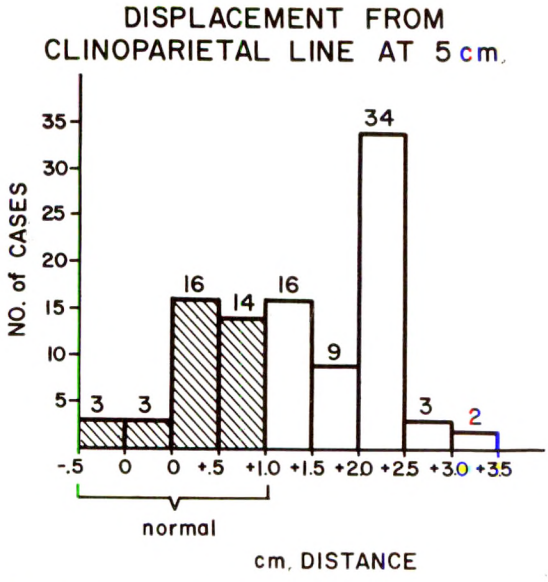



FIG. 7. Analysis of relationship of middle cerebral artery, measured 5 cm. behind the posterior clinoid process to clinoparietal line in 100 cases of centrosylvian mass lesions.

bral vessel in frontal projection, and elevation of this vessel in the lateral view. The frequency of these displacements in the 100 cases is demonstrated in Table 1. Fifty-two per cent of the cases exhibited both components with measurements which were outside the normal range. Another 18 per cent demonstrated only the frontal displacement, while in the lateral view the measurements were within normal limits (Fig. 8, *A* and *B*). It should be noted, however, that in this group the localization is clearly that of a centrosylvian mass. Twenty-three per cent had elevation of the middle cerebral vessel on lateral projection but measured within the normal range in the frontal view (Fig. 9, *A* and *B*). An accurate localization is more difficult in this group. A temporal lobe mass may cause a similar arteriographic pattern in the lateral projection but is usually distinguishable by either upward or medial displacement of the middle cerebral complex in the frontal projection. Only 7 per cent of the surveyed cases fell within the normal range in both projections by measurement, although the characteristic pattern was suggested in each instance by inspection.

TABLE I

MIDDLE CEREBRAL ARTERY PATTERNS IN THALAMIC LESIONS

| Lateral View | Anteroposterior View | No. |
|---|---|---|
| ↑ | ↓ → | 52 |
| Normal | ↓ → | 18 |
| ↑ | Normal | 23 |
| Normal | Normal | 7 |
| | | 100 |

In addition to the arteriographic changes emphasized above, other associated displacements have been noted. These include bowing and downward displacement of the

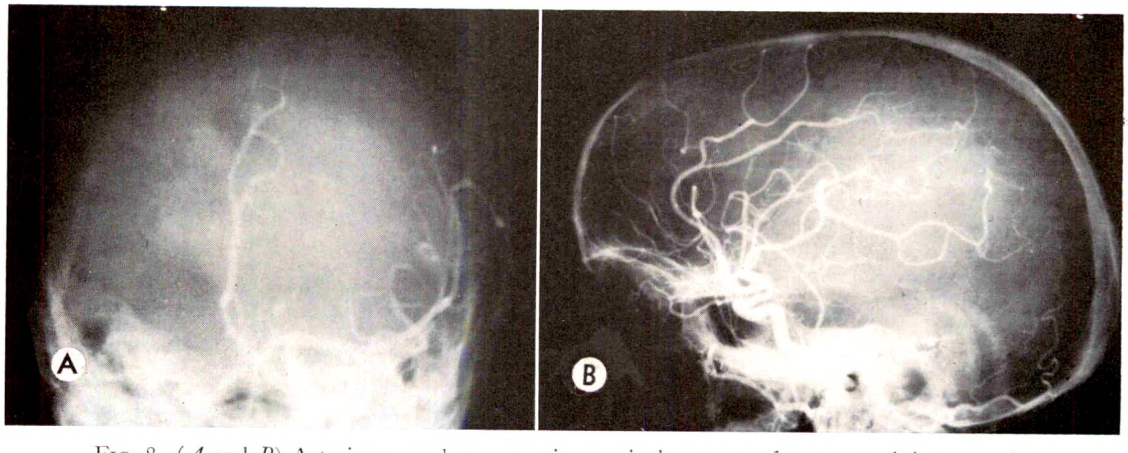



FIG. 8. (*A* and *B*) Arteriograms demonstrating typical pattern of a centrosylvian mass in the frontal projection, but a normal appearance in the lateral projection.

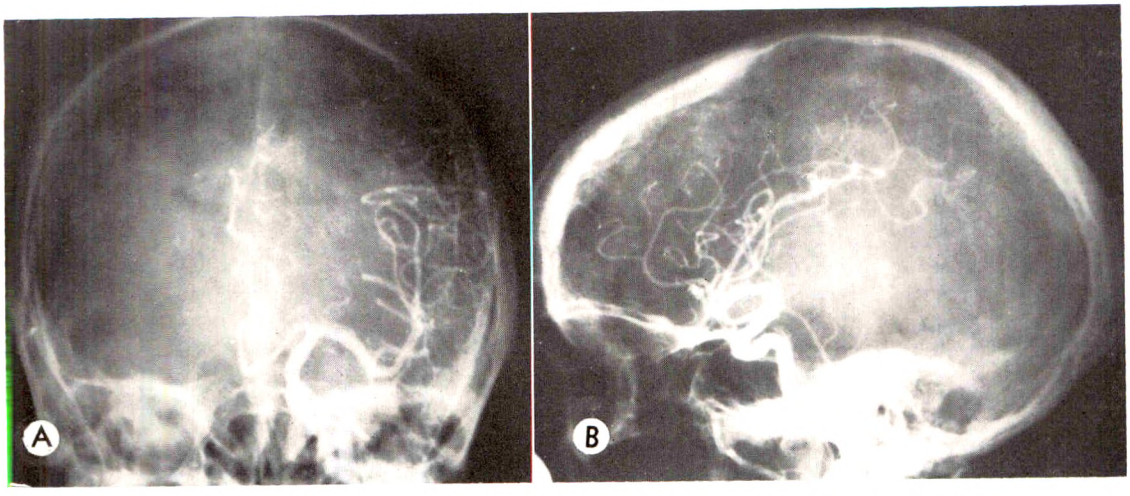



FIG. 9. (*A* and *B*) Arteriograms showing normal pattern in frontal projection, but displacement of middle cerebral vessel in lateral projection.

perforating vessels leaving the middle cerebral artery near its origin. In addition, the anterior cerebral artery has been displaced toward the opposite side in the majority of cases. Taveras and Wood[4] state, "Thalamic tumors characteristically do not produce displacement of the anterior cerebral artery across the midline, . . . . " In the present series 78 per cent had anterior cerebral displacement.

### MECHANISM UNDERLYING ARTERIOGRAPHIC PATTERNS

Since most of the thalamic masses considered in this review are hematomas of spontaneous origin, it may be useful to consider briefly their anatomic development. Hemorrhage usually originates from the central or capsulostriate vessels with extravasation of blood toward the cortical centers, or in the white matter at the junction of the central and peripheral arterial territories.[1] The usual hemorrhage causes tearing of the internal capsule with permanent neural injury and with compression of the vital diencephalic centers (Fig. 10). The hemorrhage may rupture into the ventricular system. From the internal capsule it extends along the white matter into the temporal lobe and the parieto-occipital region. Extension into the frontal lobe occurs rarely. The basic mechanism, therefore, appears to be the centrosylvian origin with extension downward into the temporal white matter.

By the correlation of angiographic patterns with anatomic distortions seen on postmortem examination, a concept has been developed to explain the arteriographic displacements. It may be noted in horizontal brain sections that the area of hemorrhage, although variable and frequently quite extensive, is centered posterior to the horizontal portion of the middle cerebral artery ($M_2$) (Fig. 11). Thus, the more distal portions of the vessel are likely to show the greatest displacement. The hemorrhagic mass occurs above the level of $M_1$ and medial to the temporoparietal portion of the sylvian fissure which con-

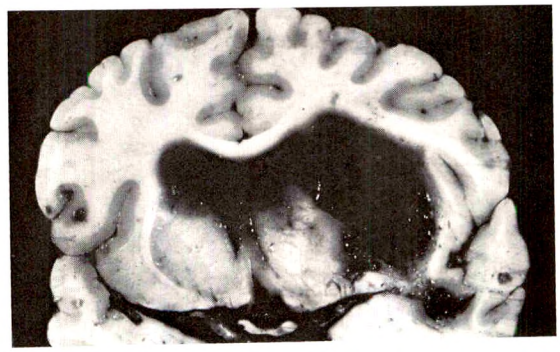


FIG. 10. Coronal section of brain showing centrosylvian hemorrhage with rupture into ventricle.

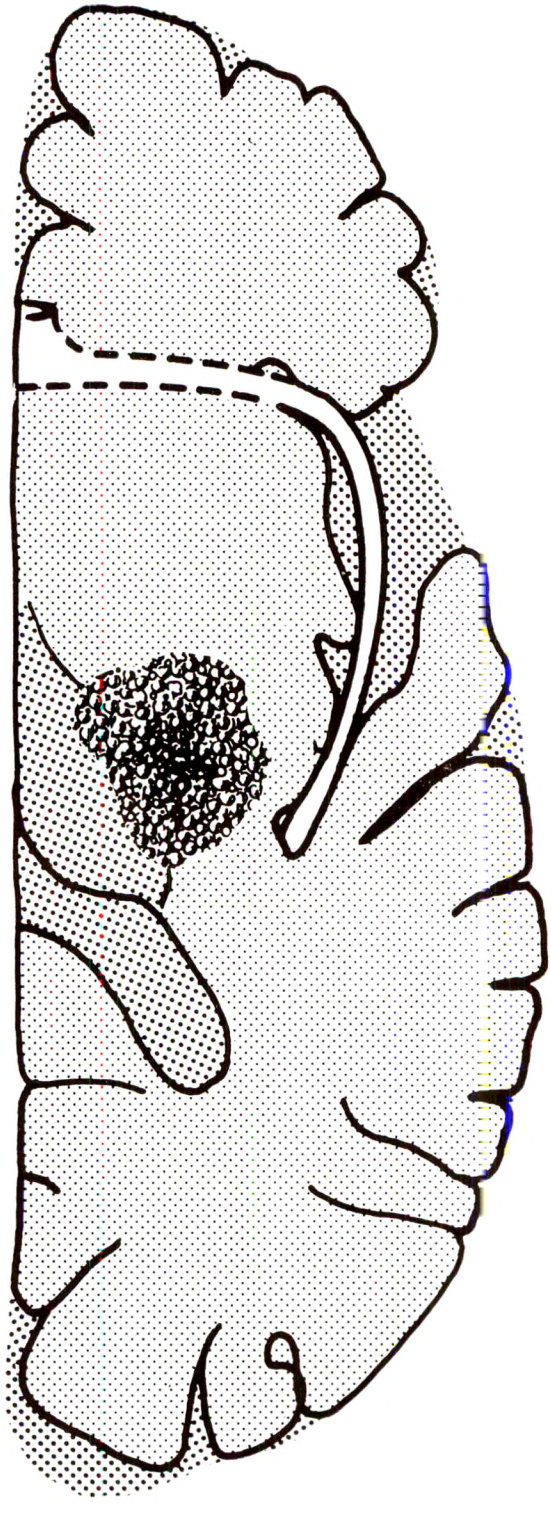


FIG. 11. Diagrammatic horizontal brain section, as viewed from above, showing relationship of centrosylvian mass to the middle cerebral artery.

tains the portions of the middle cerebral artery referred to as $M_2$, $M_3$, and $M_4$ (Fig. 12). It is apparent, therefore, that $M_1$ will be displaced downward while $M_2$, $M_3$, and $M_4$ will be moved laterally toward the skull. The sylvian point and the surface of the insula are displaced closer to the inner skull table than permitted by normal variation.

Upward displacement of the distal portions of the middle cerebral artery is somewhat more difficult to explain. From postmortem observations it appears that it results from upward migration of the sylvian fissure due to several factors. The outward pressures exerted by a deep hematoma, and the mass of the hematoma itself, must be compensated for by migration of adjacent brain substance (Fig. 13). Since the lesion is low in the hemisphere, the greatest pres-

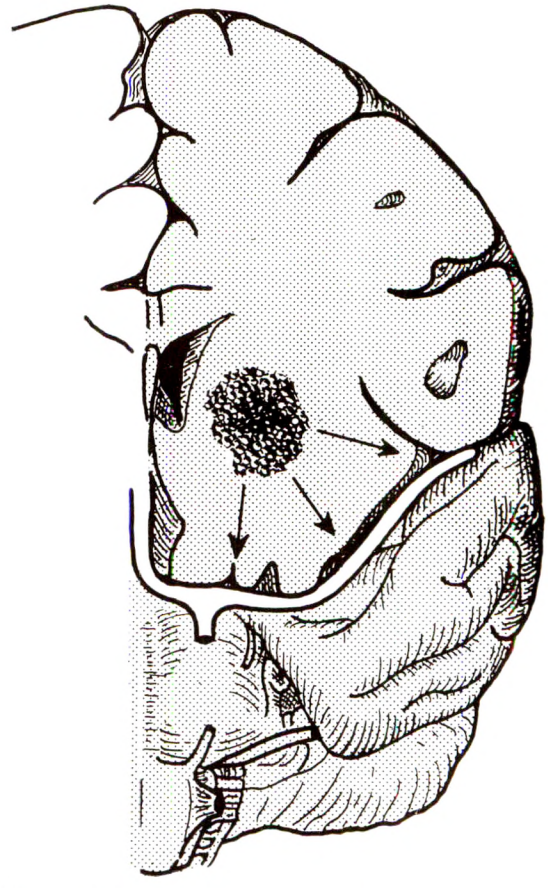


FIG. 12. Diagrammatic coronal brain section showing relationship of centrosylvian mass to middle cerebral artery.



FIG. 13. Diagrammatic coronal brain section showing lines of force which probably lead to upward migration of the sylvian fissure and the middle cerebral artery.

sure is exerted downward toward the temporal lobe. It must be recalled, however, that the temporal lobe is encased on each side, inferiorly and anteriorly, by bony structures and the only direction for migration is upward. Thus, there is a tendency for the compressed temporal lobe substance to migrate upward in its lateral portion carrying with it the sylvian fissure. The inferior portion of the insula is tipped outward and upward along with the vessels passing over its surface. Moreover, the hematoma itself frequently extends into the temporal lobe, as noted previously. This leads to the presence of a temporal expanding mass which is superimposed on the migration effect described.

## DISCUSSION

The majority of centrosylvian masses are the result of spontaneous intracerebral hemorrhage due to hypertensive and/or degenerative cerebrovascular disease. This continues to be a major cause of morbidity and mortality and until recently was usually considered untreatable. The development of angiographic techniques, however, has been the greatest single stimulus to reappraisal of the problem from the standpoint of possible improvement of results through neurosurgical intervention.

Angiographic assessment of patients with suspected intracerebral hemorrhage is of value for the following reasons: (1) to distinguish between an ischemic stroke and hemorrhagic infarction; a clinical distinction can usually be made with reliability but in occasional instances the distinction is not possible; (2) to determine if a vascular abnormality such as aneurysm or arteriovenous malformation may have been responsible for the hemorrhage; (3) to distinguish between "nuclear" (centrosylvian) and "medullary" (subcortical) hemorrhages, since the surgical and prognostic implications are entirely different; and (4) to determine whether midline shift exists, since patients with a shift have a considerably greater mortality than those whose midline structures are not displaced.[2] It is recommended, therefore, that angiography be carried out on all patients suspected of spontaneous intracerebral hemorrhage who are not moribund or terminal when seen for evaluation.

## SUMMARY

Despite implications from previous literature that angiography is unreliable in the diagnosis of centrosylvian mass lesions, the present authors believe that a specific arteriographic pattern does occur with significant consistency. The pattern includes downward and outward displacement of the middle cerebral artery as seen in the frontal projection and upward migration of the middle cerebral complex, as shown in the lateral projection. A series of 100 cases were reviewed using measurements of the sylvian (B) and "insular" (A) points from the internal skull table and of the displace-

ment of the middle cerebral complex from the clinoparietal line. The survey disclosed that only 7 per cent fell within the normal limits of these measurements. The mechanisms leading to the arteriographic pattern are postulated. Most centrosylvian mass lesions are hemorrhagic infarcts and their identification angiographically may lead to improved clinical management.

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# TERMINAL BASILAR ARTERY DEFORMITY SECONDARY TO SUPRASELLAR MASSES AND THIRD VENTRICULAR DILATATION*

*By* JAMES H. SCATLIFF, M.D.,† E. LEON KIER, M.D.,‡ LAWRENCE H. ZINGESSER, M.D.,§ *and* MANNIE M. SCHECHTER, M.D.‖

DISTORTION of the floor of the 3rd ventricle by an elongated tortuous basilar artery has been noted by several authors.[2,4,9,10] There has been little mention, however, of the reverse effect,[11] namely caudal dislocation and bending of the terminal basilar artery produced by lesions in the region of the 3rd ventricle and interpeduncular cistern. Several authors have commented on changes produced in the terminal basilar artery as a result of downward anterior transtentorial herniation.[1,3,6,7,11] That this deformity of the terminal basilar artery can also be produced by a dilated 3rd ventricle has not heretofore been described specifically.

## REPORT OF CASES

CASE I. (J.B.), a 41 year old white male was admitted with a 3 year history of personality change and intermittent visual difficulties. These were associated with headaches, ataxia, and confusion.

Physical examination revealed an obese male who was lethargic and confused. The neurologic examination, including analysis of the visual fields, was normal. The spinal fluid protein was 38 mg. per cent and the fluid was clear.

*Roentgenologic Findings.* Plain skull roentgenograms were normal. Carotid angiography showed no abnormalities of the carotid tree. Vertebral angiography was performed. Figure 1*A* demonstrates the backward bending of the terminal basilar artery. The unusual appearance of this artery suggested that the patient might have a midline lesion just above the terminal basilar artery. For this reason ventriculography was performed. A ventriculogram (Fig. 1*B*) reveals a large mass in the posterior 3rd ventricle.

*Operation.* A large cystic tumor was found in the 3rd ventricle. Histologic examination showed the tumor to be an epidermoid.

CASE II. (P.K.), a 46 year old white male had gradual onset of lethargy, ataxia, and decreasing intellectual function over a 5 year period prior to admission. The patient was diagnosed as having a presenile dementia, and he was hospitalized in a mental institution. After a period of several months in the institution, he returned to a normal state. Subsequent to this, the patient had 2 relapses with incomplete mental recovery. The next hospital admission was precipitated by further mental deterioration.

Physical examination on admission revealed an obese man who was confused. The principal neurologic finding on admission was truncal ataxia. The spinal fluid protein was 88 mg. per cent.

*Roentgenologic Findings.* Plain skull roentgenograms were negative. Pneumoencephalography revealed a mass filling most of the 3rd ventricle (Fig. 2, *A* and *B*). The floor of the 3rd ventricle was deformed and the tumor was outlined in the interpeduncular cistern. There was associated deformity of the terminal basilar artery. A subclavian artery injection showed no abnormality of the carotid arterial tree on the right. The basilar artery, however, was noted to be sharply angulated (Fig. 2*C*).

*Operation.* A large cystic mass was found in the third ventricle. Histologic examination revealed the mass to be a cystic craniopharyngioma.

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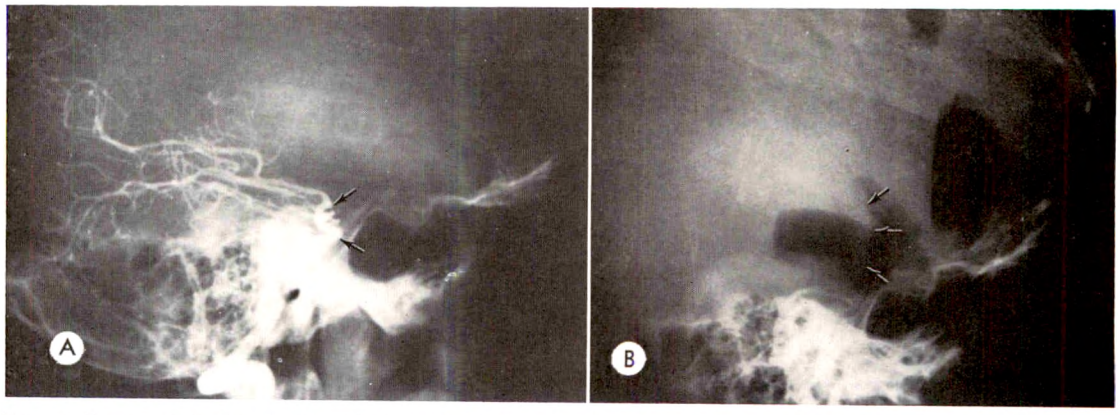



FIG. 1. Case I. (*A*) Vertebral angiogram, lateral projection. The arrows indicate the terminal segment of the basilar artery which is deformed. (*B*) Ventriculogram, brow-up, lateral projection. The arrows point to the anterior pole of the 3rd ventricular mass as outlined by air in the anterior portion of the 3rd ventricle. Note that the large mass extends below the level of the dorsum sellae. The mass is partially obscured by the temporal horns, which contain air.

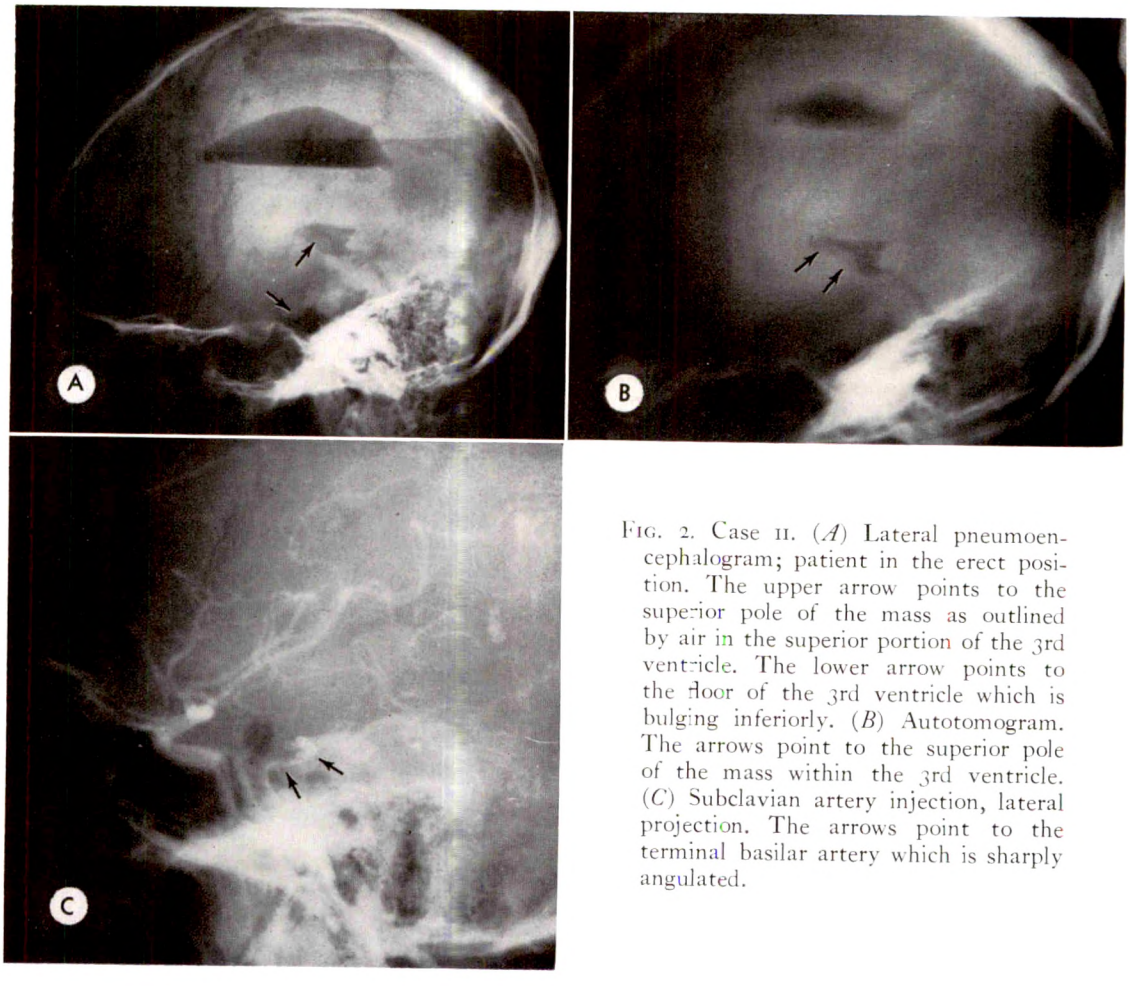



FIG. 2. Case II. (*A*) Lateral pneumoencephalogram; patient in the erect position. The upper arrow points to the superior pole of the mass as outlined by air in the superior portion of the 3rd ventricle. The lower arrow points to the floor of the 3rd ventricle which is bulging inferiorly. (*B*) Autotomogram. The arrows point to the superior pole of the mass within the 3rd ventricle. (*C*) Subclavian artery injection, lateral projection. The arrows point to the terminal basilar artery which is sharply angulated.

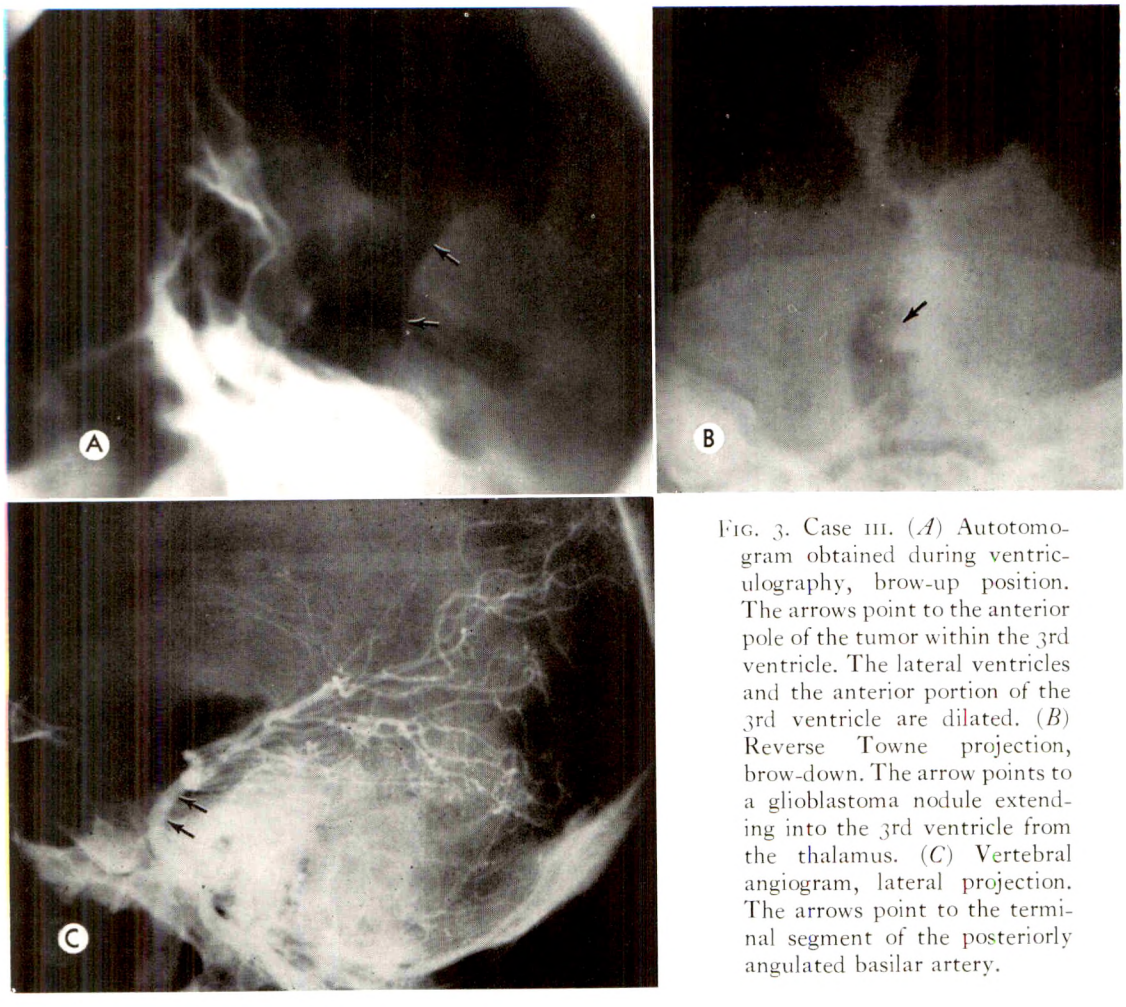



Fig. 3. Case III. (*A*) Autotomogram obtained during ventriculography, brow-up position. The arrows point to the anterior pole of the tumor within the 3rd ventricle. The lateral ventricles and the anterior portion of the 3rd ventricle are dilated. (*B*) Reverse Towne projection, brow-down. The arrow points to a glioblastoma nodule extending into the 3rd ventricle from the thalamus. (*C*) Vertebral angiogram, lateral projection. The arrows point to the terminal segment of the posteriorly angulated basilar artery.

Case III. (D.H.), a 64 year old female had a clinical history suggesting raised intracranial pressure. The presence of ataxia suggested that a posterior fossa tumor was present.

*Roentgenologic Findings.* Plain skull roentgenograms showed erosion of the posterior clinoid processes indicating the presence of raised intracranial pressure. Ventriculography revealed dilatation of the lateral ventricles and of the anterior portion of the third ventricle. The posterior 3rd ventricle was occupied by a mass which seemed to arise from the thalamus (Fig. 3, *A* and *B*). Vertebral angiography was carried out in order to exclude a tentorial meningioma. Figure 3*C* demonstrates a marked angulation of the terminal basilar artery.

*Operation.* A Torkildsen bypass procedure (ventriculocisternostomy) was carried out.

*Postmortem Examination.* A large glioblastoma arising from the thalamus and invading the posterior 3rd ventricle was noted.

## DISCUSSION

The 3 cases reported emphasize the marked alteration which may be produced in the position and configuration of the basilar artery by lesions superior and anterior to the termination of this vessel. These cases would appear to catalogue the variation in deformities that might be expected to occur in the presence of suprasellar masses. In Case I, the artery has been telescoped inferiorly into the superior portion of the pontine cistern. The vessel in Case II has been bent acutely backward in the posterior aspect of the interpeduncular cis-

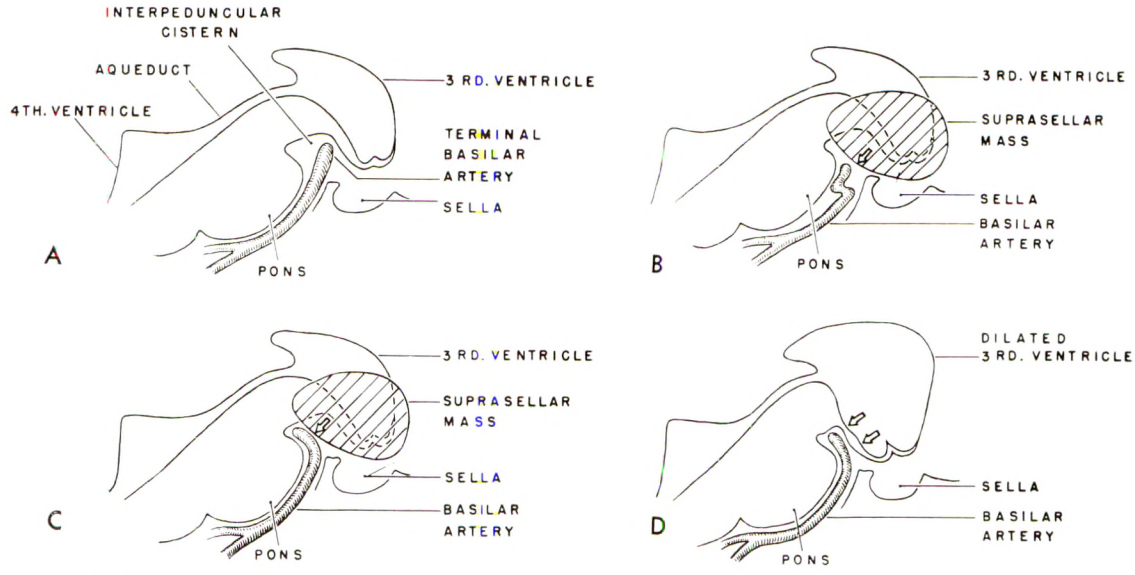



FIG. 4. (*A*) Schematic presentation of normal anatomic relationships at the tentorial notch. (*B* and *C*) Schematic presentations illustrating different deformities of the terminal basilar artery produced by suprasellar masses. (*D*) Schematic presentation illustrating deformity at terminal basilar artery produced by dilated 3rd ventricle.

tern and in Case III, the terminal basilar artery appears to be pushed back against the superior margin of the pons. Figure 4, *A–D* summarizes the types of deformities of the terminal basilar artery produced by the lesions in the reported cases.

The degree of displacement by the mass as well as the resulting configuration of the basilar artery would seem to be dictated by 3 factors: (1) length of the basilar artery segment within the interpeduncular cistern; (2) size and nature of the tumor, *i.e.*, circumscribed mass *versus* infiltrating lesion; and (3) size and configuration of the interpeduncular and pontine cisterns. Krayenbühl and Yaşargil[4] have indicated that approximately 30 per cent of basilar artery bifurcations are above the level of the posterior clinoid processes. In some instances, as shown by Krayenbühl and Yaşargil,[4] Lindgren,[5] Radner[8] and our own experience, the normal basilar artery may terminate at least 1 cm. above the superior aspect of the dorsum sellae. It would seem that a basilar artery of this kind would be most susceptible to deformation by a suprasellar lesion.

On the other hand, a short basilar artery with a termination below the level of the posterior clinoids could also be affected as in Case I, if the tumor has insinuated itself between the dorsum sellae, clivus and the pons. We postulate that there are several situations in which a tumor may be present adjacent to the terminal basilar artery without causing deformity of the terminal basilar artery, *i.e.*, when the tumor invests the terminal basilar artery and when the lesion is of insufficient size to deform the basilar artery.

It is obvious that arteriosclerotic tortuosity of the basilar artery can mimic the deformity produced by suprasellar lesions.[2,4] One hundred of our recent vertebral angiograms were surveyed in order to evaluate the configuration of the terminal basilar artery in patients where no clinical or roentgenologic evidence of suprasellar lesions could be demonstrated. Figure 5, *A–L* records a range of normal variations in the basilar artery as illustrated by 12 of 100 patients. Different levels of the termination of the basilar artery relative to the dorsum sellae are depicted. Figure 5, *K* and *L* demonstrates basilar arteries which superficially mimic the basilar arteries in the

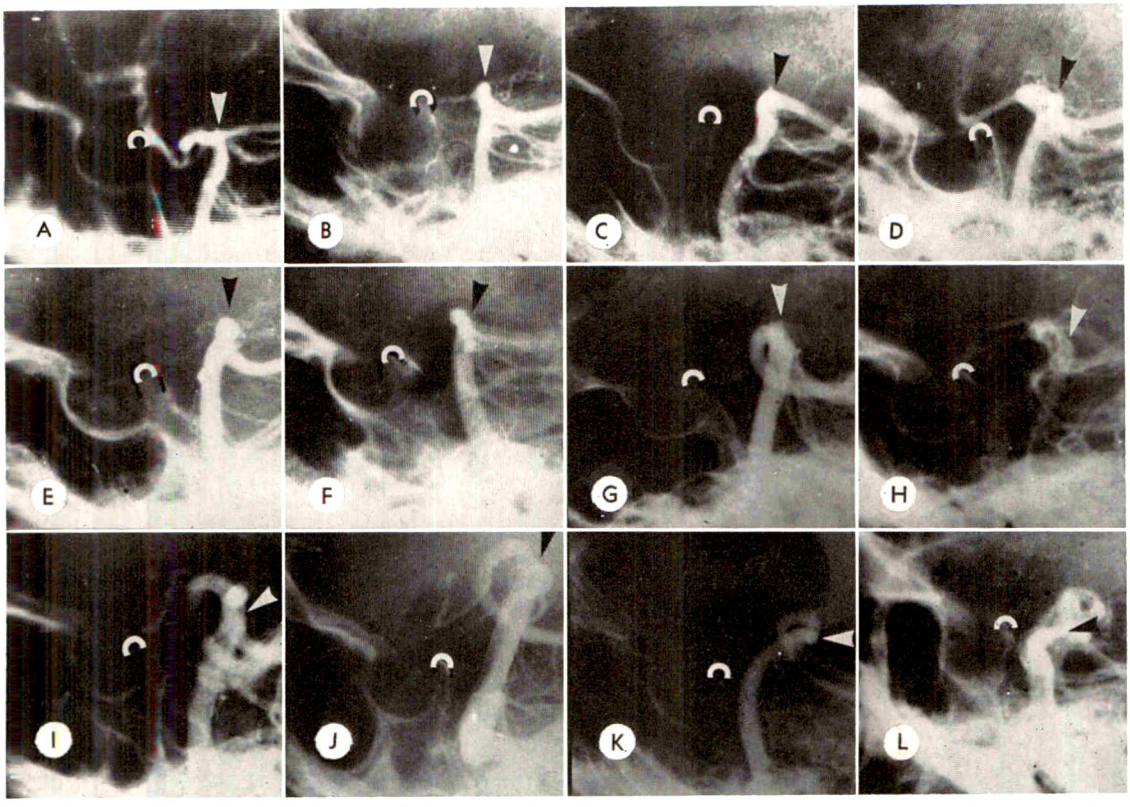



Fig. 5. (*A–L*) Twelve vertebral arteriograms selected from a "normal" series of 100 cases. Different relationships between the dorsum sellae (semi-circle) and the terminal basilar arteries (arrows) are depicted. Note the differing configurations of the basilar arteries. Only *K* and *L* simulate pathologic configurations.

cases reported by us. In Figure 5*K*, the posterior floor of the 3rd ventricle abuts on the terminal basilar artery. The 3rd ventricle, although not remarkably dilated, has seemingly compressed the terminal basilar artery against the superior belly of the pons. A similar appearance of the basilar artery is noted in Figure 6*A*. This child was shown to have aqueductal stenosis with marked enlargement of the 3rd ventricle, as demonstrated on a pantopaque ventriculogram (Fig. 6*B*). Although our present observations are limited, it seems reasonable to assume that 3rd ventricle enlargement can cause changes in the terminal basilar artery, particularly when there is an ample component of the basilar artery in the superior interpeduncular cistern.

As Lindgren[5] and others have observed, anterior cerebral hemisphere lesions can cause backward and downward displacement of the 3rd ventricle (axial herniation) and this in turn will produce a sharp posterior curve or angle in the terminal basilar artery.

Since the original observations of basilar artery deformity in our 3 cases, we have had several other cases demonstrating deformity of the basilar artery produced by 3rd ventricular lesions. In Figure 7, a colloid cyst of the 3rd ventricle is responsible for acute angulation of the terminal basilar artery. Figure 8 shows a prominent deformity of the terminal basilar artery produced by a lipoma expanding the posterior margins of the 3rd ventricle.

The authors have searched for an appropriate descriptive term for the angulation of the basilar artery described. "Concertina" or "accordion effect" as well as "hockeystick" deformity were given due consideration. The term "chordee" (angular erection of the penis secondary to gonorrheal scarring or congenital abnormal-

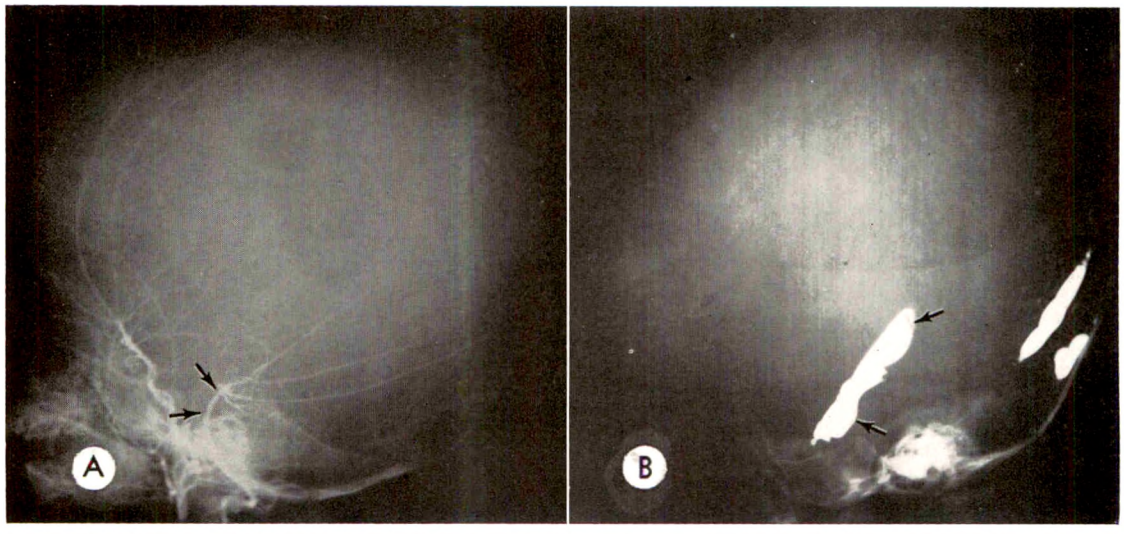



Fig. 6. (*A*) Aortic arch injection, lateral projection. The arrows point to the basilar artery which is angulated posteriorly. The child has aqueductal stenosis. (*B*) Same patient as in *A*. Brow-up lateral pantopaque ventriculogram. The arrows point to pantopaque in the posterior portion of the 3rd ventricle. Note the lack of filling of the aqueduct.

ities) was also considered. However, none of these terms seems elastic enough to encompass the various deformities noted.

## CONCLUSION

A sharp or relatively acute angulation of the terminal basilar artery may be caused by a suprasellar mass arising outside or within the 3rd ventricle, 3rd ventricular displacement and 3rd ventricular dilatation. When the terminal basilar artery has been both deformed and displaced downward into the pontine cistern, a deep midline tumor is probably present. The angular deformity of the basilar artery produced by the foregoing entities is of greater severity than that associated with advanced arteriosclerosis or hypertensive changes in this vessel. The angiographic recognition of

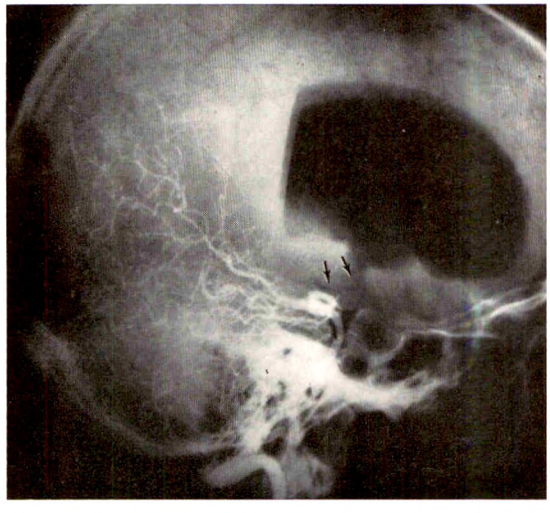


Fig. 7. Vertebral angiogram, lateral projection. There is air in the lateral ventricles and in the anterior 3rd ventricle. The arrows point to the anterior pole of a colloid cyst outlined by air in the anterior 3rd ventricle. Note the proximity to the tip of the basilar artery which is angulated and depressed.

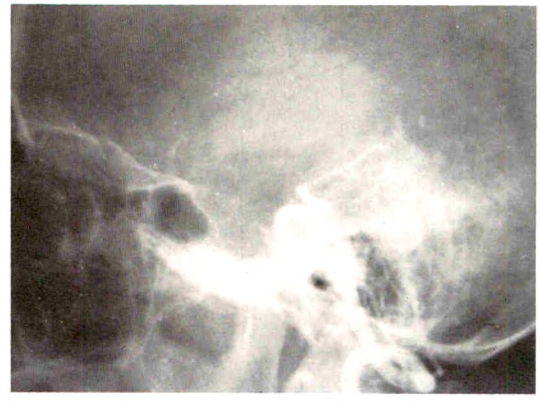


Fig. 8. Vertebral angiogram, lateral projection. Note the deformity of the terminal basilar artery. At operation the patient had a lipoma removed from within the 3rd ventricle.

abnormal configurations of the terminal basilar artery may thus serve as a stepping stone to the pneumographic confirmation of a suprasellar mass.

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# EXPERIENCE AND RESULT WITH POSTMORTEM CEREBRAL ANGIOGRAPHY PERFORMED AS ROUTINE PROCEDURE OF THE AUTOPSY*

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IN 1925 Fay[7] introduced the roentgenography of postmortem injected brain specimens for the precise demonstration of cerebral vascular structures. It is to Schlesinger's merit,[22] that a barium gelatin suspension for injection of vascular and tubular structures anywhere in the human body, post mortem, was composed.

The various postmortem vascular studies[7,11,12,20,25,31] reported in the literature are mostly directed towards one specific aim of interest. Some authors investigated single arteries and veins,[4,10,11,21,23,33] others evaluated the collateral circulation of cerebral vessels and arteriosclerotic changes of head and neck arteries.[12,24,25,28] The illustration of tumor vessels and veins was the goal of other researchers.[20] The true incidence of aneurysm cannot be better clarified than by way of postmortem injection.

During our first series of postmortem cerebral angiographic studies, we became aware of the fact that the injected brain specimen and its roentgenogram

1. present the most valuable teaching material for students and post graduates at the brain cutting session,
2. aid neuropathologists in search of small vascular lesions,
3. support the more true statistical evaluation of cerebral disease.

The injection is performed at random and is a well established procedure. Its scientific values and results are unlimited and extend beyond expectation as compared with series of uninjected brain specimens.

It is our intention to explain on hand of a few examples, the educational as well as the scientific qualities of the routinely performed postmortem cerebral angiography. At the same time, we will point out the results arrived at through the fruitful cooperation of the two supporting and leading departments, the Department of Pathology and the Department of Radiology, concerning diagnosis in any hospital.

## MATERIAL AND METHODS

In the period from June 1964, to June 1966 a total of 291 brains with attached dural linings have been removed from the cranial cavity as a routine procedure of every autopsy and the right and left carotid and vertebral arteries were injected in the fresh stage with a modified Schlesinger's[22] suspension. The modification concerns mainly the size of the particles in order to achieve filling of arteries with diameters of a fraction of a millimeter and yet not to permit passage of contrast material into the venous path.

Photographs of the base of the brain are taken immediately after, and if necessary also of the base of the skull, for reconstruction of anatomic or pathologic relations between skull and brain. One to two weeks later more extensive color photography of the fixed specimen is done for permanent recording of the exposed vessel segments as projected on the surface of the brain in the following positions:

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A. total view of base of brain
B. close up view of base (circle of Willis)
C. both lateral surfaces
D. superior surface of total brain
E. front view of brain
F. occipital area of brain.

Roentgenograms are taken subsequently:

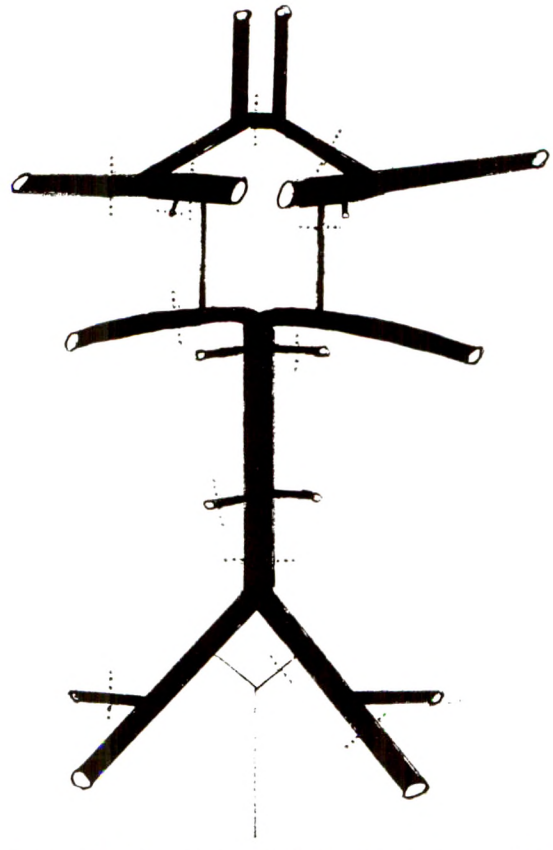
A. in base-down position
B. in base-up position
C. in lateral projection
D. in anteroposterior projection.

TABLE I

CIRCLE OF WILLIS
Frequent Deviations from the Normal

| Artery | Type | R | L | Single | Bilateral |
|---|---|---|---|---|---|
| ADS | Demonstrated | 2 | 16 | — | — |
| $ACA_1$ | Fenestration | 9 | 5 | — | — |
| $MCA_1$ | | 1 | — | — | — |
| BA | | — | — | 12 | — |
| VA | | — | 1 | — | — |
| ACCA | Absent | — | — | 1 | — |
| PCCA | | 2 | 1 | — | 3 |
| AICIA | | — | 1 | — | — |
| PICIA | | 26 | 16 | — | 5 |
| VA | | 1 | — | — | — |
| ASpA | | 24 | 21 | — | — |
| PCCA | Infundibular Widening | 13 | 18 | — | 8 |
| ICA/ACA | | 1 | — | — | — |
| BA/SCIA | | — | 1 | — | — |
| VA/PICIA | | 1 | — | — | — |
| PCA | Fetal Type | 23 | 21 | — | 17 |
| ACA | Single | — | — | 3 | — |
| ACCA | Doubling-Variation | — | — | 42 | — |
| VA | | 1 | 1 | — | — |
| $ACA_1$ | Hypoplastic | 19 | 9 | — | 7 |
| VA | | 7 | 4 | — | — |
| ACHA | Hyperplastic | 8 | 9 | — | 14 |

*Key to abbreviations:* ADS = artery of Davidoff and Schechter; ACA = anterior cerebral artery; MCA = middle cerebral artery; BA = basilar artery; VA = vertebral artery; ACCA = anterior communicating artery; PCCA = posterior communicating artery; AICIA = anterior inferior cerebellar artery; PICIA = posterior inferior cerebellar artery; ASpA = anterior spinal artery; ICA = internal carotid artery; ACA = anterior cerebral artery; SCIA = superior cerebellar artery; ACHA = anterior choroidal artery.



SKETCH I. Ideal circle of Willis. The punctate lines indicate where the measurements of the diameter of the lumen of the vessels were made.

It is understood that the specimen is handled with the greatest of care. The measurements of the vessel diameters are recorded on preprinted sheets in a manner described later. This particular undertaking originated from the idea to investigate and establish the mean value of vessel diameters on the base of the brain in a large series for cerebral blood flow studies in the near future.[14] The ratios of certain vessel diameters and their significance in arteriographic interpretation have been previously presented. For instance, we found the normal anterior cerebral artery/internal carotid artery (ACA/ICA) ratio[30] (Table v) of vessel diameter to be 0.459 post mortem and 0.5241 pre mortem and learned that a drop of postmortem ACA/ICA ratio to 0.28 is seen when there is evidence of hypoplasia, mainly of $A_1$. For this reason we

TABLE II a

DIAMETER OF ARTERIES OF BASE OF BRAIN
Anterior Circulation

| Artery | Side | No. | Mean in mm. | Standard Deviation | Range Middle: 60% |
|---|---|---|---|---|---|
| ICA | R | 223 | 3.63 | .9211 | |
| | | 209 | 3.62 | | 2.70–4.55 |
| | L | 224 | 3.62 | .8911 | |
| | | 209 | 3.65 | | 2.72–4.51 |
| MCA | R | 224 | 2.49 | .6120 | |
| | | 209 | 2.52 | | 1.87–3.10 |
| | L | 223 | 2.55 | .6103 | |
| | | 208 | 2.55 | | 1.94–3.16 |
| ACA | R | 224 | 1.76 | .5895 | |
| | | 210 | 1.76 | | 1.17–2.34 |
| | L | 223 | 1.89 | .5553 | |
| | | 219 | 1.89 | | 1.33–2.44 |
| ACCA | | 219 | 1.39 | .7212 | |
| | | 212 | 1.33 | | .67–2.11 |
| | Length | 211 | 2.72 | 1.9850 | |
| | | 198 | 2.34 | | .73–4.70 |
| ACHA | R | 219 | .39 | .2131 | |
| | | 209 | .36 | | .17–.60 |
| | L | 220 | .38 | .2405 | |
| | | 208 | .35 | | .13–.62 |
| PCCA | R | 219 | .94 | .6358 | |
| | | 211 | .88 | | .30–1.58 |
| | L | 220 | .91 | .6257 | |
| | | 209 | .82 | | .28–1.54 |

have applied the same criteria in the evaluation of single arteries, in this series.[16] According to our previous results we considered all anterior cerebral arteries with a diameter of 1.0 mm. or less hypoplastic and recorded in this series 8.63 per cent on the right, 4.09 per cent on the left, and 3.18 per cent bilaterally (Table 1). In general, the measurements are read 3.0 mm. from the origin or bifurcation of a vessel (Sketch 1). This concerns the internal carotid artery, the anterior cerebral artery, the anterior choroidal artery (ACHA), the posterior communicating artery (PCCA), the middle cerebral artery (MCA), the posterior cerebral artery (PCA), the superior cerebellar artery (SClA), the anterior inferior cerebellar artery (AIClA), the posterior inferior cerebellar artery (PIClA), the anterior spinal artery (ASpA). The diameter of the basilar artery (BA) is taken approximately 3.0 mm. from the junction of both vertebral arteries (VA). The vertebral arteries are measured 3.0 mm. proximal to the origin of the posterior inferior cerebellar artery, and so on. Also the lengths of the basilar artery, and the anterior communicating artery (ACCA), the $A_1$, and the posterior communicating artery (PCCA), the pars pos-

TABLE II b

DIAMETER OF ARTERIES OF BASE OF BRAIN
Posterior Circulation

| Artery | Side | No. | Mean in mm. | Standard Deviation | Range Middle: 60% |
|---|---|---|---|---|---|
| BA | | 228 | 3.26 | 1.0225 | |
| | | 217 | 3.29 | | 2.7–4.28 |
| | Length | 227 | 31.64 | 6.8718 | |
| | | 219 | 32.18 | | 24.8–38.50 |
| PCA | R | 224 | 1.95 | .4531 | |
| | | 212 | 1.96 | | 1.49–2.40 |
| | L | 227 | 1.96 | .5121 | |
| | | 218 | 1.98 | | 1.44–2.47 |
| S Cl A | R | 227 | 1.12 | .3852 | |
| | | 213 | 1.09 | | .73–1.50 |
| | L | 227 | 1.11 | .3872 | |
| | | 212 | 1.08 | | .72–1.49 |
| A ICl A | R | 212 | .82 | .4399 | |
| | | 214 | .79 | | .38–1.26 |
| | L | 220 | .79 | .4291 | |
| | | 209 | .73 | | .36–1.21 |
| P ICl A | R | 178 | 1.23 | .5318 | |
| | | 174 | 1.25 | | .70–1.76 |
| | L | 190 | 1.22 | .5632 | |
| | | 188 | 1.20 | | .65–1.78 |
| VA | R | 222 | 2.51 | 1.5861 | |
| | | 221 | 2.42 | | .92–4.09 |
| | L | 223 | 2.60 | .9830 | |
| | | 207 | 2.62 | | 1.60–3.60 |
| ASpA | R | 197 | .19 | .1188 | |
| | | 187 | .17 | | .07–.30 |
| | L | 190 | .16 | .0927 | |
| | | 182 | .15 | | .06–.25 |

For Key to Abbreviations see Table 1.

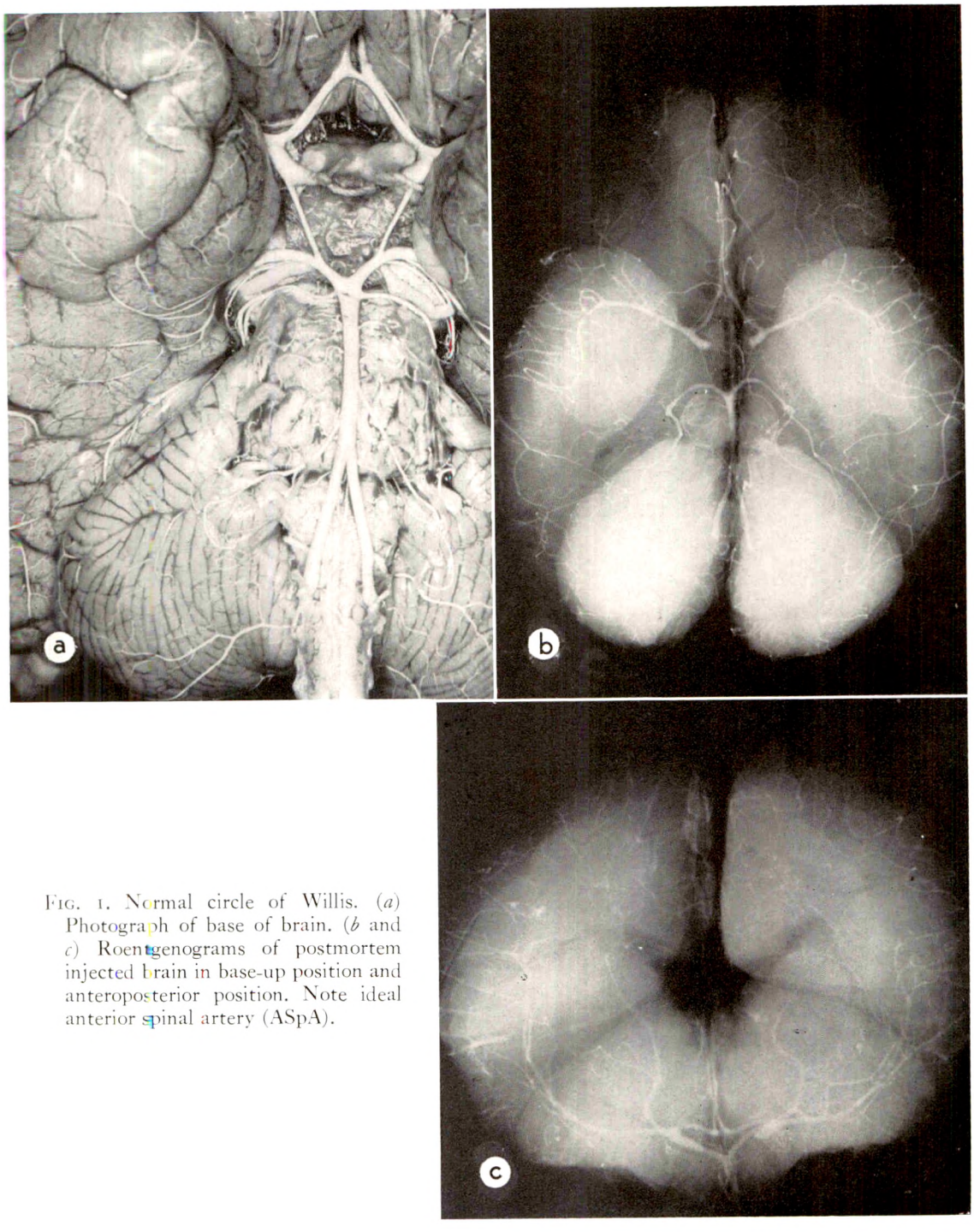



FIG. 1. Normal circle of Willis. (*a*) Photograph of base of brain. (*b* and *c*) Roentgenograms of postmortem injected brain in base-up position and anteroposterior position. Note ideal anterior spinal artery (ASpA).

terior circularis or $PCA_1$ were routinely reported. In order to avoid a misunderstanding of reports or a misinterpretation of photographs and roentgenograms, a drawing is obtained of each circle of Willis and anomalous or pathologic or otherwise unusual findings are sketched. The results of these measurements enable us to illustrate an ideal circle of Willis with the mean value from 220 completely injected and worked

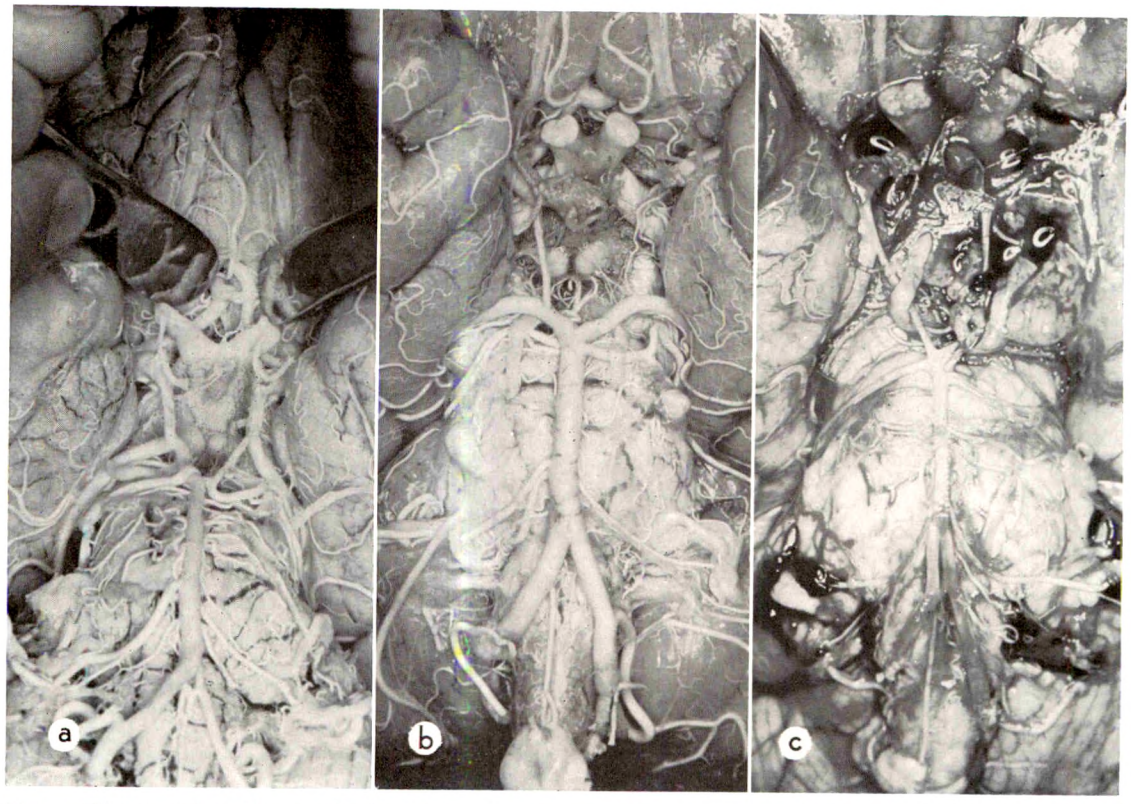



FIG. 2. Photographs of base of brain. (*a*) Small aneurysm of anterior cerebral artery (ACA) at junction of right ACA and anterior communicating artery (ACCA) and fenestration of ACCA. Bilateral fetal type of posterior cerebral artery (PCA), right incomplete type, left complete type. Normal ASpA. (*b*) Normal sized left anterior choroidal artery (ACHA), originating from internal carotid artery (ICA) and disappearing under the uncus of temporal lobe. Absent left posterior communicating artery (PCCA). Prominent right ASpA and remnant left. (*c*) Fenestration of basilar artery (BA). Prominent right ASpA.

up brains out of the series of 291 injected specimens (Sketch 1; Tables IIa and IIb; Fig. 1, *a*, *b* and *c*).

## RESULTS

The deviation from the normal pattern of circle of Willis is observed in more than 50 per cent and extensive series are reported in the literature.[1,3,5,13,17,18,26] These presentations, of course, are based in the majority on the observation of uninjected brain specimens, [17] when it is difficult to make statements about absent or hypoplastic arteries, according to the authors' personal experience. For instance, a very hypoplastic but patent posterior communicating artery may tear off at time of removal and be lost in the tangle of small perforant arteries. Only the injection with contrast material would shed light on such accident because spillage from the severed artery indicates that such a vessel existed.

The absence of arteries is most frequent in the posterior circulation with the highest incidence placed on the posterior inferior cerebellar arteries. The anterior spinal artery occupies a special position inasmuch as it is not absent bilaterally (Fig. 12, *a* and *b*). This vessel takes off from each vertebral artery in the range from 0.5–1.0 cm. proximal to the junction of the right and left vertebral artery and forms, in the normal, a single artery in the median ventral fissure of the spinal cord on its caudal course. However, there are a great number of variations.[21] The posterior inferior cerebellar artery is bilaterally missing in 2.27 per cent, and absent on the right in 11.81

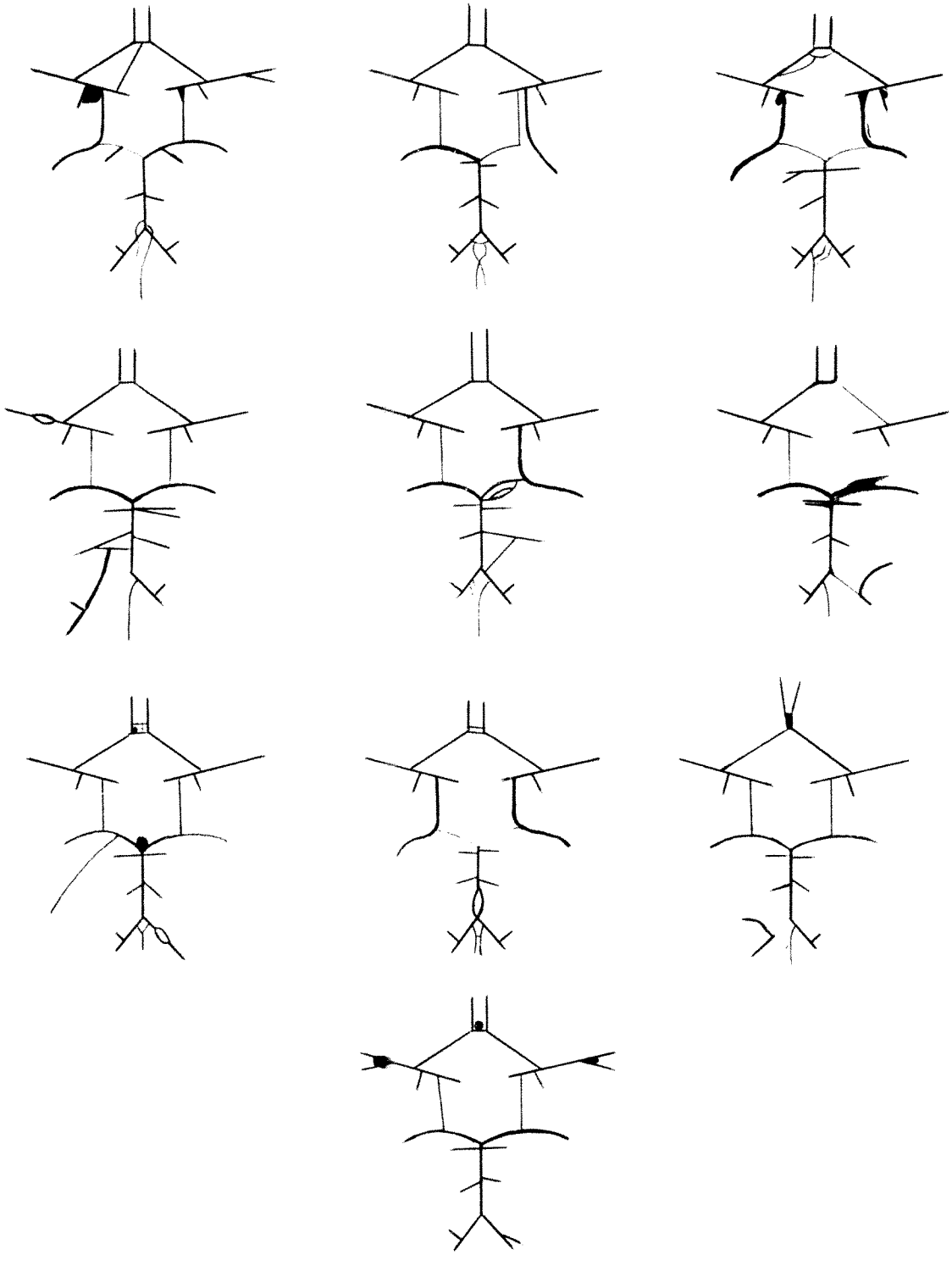
per cent and on the left in 7.27 per cent (Fig. 3, *a* and *b*). The anterior spinal artery frequently emerges from one site only; *i.e.*, from the right vertebral artery in 10.9 per cent and from the left in 9.45 per cent. The ideal configuration of a confluent right and left anterior spinal artery in the anterior median raphe (Fig. 1, *a* and *b*; and 2 *a*) is often replaced by a strong right or left single anterior spinal artery and one or more small remnants on the opposite side (Fig. 2, *b* and *c*). The anterior communicating artery was definitely absent in only 1 case. The term absent is interpreted as not permitting flow of contrast material to the contralateral side via the anterior communicating artery. The posterior communicating artery was counted as missing in 0.9 per cent on the right and 0.45 per cent on the left (Fig. 2 *b*) and in 1.36 per cent bilaterally. The anterior inferior cerebellar artery failed to be present in only 1 case on the left. The vertebral artery, too, did not show any "Anlage" in one single instance; however, it was found to be hypoplastic in 3.18 per cent on the right (Fig. 10), and in 1.81 per cent on the left (Fig. 9, *a* and *b*). Doubling of VA was discovered twice (Sketch II; Table 1).

Likewise, the fenestration of an artery is sometimes first picked up on the postmortem roentgenogram and then confirmed by close inspection and cross section of lumen. The fenestration of the basilar artery is noted in 5.26 per cent according to our present series (Fig. 2 *c*; and 9, *a* and *b*). Fenestration of the posterior cerebral artery, superior cerebellar artery, anterior inferior cerebellar artery, posterior inferior cerebellar artery, and vertebral artery is less frequent and is of particular interest when the window is small and forms a tight pulsating ring around an emerging cranial nerve. Duplication channels of middle cerebral artery and anterior cerebral artery are rare and more or less curiosities concerning blood flow.

Doubling or even tripling of the anterior communicating artery with spider-like or so-called V or Y shaped connections (Fig. 2 *a*; and 12, *a* and *b*), is a rather common finding on postmortem injections, *i.e.*, 19.06 per cent. Not too seldom it causes resemblance to an aneurysm of the anterior communicating artery (Fig. 3, *a* and *b*) and the diagnosis is usually clarified by oblique roentgenographic projections.

The so-called fetal type of posterior cerebral artery, complete (Fig. 3, *a*, *b* and *c*) or incomplete (Fig. 2 *a*), unilateral or bilateral (Fig. 2 *a*; and 4, *a* and *b*), extra- or intracircular, is not too unusual and is observed in 10.44 per cent of the specimens on the right, 9.54 per cent on the left and in 7.72 per cent bilaterally (Fig. 3, *a*, *b* and *c*); Sketch III). The term fetal type of posterior cerebral artery is applied when the posterior cerebral artery originates from the internal carotid artery and receives its blood supply mainly from the anterior circulation.

The infundibular widenings of the posterior communicating artery are routinely seen and recorded in 5.90 per cent on the right and 8.18 per cent on the left with 3.63 per cent occurrence bilaterally. The junction of the internal carotid artery and posterior communicating artery is a predestinated location for infundibular widenings[8,19] (Fig. 10). However, occasionally we noticed one at the junction of the internal carotid artery and anterior cerebral artery, at the junction of the basilar artery and superior cerebellar artery, and at the junction of the vertebral artery and posterior inferior cerebellar artery. These funnel-shaped prominences sometimes raise the question of an aneurysm (Fig. 4, *a* and *b*). The differentiation of infundibular widenings and aneurysm at the junction of the posterior communicating artery and internal carotid artery is more exact in injected condition. Only an asymmetric saccular outpouching at the offspring of the posterior communicating artery is called an aneurysm (Fig. 4, *a* and *b*; Sketch IV). In our extensive series of more than 700 postmortem injected brain specimens, we did not observe a bleeding from an infundibular widening in contrast to true aneurysm at the same



SKETCH II. Various types of unusual circles of Willis.

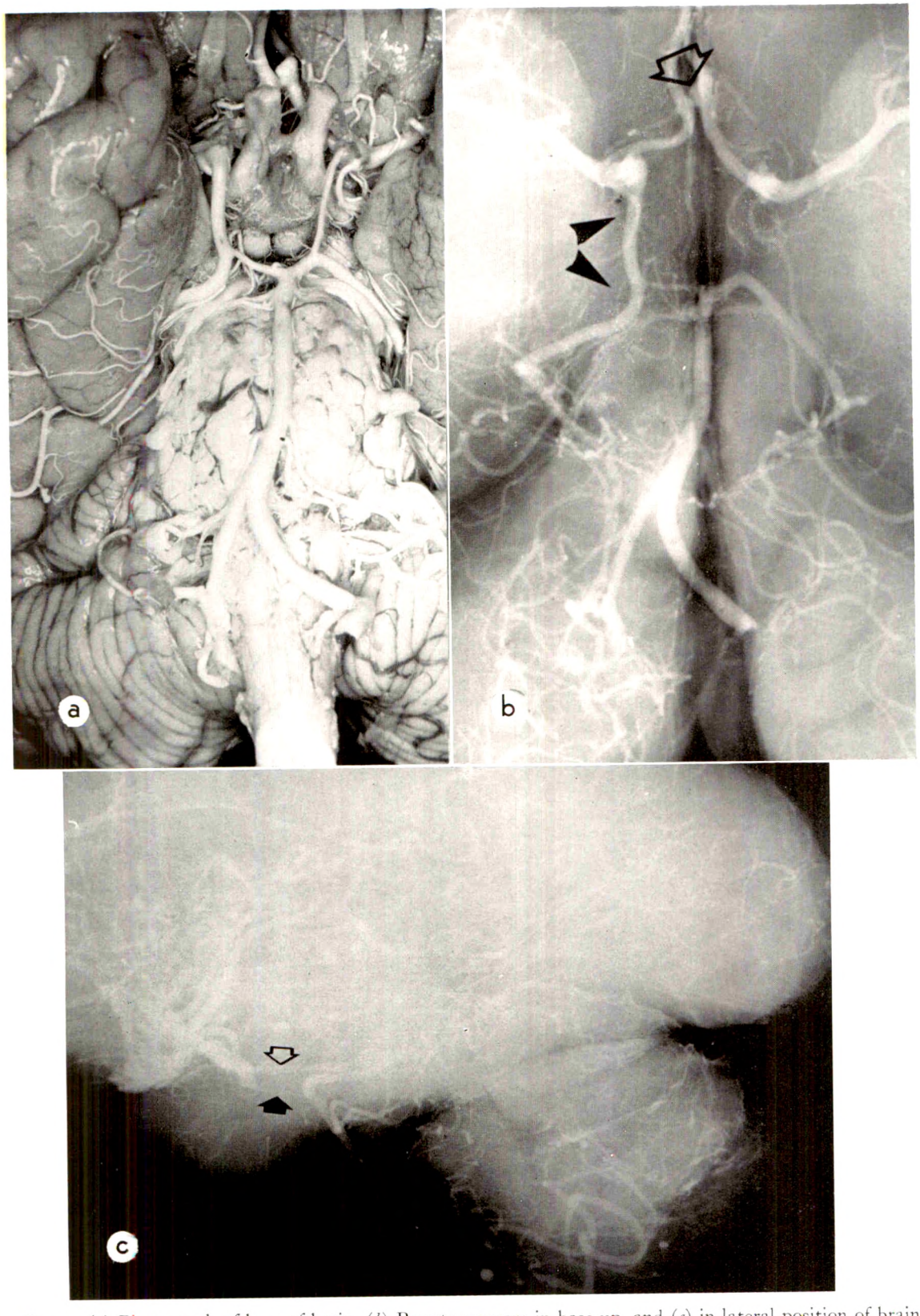



FIG. 3. (*a*) Photograph of base of brain. (*b*) Roentgenogram in base-up, and (*c*) in lateral position of brain specimen. Complete right fetal type PCA, left normal PCA. Aneurysm of ACCA. Hyperplastic left ACHA. Absent posterior inferior cerebellar artery (PICIA), left.

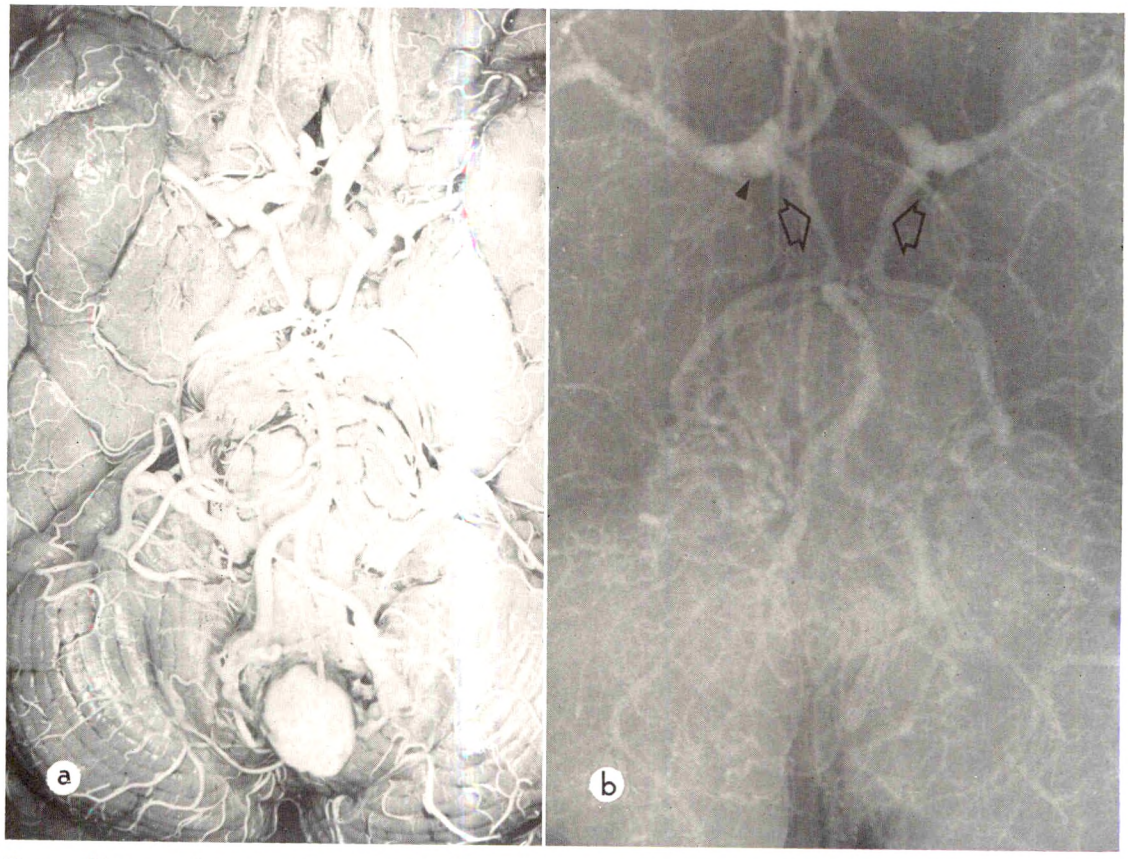
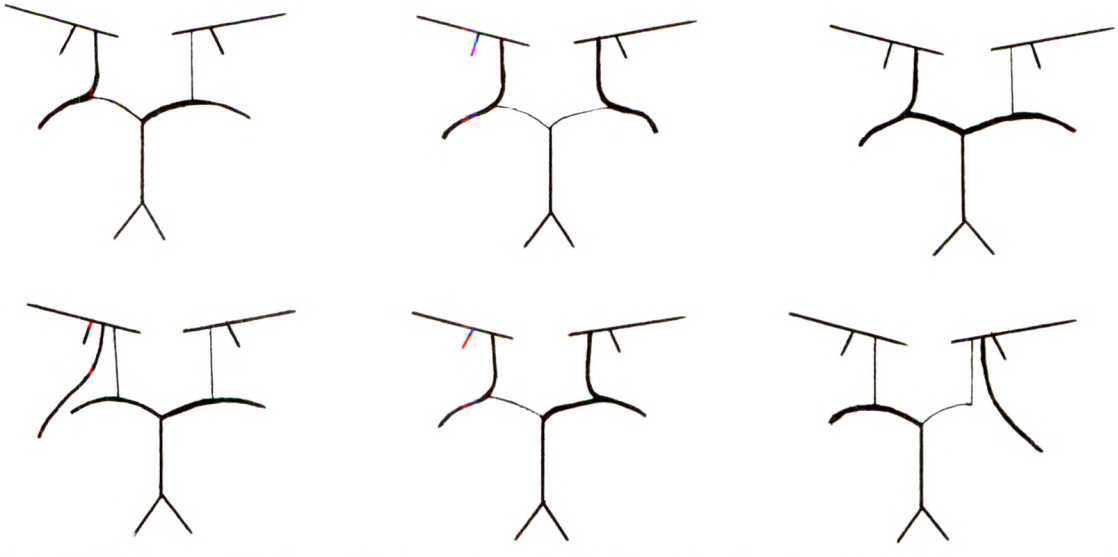
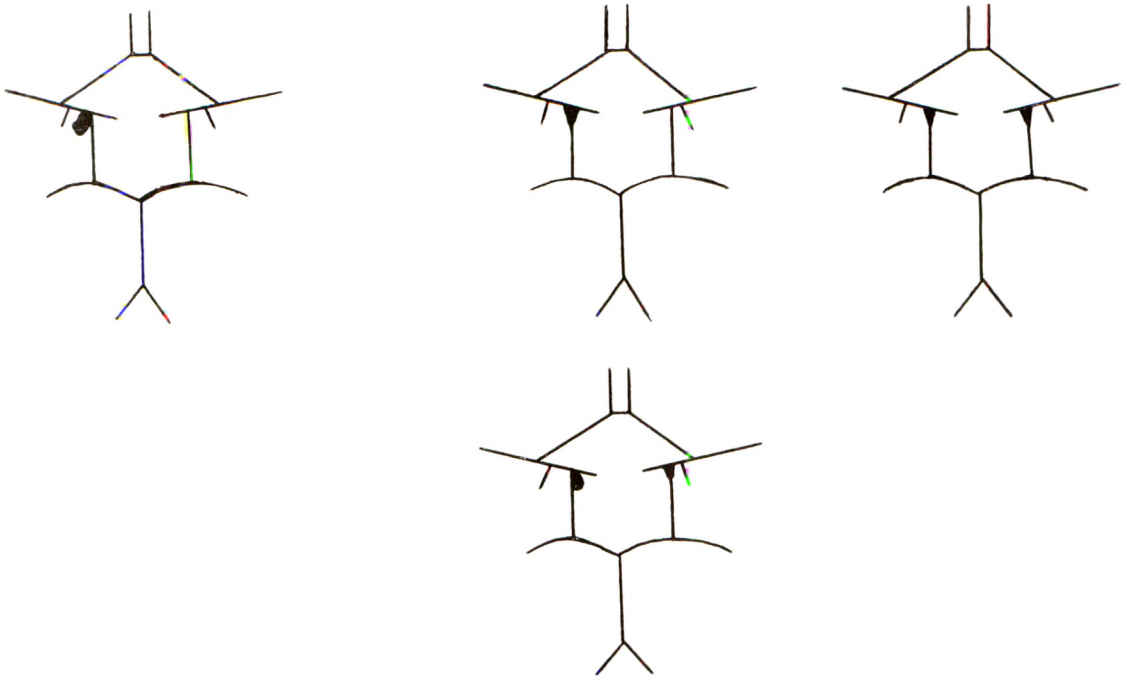



FIG. 4. Photograph and roentgenogram in base-up position of injected brain. (*a* and *b*) Complete bilateral fetal type of PCA. Aneurysm of ACCA at junction of right ACA and ACCA. Aneurysm at junction of ICA and PCCA.



SKETCH III. Types of fetal posterior cerebral artery (PCA). *Upper left:* Right fetal PCA, complete and intracircular. *Upper center:* Bilateral fetal PCA, complete and intracircular. *Upper right:* Right fetal PCA, incomplete and intracircular. *Lower left:* Right fetal PCA, extracircular with paired PCA. *Lower center:* Bilateral fetal PCA, intracircular, left incomplete. *Lower right:* Left fetal PCA, extracircular and isolated.



Sketch IV. Infundibular widening and aneurysms at posterior communicating artery/internal carotid artery (PCCA/ICA) junction. *Upper left:* Aneurysm. *Upper center:* Infundibular widening. *Upper right:* Infundibular widening. *Lower center:* Aneurysm and infundibular widening.

site.[19] It is therefore necessary to recognize these characteristics which nowhere can be taught so impressively by comparison of postmortem roentgenograms and specimens (Sketch IV).

Table III

ANEURYSM OF 2 INJECTION SERIES

| | AECM* 152 Brains | | UMMC† 178 Brains | |
|---|---|---|---|---|
| | No. of Patients | No. of Aneurysms | No. of Patients | No. of Aneurysms |
| Total | 22 | 31 | 20 | 25 |
| | 14 | 1 | 16 | 1 |
| | 7 | 2 | 3 | 2 |
| | 1 | 3 | 1 | 3 |

| Anterior Circulation 82% | Posterior Circulation 18% |
|---|---|
| Female:Male 40%:60% | Female:Male 16.7%:83.3% |

* AECM = Albert Einstein College of Medicine.
† UMMC = University of Missouri Medical Center.

Many of the cerebral vessels have provided us with significant information. The anterior choroidal artery originating from the internal carotid artery presents in over 73 per cent a normal diameter of 0.1 to 0.3 mm. (Fig. 2 *b*). Only 13 per cent are hyperplastic with a diameter of more than 0.6 mm. (Fig. 3, *a* and *b*); 1.0 mm. is the largest diameter for anterior choroidal arteries noted by us. Furthermore, we can confirm our previous statements, that hyperplastic anterior choroidal arteries are usually blood suppliers for cerebral lesions of any type and should be strongly considered as such (Table I and Table IV; Fig. 5, *a* and *b*).

*Comment.* The findings described are documentary for the reason that all specimens are removed and injected by the same person and the roentgenograms of specimens are critically reviewed by the authors. If necessary, additional roentgenograms or laminagrams are obtained. Prior to brain cuttings an interpretation is given, and discussions of important findings with

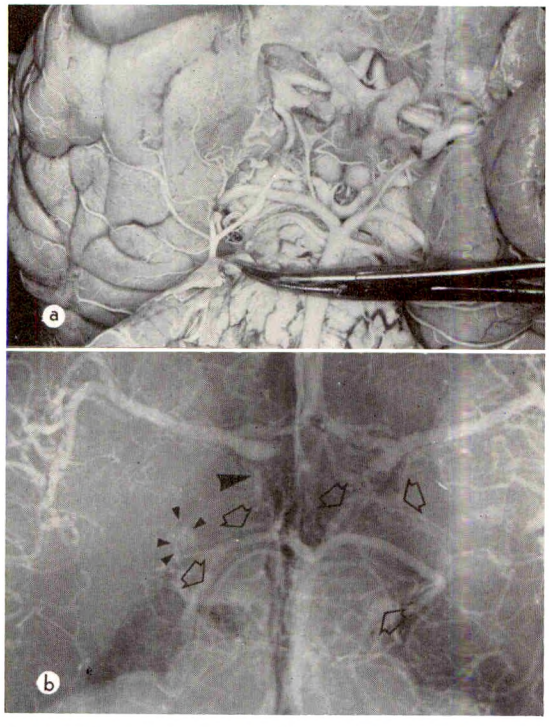


FIG. 5. Arteriovenous malformation. (*a*) Close up photograph of base of brain. (*b*) Base-down roentgenogram of injected brain.

TABLE IV

DIAMETER OF ANTERIOR CHOROIDAL ARTERY
(Post Mortem—Including Wall)
162 cases

| Diameter (mm.) | Per Cent of Cases |
|---|---|
| 0.1 | 11.9 |
| 0.2–0.5 | 73.4 |
| 0.6–0.9 | 13.5 |
| 1.0 | 1.2 |
| above 1.0 | no |

introduction of current literature are presented for the benefit of the students and residents.

## TEACHING VALUE OF BRAIN CUTTING CONFERENCE

This brings us to an important didactic approach of our brain cutting conference. The brains are sliced in most instances on the basis of superficial macroscopic and postmortem angiographic findings in coronal, sagittal, or horizontal planes. According to our experience, we believe that this

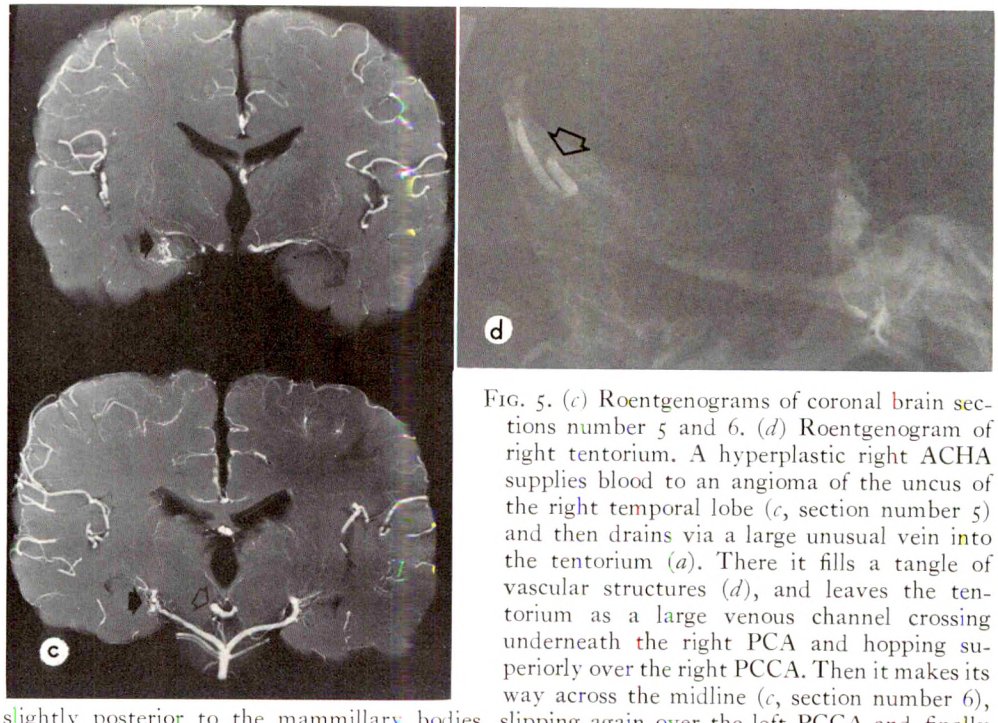


FIG. 5. (*c*) Roentgenograms of coronal brain sections number 5 and 6. (*d*) Roentgenogram of right tentorium. A hyperplastic right ACHA supplies blood to an angioma of the uncus of the right temporal lobe (*c*, section number 5) and then drains via a large unusual vein into the tentorium (*a*). There it fills a tangle of vascular structures (*d*), and leaves the tentorium as a large venous channel crossing underneath the right PCA and hopping superiorly over the right PCCA. Then it makes its way across the midline (*c*, section number 6), slightly posterior to the mammillary bodies, slipping again over the left PCCA and finally joining the left basilar vein of Rosenthal (*a* and *b*).

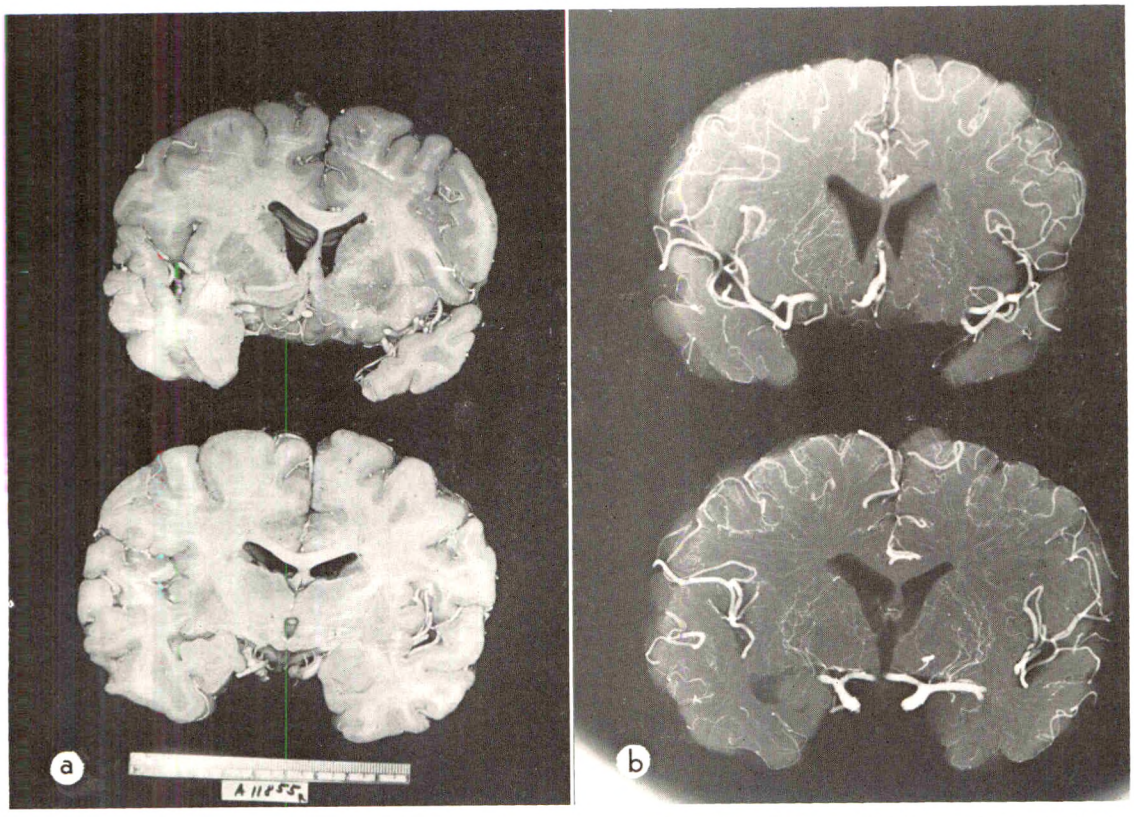



Fig. 6. Triangle of Reil. (*a* and *b*) Photographs and roentgenograms of coronal brain sections number 4 and 5, showing the distribution of the middle cerebral artery (MCA) within the island of Reil. Note also the magnificent pattern of the cortical, nuclear and perforant arteries.

represents a valuable mode of education to tridimensional understanding of cerebral anatomy and pathology (Fig. 6, *a–f*). It is an excellent way to appreciate relations of vessels to gyri, sulci and nuclei, and also to recognize the relations of vascular structures to the ventricle itself. The triangle of Reil,[32] formed by the branches of the middle cerebral artery within the island of Reil, is essential for diagnosis in cerebral arteriography. Its features, hardly ever completely realized by the students, are defined figuratively with the tridimensional method of brain sectioning; this helps to elucidate the alteration of the triangle by various impacts due to space-occupying lesions.

This systematic teaching and comparison of injected specimens with pre- and postmortem arteriograms facilitates the understanding of related clinical symptoms and neuroradiologic findings. The pathologic findings and differential diagnosis are discussed by the neuropathologist. It is clear, that this combined effort results in a complete postmortem brain evaluation.

After the brain cutting session, the brain slices are roentgenographed and photographed. Again the roentgenograms are carefully evaluated for vascular anatomy and searched for tiny lesions unidentified macroscopically.

### ANEURYSMS

The first case (Fig. 7, *a–e*), in particular, is an example for pathologic processes demonstrated by roentgenography of brain sections and confirmed by microscopy. An arteriovenous malformation was removed completely at surgery and its evacuation confirmed by postoperative arteriography in a 13 year old male. Two years later, a

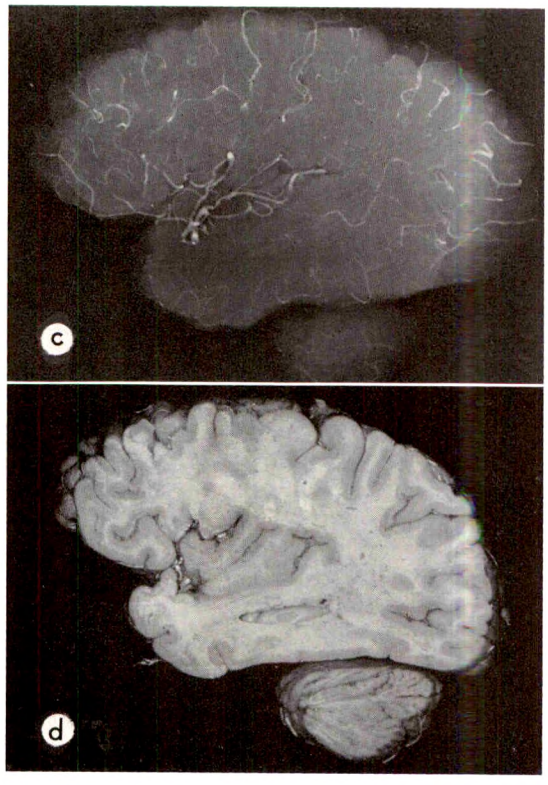



FIG. 6. (*c* and *d*) Roentgenogram and photograph of sagittal brain section number 3 from left. The angiographic triangle of Reil is formed by the branches of the MCA in the island of Reil.

massive intracerebral bleeding occurred, which ended fatally. Only a single brain section demonstrated on roentgenographic study a small conglomeration of vessels, which proved to be a remnant of the angioma hidden in a large hematoma.

The second case, a 9 year old boy, admitted comatose with grossly bloody spinal fluid, died soon thereafter. A diffuse left peripheral subarachnoid bleeding was observed; no aneurysm of the circle of Willis or of the posterior circulation was found. On postmortem roentgenographic examination an aneurysm of the parietal branch of the left middle cerebral artery was discovered in a large intracerebral hematoma (Fig. 8, *a* and *b*).

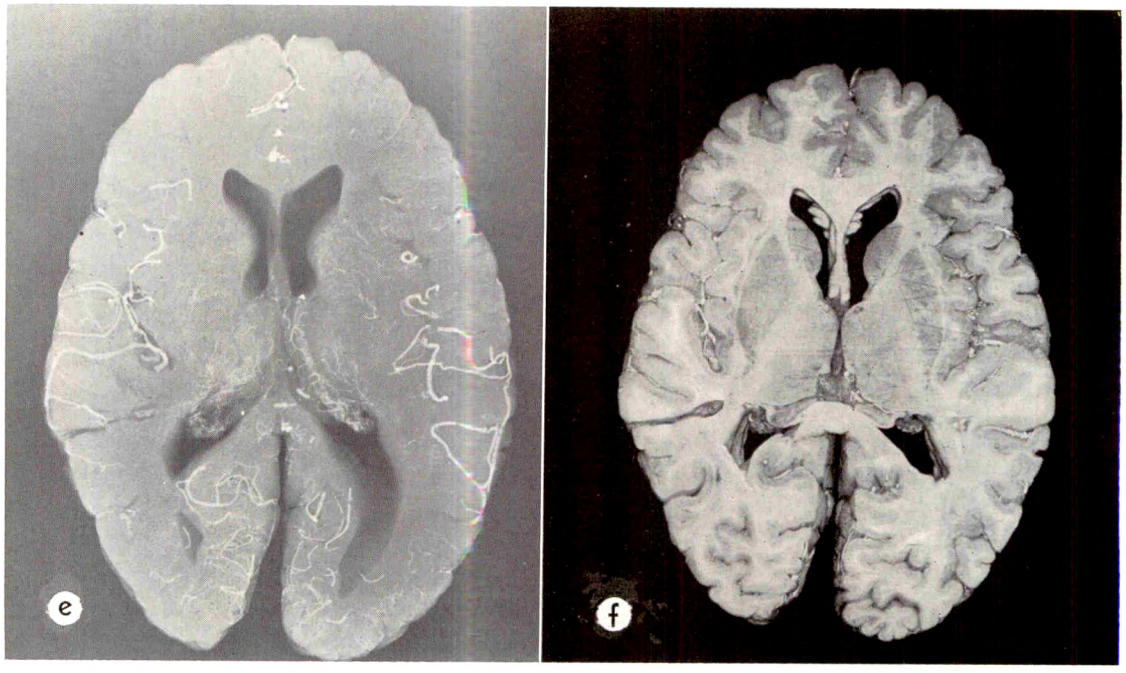



FIG. 6. (*e* and *f*) Roentgenogram and photograph of horizontal brain section number 5 from top. The horizontal arm of the triangle of Reil is here formed by the turn of the branches of the MCA out of the island of Reil. Note the fine vessels of the choroid plexus and of the nuclei.

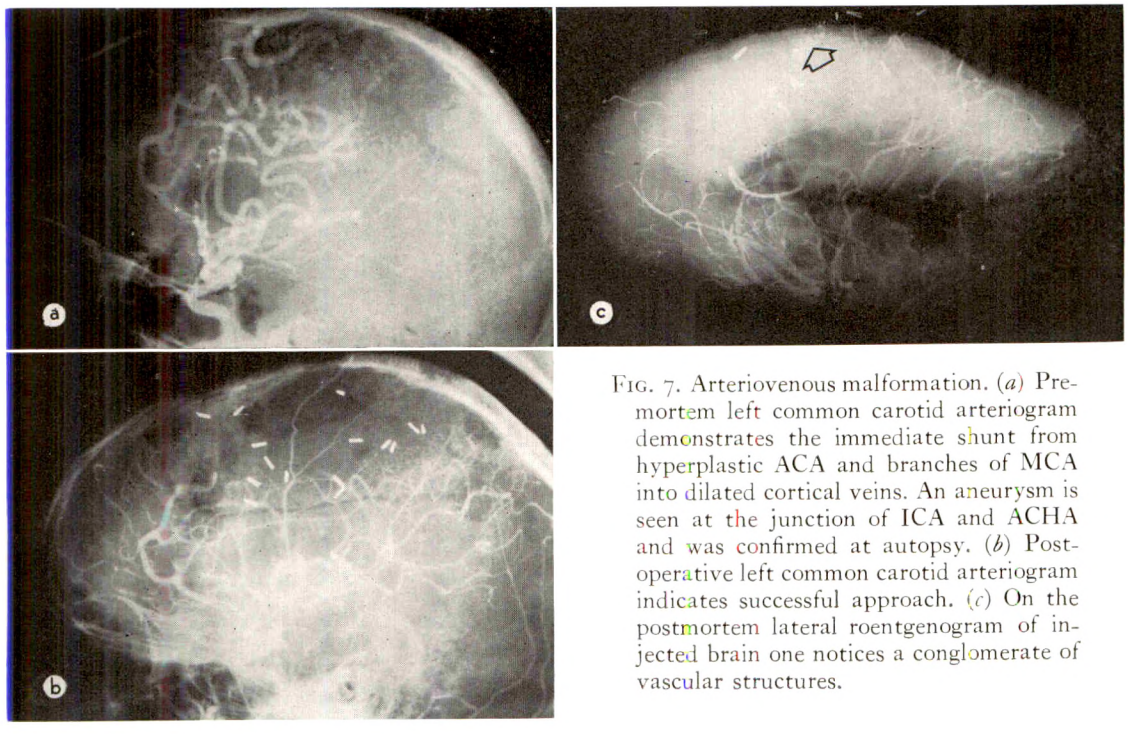



Fig. 7. Arteriovenous malformation. (*a*) Premortem left common carotid arteriogram demonstrates the immediate shunt from hyperplastic ACA and branches of MCA into dilated cortical veins. An aneurysm is seen at the junction of ICA and ACHA and was confirmed at autopsy. (*b*) Postoperative left common carotid arteriogram indicates successful approach. (*c*) On the postmortem lateral roentgenogram of injected brain one notices a conglomerate of vascular structures.

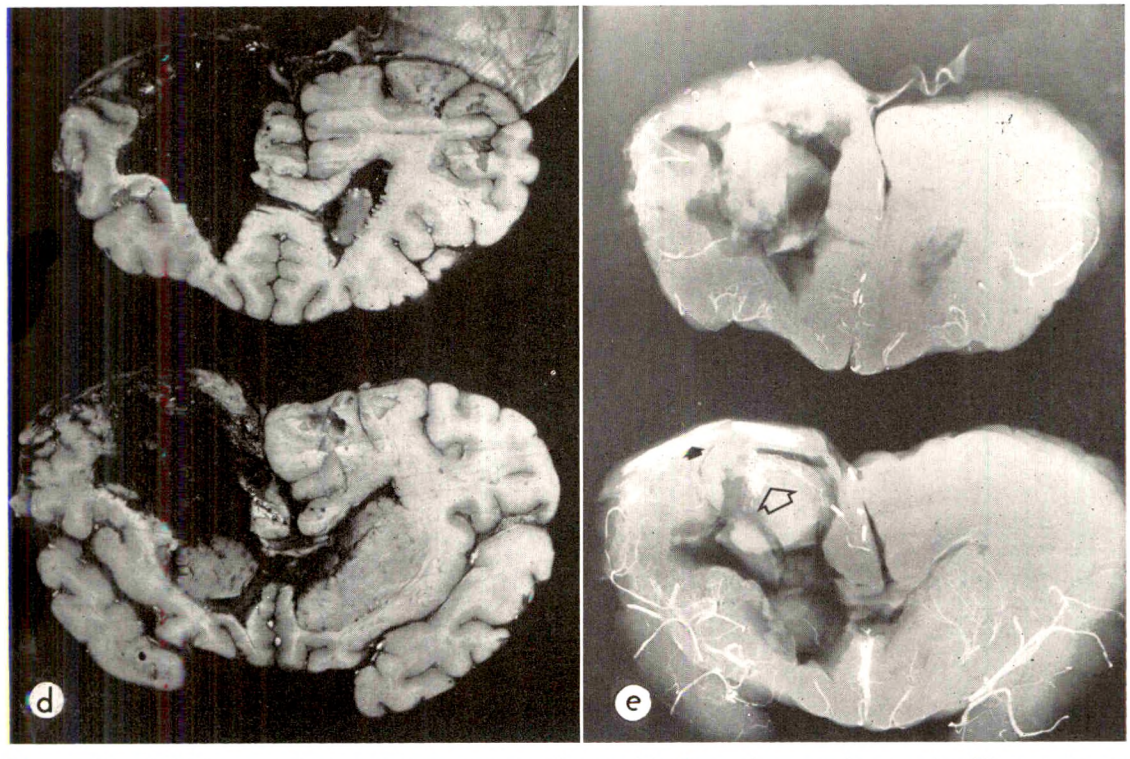



Fig. 7. (*d* and *e*) Photographs and roentgenograms of coronal brain sections number 3 and 4. The roentgenograms illustrate the tangles of angiomatous vessels as cause of fatal intracerebral bleeding.

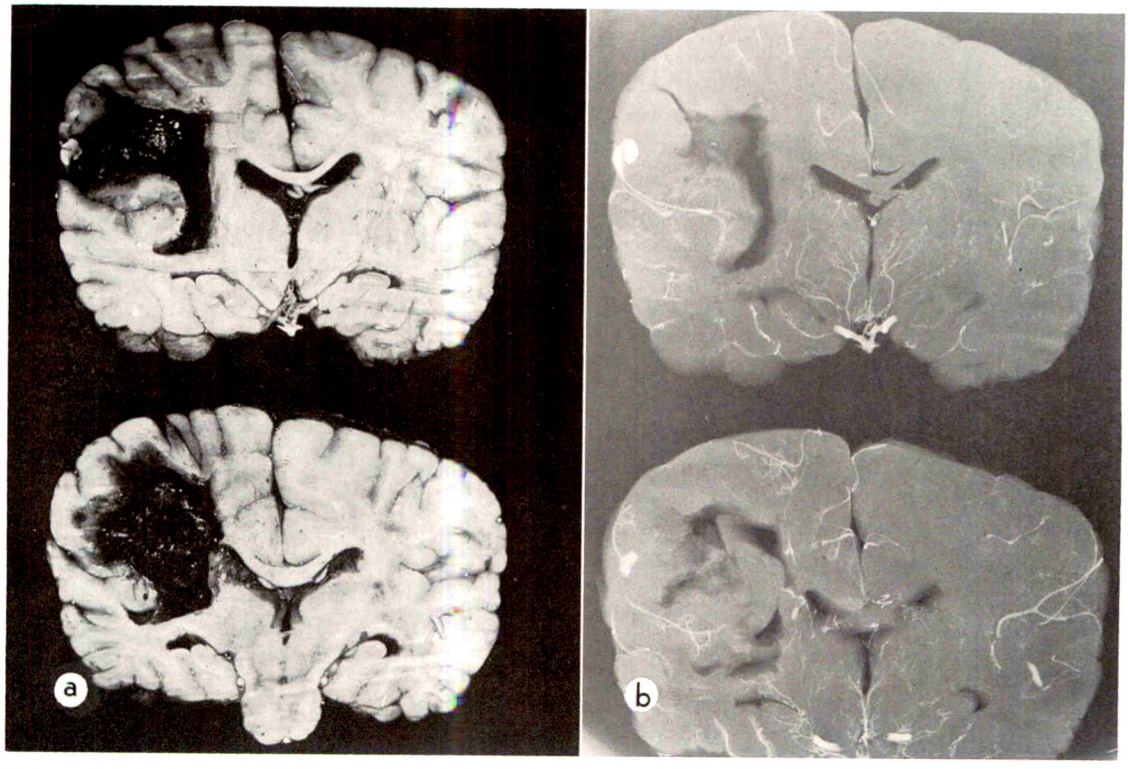



FIG. 8. Aneurysm of parietal branch of MCA. (*a* and *b*) Photographs and roentgenograms of coronal brain sections number 6 and 7. Arterial entrance and exit of aneurysm is clearly demonstrated on the roentgenograms; only part of aneurysm is filled with contrast material, the remainder contains a blood clot. The aneurysm could not be identified in uninjected condition, for it was hidden in a hematoma and its sac rusty from bleeding.

*Comment.* Needless to say, the evaluation of the frequency of occurrence of aneurysm is superior by this method. In 1966, the authors have demonstrated two different population series of postmortem cerebral angiograms[31] (Table III). The results in exact detail were published and should be mentioned only briefly. The over-all rate of aneurysms of our combined series is 15 per cent; 82 per cent are found in the anterior circulation and 18 per cent in the posterior circulation. The ratio female:male is 40%:60% in the anterior circulation. These figures are considerably higher than the 21 per thousand reported by DuBoulay[6] in 1965. Riggs and Rupp[18,27] reviewed 1,437 circles of Willis and reported an incidence of 10 per cent. It is obvious that this altered method of investigation offers much more reliable data.[15] Small aneurysms (Fig. 12, *a* and *b*; 10; 2 *a*; 3, *a* and *b*; and 4, *a* and *b*) are usually collapsed at the time of autopsy and stand out only when injected with contrast material.

TABLE V

AVERAGE RATIO OF VESSEL DIAMETER

| | ACA/ICA | MCA/ICA | ACA/MCA/ICA |
|---|---|---|---|
| Angiogram | 0.52 | 0.74 | 1.26 |
| Post Mortem | 0.46 | 0.68 | 1.14 |
| Difference | 0.06 | 0.06 | 0.12 |

ACA = anterior cerebral artery; ICA = internal carotid artery; MCA = middle cerebral artery.

### CAROTID-BASILAR ANASTOMOSIS

The carotid-basilar anastomosis[2,3,5,29] presents a category of interesting cerebral vascular structures or remnants of channels persistent beyond fetal life. Numerous persistent primitive anastomoses are re-

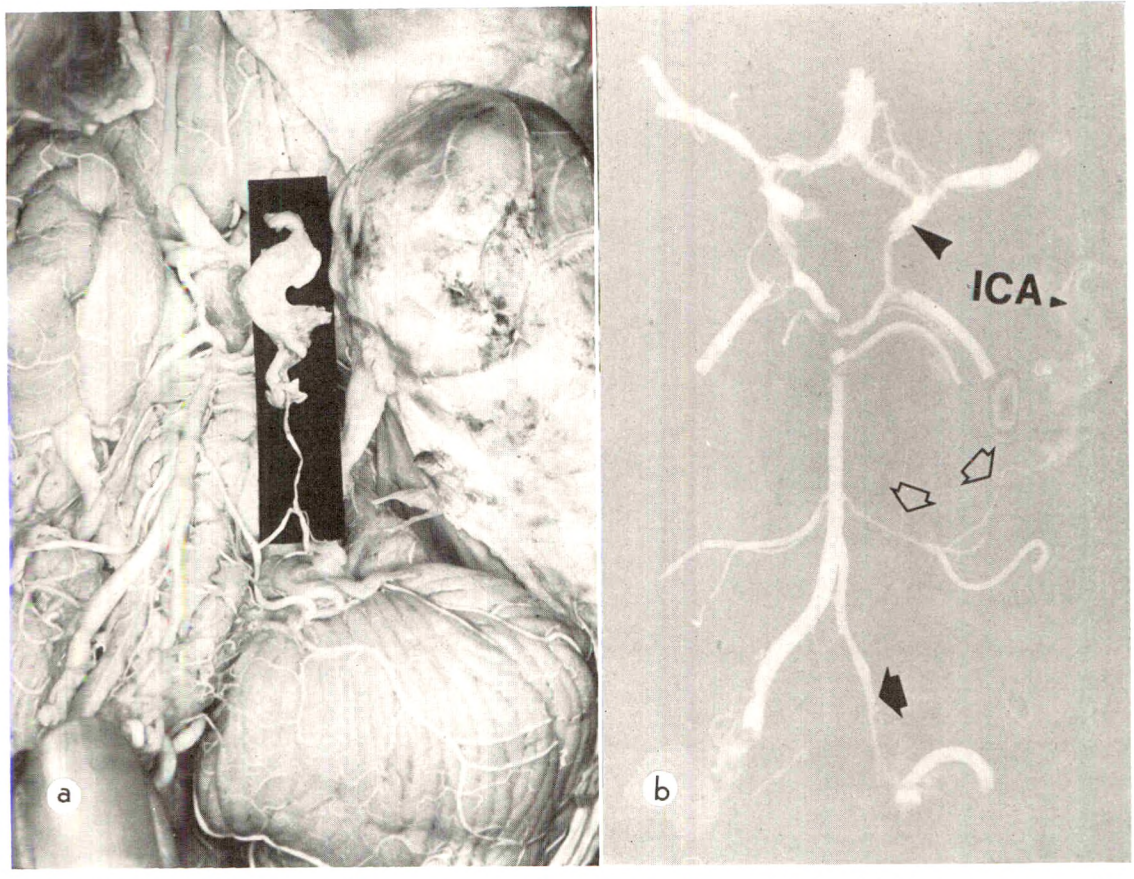



Fig. 9. Persistent primitive auditive artery. (*a*) Photograph of base of brain. (*b*) Roentgenogram of removed and injected circle of Willis. A fine arterial remnant, patent and filled with contrast medium connects the left anterior inferior cerebellar artery (AICIA) and ICA. There is also a basilar artery fenestration and a partial hypoplastic left vertebral artery (VA) with a prominent posterior inferior cerebellar artery (PICIA). Note the double ring density lateral to the left PCA which is an artifact (Newton's ring).

ported in the literature, most of them demonstrated by angiographic means; only a few are proven by surgery or autopsy. Two primitive trigeminal arteries and one primitive auditive artery were recognized by us and illustrated by postmortem arteriography (Fig. 9, *a* and *b*).

It should be emphasized that superimposition of arteries and the coincidence of an arterial pressure difference in the posterior circulation with filling of the distal basilar artery may tempt the diagnosis of a carotid-basilar anastomosis *in vivo*. Occasionally a faultily directed roentgen-ray beam or tilted position of the patient's head, plus partial reflux of contrast medium into the posterior circulation, may mimic the feature of a primitive trigeminal artery. One patient with a ruptured aneurysm of the anterior communicating artery and such described arterial pattern disproved a premortem diagnosis during a careful autoptic investigation and arteriography.

### ARTERIOVENOUS SHUNTS

The postmortem angiography taught us that the interpretation of premortem angiography demands a very critical observation.

It was stated above that, as a rule, venous channels are not filled by our technique of injection. The exception from the normal is the tumor, like glioblastoma multiforme with intrinsic vascular shunts, or a non-space occupying lesion such as an arteriovenous malformation with a direct shunt

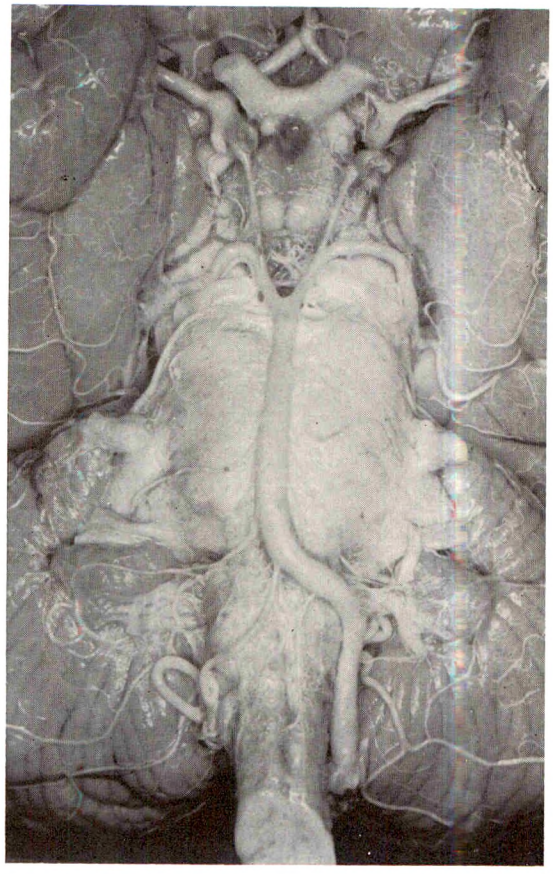


FIG. 10. Photograph of base of brain, mainly for demonstration of the hypoplastic right VA, a situation which can erroneously lead to the impression of a VA or BA occlusion. There is also an infundibular widening of the PCCA, right. An aneurysm of the right ACHA is present in addition.

into veins. Figure 11 *a* illustrates a premortem angiogram of a glioblastoma multiforme with a rapid blood transport into venous channels. The same channels are verified and illustrated at postmortem angiography due to the fact that the entire dura was removed with the brain and contrast material was noted in the superior sagittal sinus (Fig. 11, *b* and *c*).

Another patient, who died of the consequences of cardiac disease, demonstrated on postmortem injection an unexpected, immediate shunting of contrast material into the very unusual dilated venous channels draining an angioma of the uncus of the temporal lobe, supplied by the right hyperplastic anterior choroidal artery (Fig. 5, *a–d*), and drained via a dilated venous plexus of the tentorium. We have observed a similar arteriovenous malformation *in vivo*.

Finally, the postmortem cerebral investigations rewarded us with the demonstration of a meningeal vessel supplying the inferior surface of the mid-tentorium.[33] The interest of this vessel rests in the origin of the artery from the posterior cerebral artery, the so-called $PCA_1$ or pars circularis posterior (Fig. 12, *a* and *b*). The significance of this artery was discussed in connection with infratentorial tumors, which may take their blood supply from this vessel. The diameter ranges from 0.1 to 0.5 mm. in the normal. This is one of the reasons why it is hardly picked up at carotid arteriography. Secondly, its course around the peduncles, parallel with the posterior cerebral artery and partially with the posterior medial choroidal artery, prevents it from isolated demonstration unless it becomes a hyperplastic vessel in the presence of tumors, meningioma, glioblastoma multiforme, and hemangioma. We believed that the origin of this vessel is constant and that its demonstration on postmortem angiography depends merely on the careful removal of the brain specimen with dura and prevention of pull on the tentorium. We now have to add that we found this vessel in 2 instances also originating from the right $PCA_1$, whereas the majority of offsprings are from the left side. Occasionally a small arterial connection to the superior cerebellar artery is found, which leads rostrally to the quadrigeminal plate. In this connection, the artery of Bernasconi and Cassinari should be mentioned, which originates from the internal carotid artery and courses towards the tentorium.[9]

## DISCUSSION

The teaching value of the postmortem injected brain specimen and its angiographic appearance has not been pointed out in the literature. The superiority of this method is very obvious, as demonstrated above.

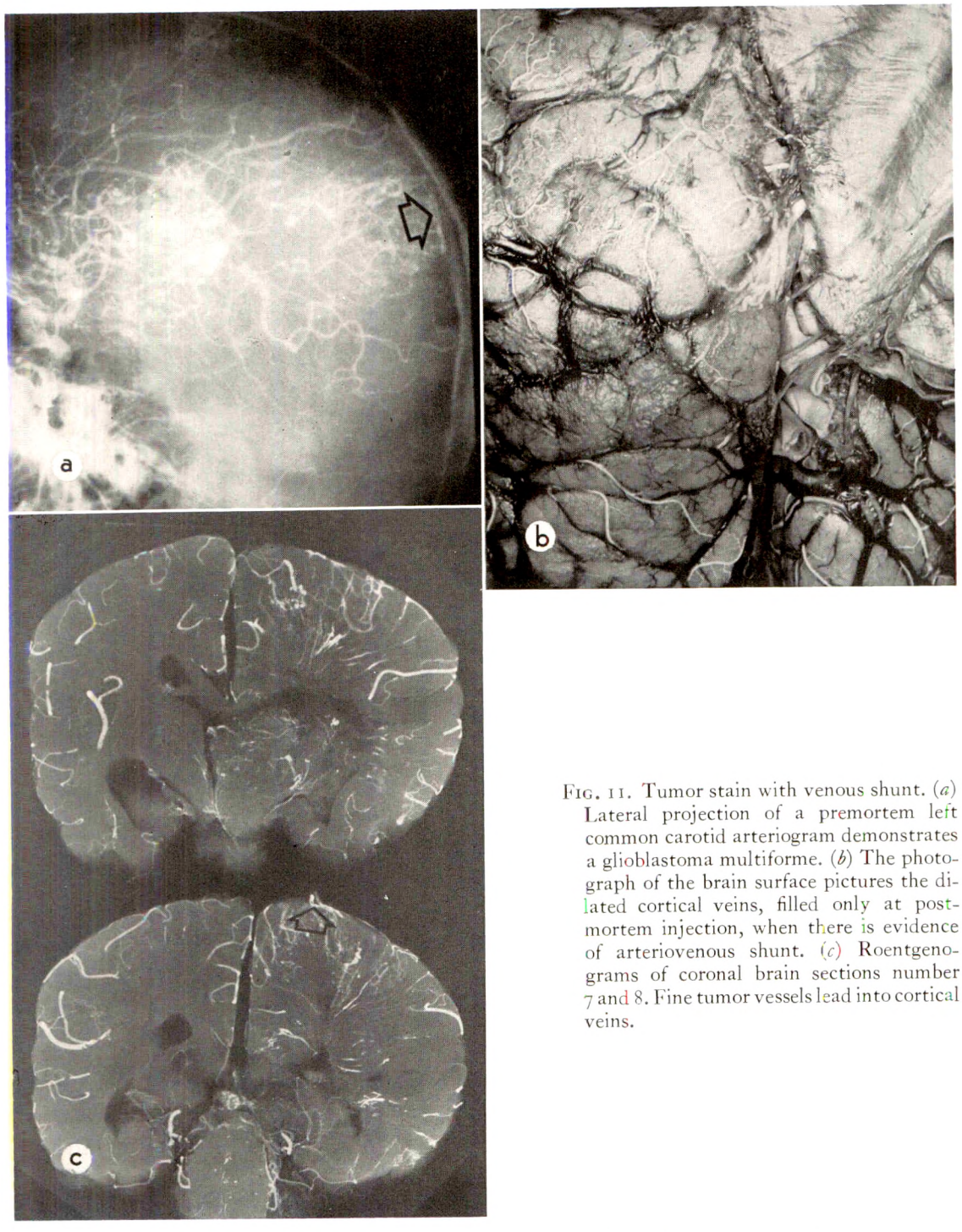



Fig. 11. Tumor stain with venous shunt. (*a*) Lateral projection of a premortem left common carotid arteriogram demonstrates a glioblastoma multiforme. (*b*) The photograph of the brain surface pictures the dilated cortical veins, filled only at postmortem injection, when there is evidence of arteriovenous shunt. (*c*) Roentgenograms of coronal brain sections number 7 and 8. Fine tumor vessels lead into cortical veins.

The removal of the brain with the dura attached to it is technically more difficult, but many entities can only be recognized and evaluated using this method.

No comparative data are reported of larger series of injected brain specimens. Therefore, a parallelism of our data (measurement, frequencies of vessel variations and of aneurysms) with those of uninjected reported brain series appears to us unreal-

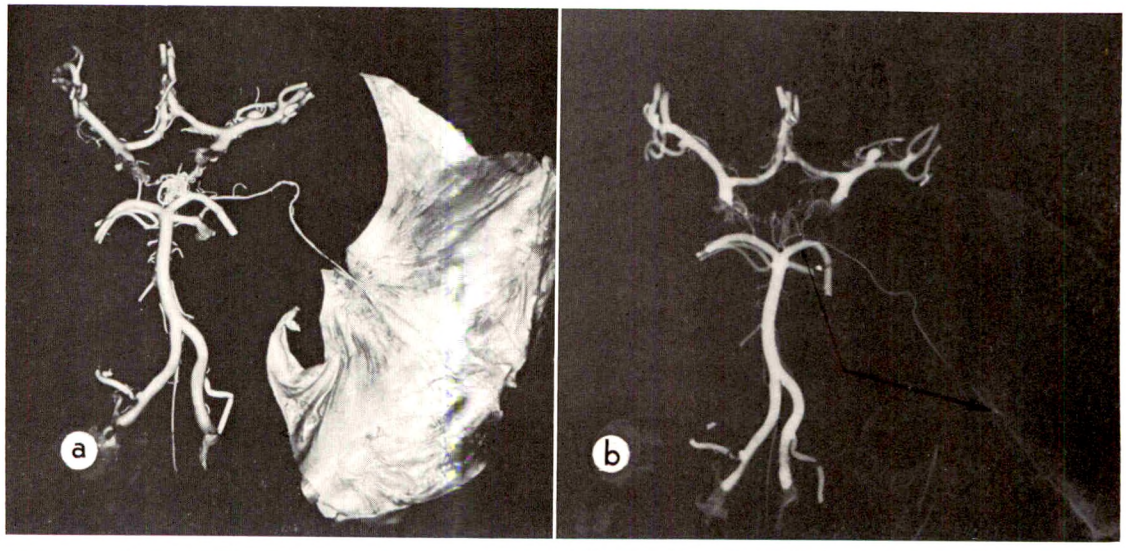



FIG. 12. Infratentorial meningeal artery, the artery of Davidoff and Schechter (ADS). (*a*) Photograph of removed injected circle of Willis with tentorium. A fine artery connects the PCA ($PCA_1$ or pars circularis posterior) and the midline of the inferior surface of the tentorium, where it supplies the dura in an area of 2–3 cm. to either side. (*b*) Roentgenogram of the same circle to point out the entrance of the ADS at the tentorium. Note aneurysm at right and left MCA bifurcation.

istic. Our injection of arteries amplifies minor variations and their pathology, which are then more easily detected at brain cutting sessions.

## SUMMARY

1. The teaching value of postmortem injection of cerebral vessels and angiography as a routine part of every autopsy is pointed out.

2. Measurements of the vessel diameters are recorded.

3. The importance of injection for easy recognition of aneurysms, carotid-basilar anastomoses and arteriovenous shunts is stressed.

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# THE NORMAL MIDDLE CEREBRAL ARTERY AXIS*

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THE significance of displacement of the major middle cerebral artery trunks in the angiographic localization of expanding intracranial lesions has been appreciated since the initial studies of Moniz,[3] who considered the "groupe sylvien" to represent the arterial axis of the brain. Gross elevation of these vessels by both intracerebral and extracerebral masses in the middle fossa is easily recognized. However, a minor upward displacement of this arterial complex may be more difficult to assess, particularly in infants and children.

Methods for measuring the normal and pathologic locations of the middle cerebral artery trunks from the lateral carotid arteriogram have been described by Taveras and Poser,[5] and Woringer and Gernez.[6]

The purpose of this paper is to present a simple method for determining the normal middle cerebral artery axis as visualized on the lateral projection of the carotid arteriogram. The accuracy of this method is to be evaluated in both the normal child and adult, and also in a series of verified mass lesions in the middle fossa.

## MATERIALS AND METHODS

From the cerebral angiographic material in the teaching file of the Department of Radiology at Duke University Medical Center, 500 normal lateral carotid arteriograms in adult patients were selected as representing technically satisfactory examinations. Particular attention was paid to rotation of the skull. Any case with rotation of the mid-sagittal plane amounting to more than several millimeters, defined by measuring the degree of incomplete superimposition of both the orbital plates and the petrous pyramids (Fig. 1, *A* and *B*) was discarded, since this limitation will obviously alter the position of the middle cerebral artery trunks. A second group of 75 normal carotid arteriograms was similarly selected from the pediatric material for

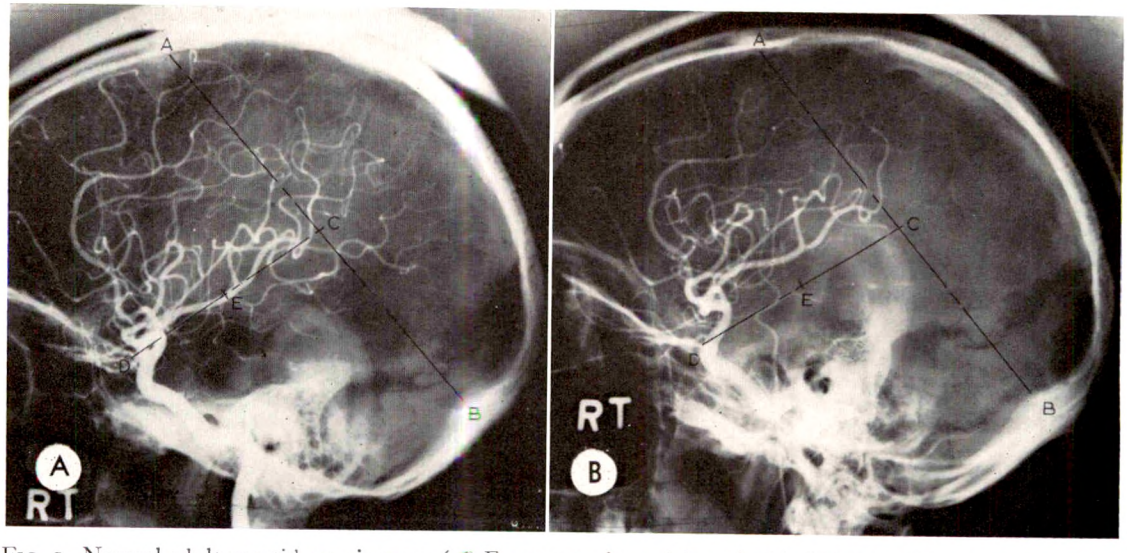
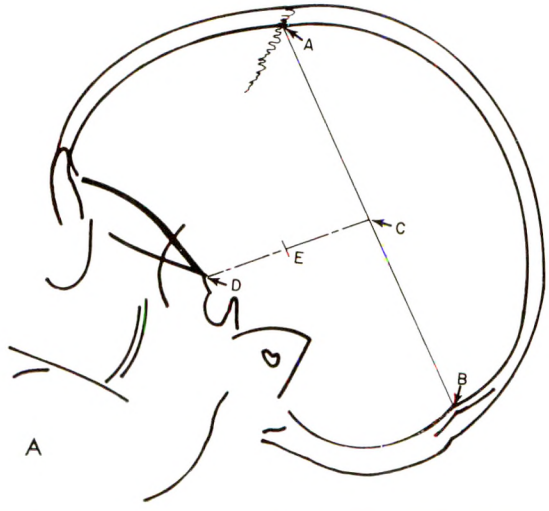



FIG. 1. Normal adult carotid arteriogram. (*A*) Exact superimposition of orbital plates and petrous pyramids. (*B*) Pseudo-elevation of middle cerebral artery axis from rotation of skull. Same case as in *A*.

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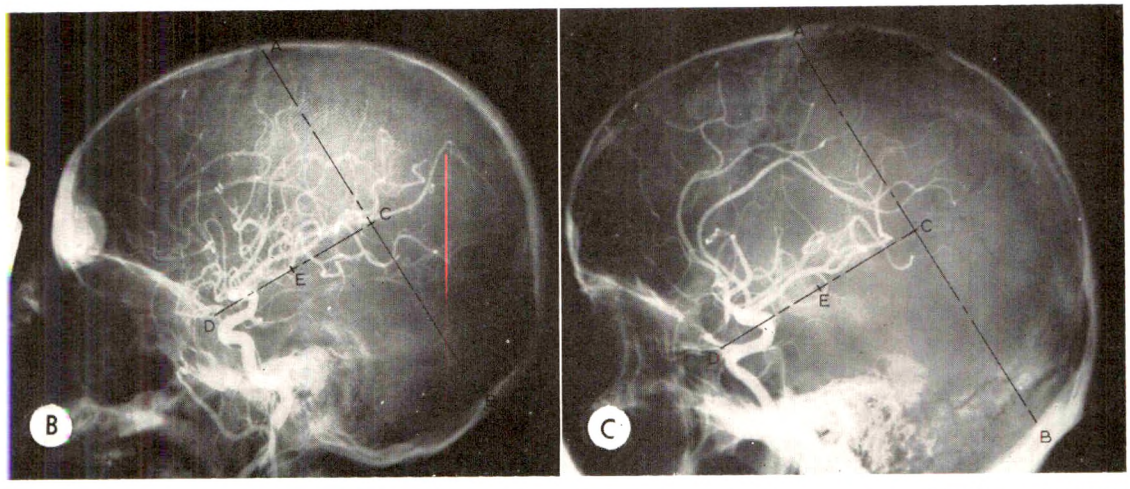




Fig. 2. (*A*) Diagram of the method of determining the middle cerebral artery axis. (*B*) Normal middle cerebral artery axis in a 4 year old child. (*C*) Normal middle cerebral artery axis in an adult.

study, the patients ranging in age from 8 months to 15 years. A third group was selected, consisting of 100 cases of verified expanding lesions in the middle fossa. As in the normal cases, only arteriograms with very little or no rotation were included. All of the patients in the pathologic group were adults.

Two points on the inner table of the skull were utilized, namely, the inner table margin of the bregma (A) and the internal occipital protuberance (B) (Fig. 2, *A*, *B* and *C*). A line was drawn to connect these two points of reference and the mid-point (C) was considered to be the posterior limit of the middle cerebral artery axis. The limbus sphenoidalis (D) was selected as an additional landmark. This structure is reasonably well seen on the lateral carotid arteriogram, and as Holman[1] points out, the pituitary fossa is in a relatively lower position with respect to the level of the planum sphenoidale in the newborn than it is in older people (Fig. 3, *A* and *B*). Therefore, bone landmarks more closely related to the sella turcica proper, such as the tuberculum sellae and the anterior clinoid processes, will tend to give a lower estimate of the middle cerebral artery axis than if the limbus sphenoidalis is utilized, particularly in infants and children. Occasionally, the exact point of the limbus was difficult to

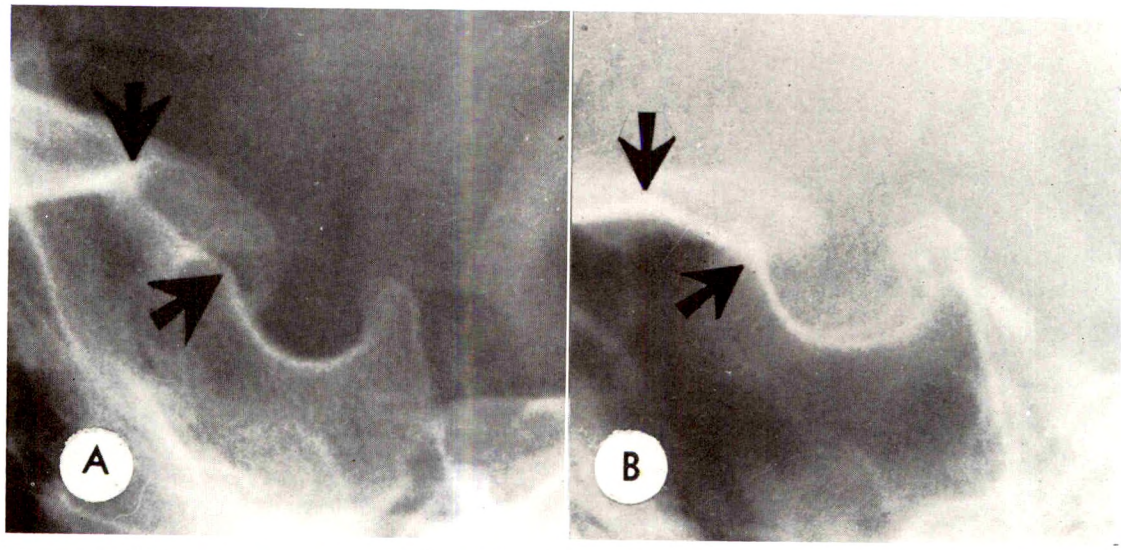



FIG. 3. (*A*) Sella turcica in a normal infant. (*B*) Sella turcica in a normal adult. The upper arrow points to the limbus sphenoidalis and the lower arrow is at the tuberculum sellae.

define. In such a case, the most posterior point visible along the planum sphenoidale was considered to be point D. The estimated middle cerebral artery axis, considered here as merely the trend of direction and location of the major arterial trunks of the middle cerebral artery, was then drawn directly on each roentgenogram. The main trunks of the middle cerebral artery have been arbitrarily defined by Ring[4] as those major vessels passing without interruption through the middle third of the sylvian fissure. A line (CD) was then constructed (Fig. 2, *A*, *B* and *C*) and the distance in millimeters between the line CD and the estimated axis, measured at the mid-point E, was recorded in each case. When the middle cerebral artery trunks were found to be clearly bowed upward by an expanding lesion, it was obviously impossible to define a rectilinear axis. In such cases, the measurement to the major middle cerebral artery trunks opposite point E was made without a middle cerebral artery axis estimation.

## RESULTS

The measurements in the normal adult material are listed in Table I. The figures for normal infants and children are shown in Table II and the data for the group of verified expanding lesions in the middle fossa are listed in Table III. Table IV compares the average mean and the range between these 3 groups.

The range of elevation of the middle

TABLE I

THE MIDDLE CEREBRAL ARTERY AXIS IN 500 NORMAL ADULT CAROTID ARTERIOGRAMS

| mm. Above Line CD | No. of Cases | Per Cent |
|---|---|---|
| 12 | 1 | 0.2 |
| 11 | 0 | 0 |
| 10 | 0 | 0 |
| 9 | 0 | 0 |
| 8 | 1 | 0.2 |
| 7 | 2 | 0.4 |
| 6 | 8 | 1.6 |
| 5 | 31 | 6.2 |
| 4 | 42 | 8.4 |
| 3 | 67 | 13.4 |
| 2 | 65 | 13.0 |
| 1 | 24 | 4.8 |
| Line CD - - - - 0 | 158 | 31.6 |
| 1 | 19 | 3.8 |
| 2 | 45 | 9.0 |
| 3 | 21 | 4.2 |
| 4 | 13 | 2.6 |
| 5 | 2 | 0.4 |
| 6 | 1 | 0.2 |
| mm. Below Line CD | | |

TABLE II

THE MIDDLE CEREBRAL ARTERY AXIS IN 75 NORMAL CAROTID ARTERIOGRAMS IN CHILDREN

| mm. Above Line CD | No. of Cases | Per Cent |
|---|---|---|
| 10 | 3 | 4.0 |
| 9 | 6 | 8.0 |
| 8 | 8 | 10.6 |
| 7 | 6 | 8.0 |
| 6 | 11 | 14.6 |
| 5 | 12 | 16.0 |
| 4 | 2 | 2.7 |
| 3 | 10 | 13.4 |
| 2 | 5 | 6.6 |
| 1 | 1 | 1.4 |
| 0 | 11 | 14.7 |

TABLE III

THE MIDDLE CEREBRAL ARTERY AXIS IN 100 EXPANDING LESIONS IN THE MIDDLE FOSSA

| mm. Above Line CD | No. of Cases | Per Cent |
|---|---|---|
| 41 mm.–15 mm. | 76 | 76 |
| 14 | 6 | 6 |
| 13 | 8 | 8 |
| 12 | 2 | 2 |
| 11 | 1 | 1 |
| 10 | 2 | 2 |
| 9 | 2 | 2 |
| 8 | 1 | 1 |
| 7 | 1 | 1 |
| 6 | 1 | 1 |

cerebral artery axis in the 100 pathologic cases was 6 to 41 mm. above line CD, with a single case having as little as 6 mm. elevation and 95 cases measuring 10 mm. or more above this line. On the contrary, only a single case in the normal adult series was found to have a middle cerebral artery axis of more than 8 mm. above line CD. Since there is no surgical or autopsy evidence for the presence of a disease process in the middle fossa of this patient, her clinical findings are briefly reviewed.

J.B.M., DUMC No. F-5245. A 22 year old white female was involved in an automobile accident 12 days before admission to this hospital. She was thrown from the car and rendered unconscious. Upon admission to her local hospital, she was found to have lacerations of the right scalp, an alternating state of consciousness and right occipital and temporal linear skull fractures. She became less responsive over the following week and examination upon admission to this hospital revealed a moderately obtunded state of consciousness, hyperactive reflexes and a left extensor plantar response. A right carotid arteriogram was performed 2 days later (Fig. 4) and was interpreted as normal. The patient subsequently improved, her neurologic status reverted to normal and she was discharged with the diagnosis of cerebral contusion.

*Comment.* The measurements made in this case are shown in Figure 4. There is an elevation of the middle cerebral artery axis of 12 mm. above line CD. It is assumed that this finding represents some swelling of the right temporal lobe, probably from contusion, although it would be impossible to exclude hemorrhage.

In the 100 verified pathologic cases, minor elevations of the middle cerebral artery axis (10 mm. or less), were found to be due to hemorrhage in 3 cases. The other small elevations were seen in 2 cases of temporal lobe astrocytoma, one case of

TABLE IV

THE RELATIONSHIP BETWEEN LINE CD AND THE MIDDLE CEREBRAL ARTERY AXIS

| | Average Distance Above Line CD (mm.) | No. of Cases | Range Above and Below Line CD (mm.) |
|---|---|---|---|
| Children | 4.86 | 75 | 0–10 above |
| Adults | 1.02 | 500 | 6 below—8 above* |
| Pathologic | 19.6 | 100 | 6–41 above |

* Excluding one case 12 mm. above (see Fig. 4).

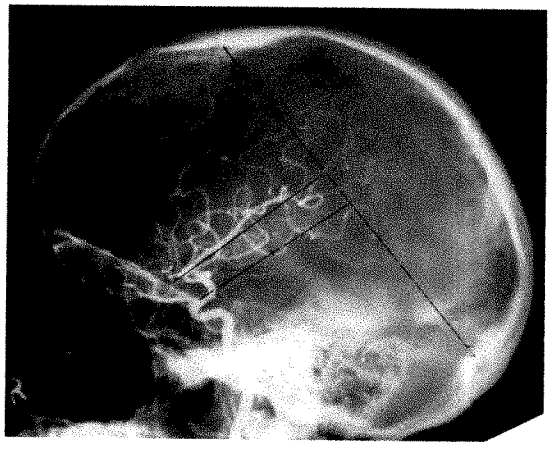


FIG. 4. Elevation of the middle cerebral artery axis in a case of probable contusion of the temporal lobe.

glioblastoma multiforme of the temporal lobe, and a single instance of (pterional) meningioma. Elevation of the middle cerebral artery axis was found to be as little as 6 mm. in a single case of intracerebral hematoma and 7 mm. in one instance of glioblastoma multiforme.

The 75 pediatric measurements produced a maximum normal distance of the middle cerebral artery axis above line CD of 10 mm. in 3 cases, with no particular differences noted in the various age groups.

## DISCUSSION

One of the fundamental considerations in attempting to establish a linear measurement for the normal middle cerebral artery axis is that most measurements in diagnostic roentgenology are sharply limited in value and the "absolute" demarcation between normal and pathologic is difficult to define. Any measurement must be utilized in context with the total roentgenographic picture and an individual measurement that just exceeds "the upper limits of normal" in the face of an otherwise negative examination must be viewed with strong reservations.

A widely utilized measurement of the middle cerebral artery axis is "the clino-parietal line" described by Taveras and Poser.[5] This measurement requires the anterior clinoid process and the lambda (or internal occipital protuberance) for reference points. These authors have stated that "occasionally the line is just above the lowest major branch in the adult. In children, the major middle cerebral branches are always above the clino-parietal line." It is noted that in 11 of the 75 children listed in Table II, the middle cerebral artery axis coincided exactly with line CD. Taveras and Poser[5] also mentioned that "it may be noted that in younger children the distance from the clino-parietal line to the lowest major branch of the middle cerebral artery . . . is greater than in older children." We have found no such change in the middle cerebral artery axis with advancing age (Fig. 5) other than the gross comparison between the pediatric and adult series.

Woringer and Gernez[6] attempted to establish a normal middle cerebral artery axis by measuring the angle between the middle cerebral artery trunks on the lateral arteriogram and a line drawn from the internal occipital protuberance to the center of the sella turcica. Since it is often the case that an expanding process elevates the middle cerebral artery trunks in a fashion parallel to the original course of these vessels, this measurement has limited value.

The general appearance of the middle cerebral artery trunks, the status of the anterior choroidal, posterior communicating

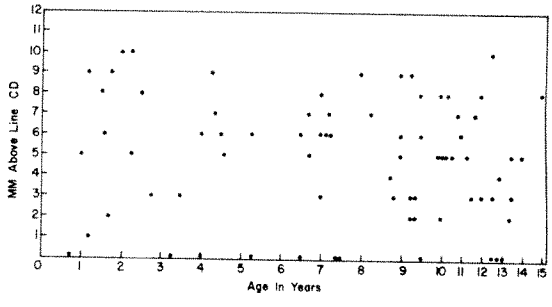



FIG. 5. Relation of the middle cerebral artery axis to line CD in 75 normal infants and children.

and posterior cerebral arteries and the basilar vein, midline dislocations, the appearance of the middle cerebral artery in the frontal plane of arteriography, and the presence and type of tumor neovascularity are all important in the angiographic diagnosis of middle fossa expanding lesions. In addition, there is a certain degree of observer variation in estimating the middle cerebral artery axis from the lateral roentgenogram. Nevertheless, it is believed that the combination of this simple linear measurement, together with the insistence upon an accurately positioned patient during the examination, will improve the evaluation of the middle cerebral artery axis in the problem case.

SUMMARY

A simple method for the evaluation of the normal and pathologic middle cerebral artery axis as determined from the lateral carotid arteriogram is presented. These measurements have been applied to both adults and children and have been found to be of assistance in cerebral angiographic interpretation.

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# RACIAL DIFFERENCES IN CEREBROVASCULAR DISEASE*

## ANGIOGRAPHIC EVALUATION OF JAPANESE AND AMERICAN POPULATIONS

*By* STEPHEN A. KIEFFER, M.D.,† YO TAKEYA, M.D., JOSEPH A. RESCH, M.D., *and* KURT AMPLATZ, M.D.

AS PART of a comprehensive program of study of cerebrovascular disease at the University of Minnesota, 4 vessel angiography has been performed on over 100 carefully selected patients in the past 5 years. In an effort to ascertain possible racial differences in the stroke syndrome, a similar program has been carried out at Kyushu University in Japan. Selection of patients, method of angiography,[1] and injection technique have been standardized and were for all practical purposes identical in both universities. Evaluation of the angiographic data reveals significant differences between the Japanese and American populations.

### MATERIAL AND METHODS

At both Universities, patients with a diagnosis of stroke syndrome which had occured less than 1 month prior to hospitalization were selected for 4 vessel angiography. The symptoms of the acute episode generally included hemiparesis, hemihypesthesia, or brain stem dysfunction.[2] Those studied survived the acute phase of the stroke and their clinical status was either stable or improving. Patients with a likely alternative clinical diagnosis to cerebral thromboembolism were excluded from the study. In spite of these precautions, in the Minnesota group, 4 such cases were found: 2 brain tumors, 1 intracerebral hematoma, and 1 subdural hematoma.

An effort was made in each patient to obtain a complete angiographic evaluation of the extracranial and intracranial circulation without unnecessary arterial punctures. This can only be attained by selective catheterization of each of the 4 major vessels supplying the head and neck (Fig. 1). Specially molded catheters were introduced percutaneously into either the axillary or subclavian arteries and manipulated into the brachiocephalic vessels. More recently, the femoral artery has been the portal of entry for the catheter. Ideally, visualization of all 4 arteries in 2 planes including both the extracranial and intracranial distribution of each vessel and its branches was desired. Often, due to limitations in time and in quantity of contrast medium, this goal could not be attained in one sitting.

Included in this report are 66 patients with established stroke at Kyushu University and 92 patients at the University of Minnesota.

A strictly anatomic angiographic grading system was developed. The diameter of the arterial lumen in the area of the atherosclerotic plaque was measured on the roentgenogram and expressed as a percentage relative to the diameter just proximal to the area of narrowing. The length of the area of narrowing was also noted, although for the purposes of this study comparisons were based only on the percentage reduction of lumen diameter. Since the comparison was made on a ratio, absolute numerical differences between the two institutions based on variations in technique were eliminated.

Because of the fact that an atheromatous

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† Scholar in Radiological Research of the James Picker Foundation.

plaque may not be readily appreciated if seen *en face* in only one projection, roentgenograms in 2 views of each vessel were made. It was agreed to judge the degree of narrowing in the projection where it was best demonstrated (Fig. 2).

Atherosclerotic stenoses were divided into 4 major groups. Group I, labeled as "normal," included lesions causing less

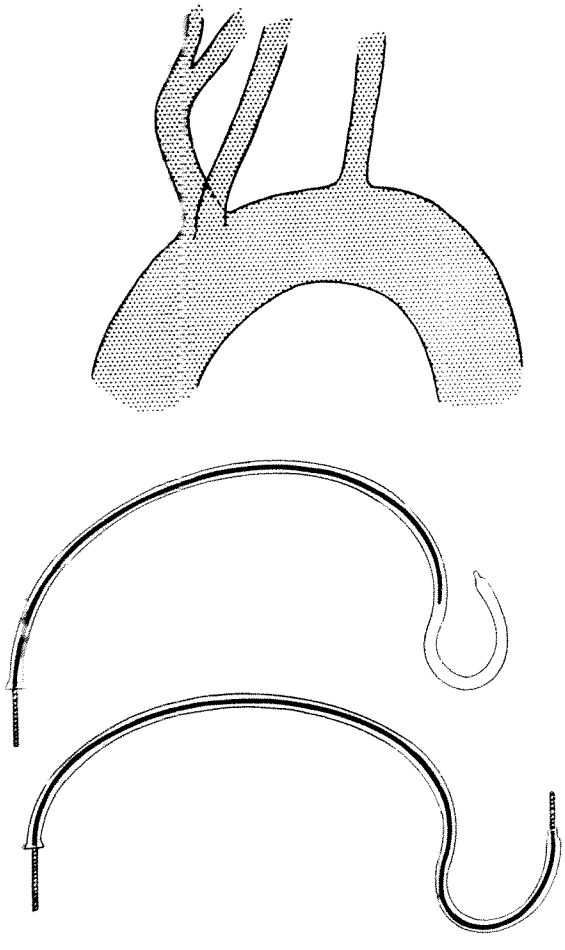


Fig. 1. *Upper.* Changes due to arteriosclerosis. The distance between the left carotid and left subclavian arteries is increased, but the left carotid remains in close anatomic relationship with the innominate artery. *Middle.* Catheter with guide wire. Specially molded catheter can adjust for changes described above. Here, guide wire is withdrawn partially, restoring sharp curvature of catheter tip. *Lower.* By extension of the guide wire, the catheter "loop" can be opened up. This maneuver is usually helpful for catheterization of the left subclavian artery.

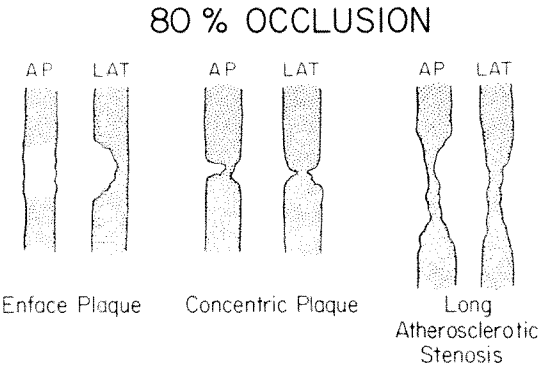



Fig. 2. Common appearances of atherosclerotic occlusions. Diagram at left shows importance of demonstrating lesions in 2 planes.

than 25 per cent constriction of the lumen diameter since such lesions were regarded as being of no hemodynamic significance. Group II included those lesions causing between 26 and 50 per cent narrowing, Group III 51 to 75 per cent narrowing, and Group IV 76 to 100 per cent occlusion.

## RESULTS

Complete angiographic evaluation of the arteries of the head and neck was obtained in 44 of the 66 patients with established stroke at Kyushu University. The remainder had incomplete angiography, usually amounting to a study of the vessels on the side of the expected lesion. Thirty-one of the 66 patients were classified as "normal," as defined above (Table I).

In the Minnesota group, 67 of 92 patients had complete angiography and the remaining 25 patients had incomplete studies. Of the total of 92 patients, 50 were classified as

Table I

PATIENTS STUDIED BY CEREBRAL ANGIOGRAPHY

| | Kyushu | Minnesota |
|---|---|---|
| Total number of patients studied | 66 | 92 |
| Complete (4 vessel) angiographic evaluation | 44 (67%) | 67 (73%) |
| Normal studies (less than 25 per cent narrowing) | 31 (47%) | 50 (54%) |
| Abnormal studies | 35 (53%) | 42 (40%) |

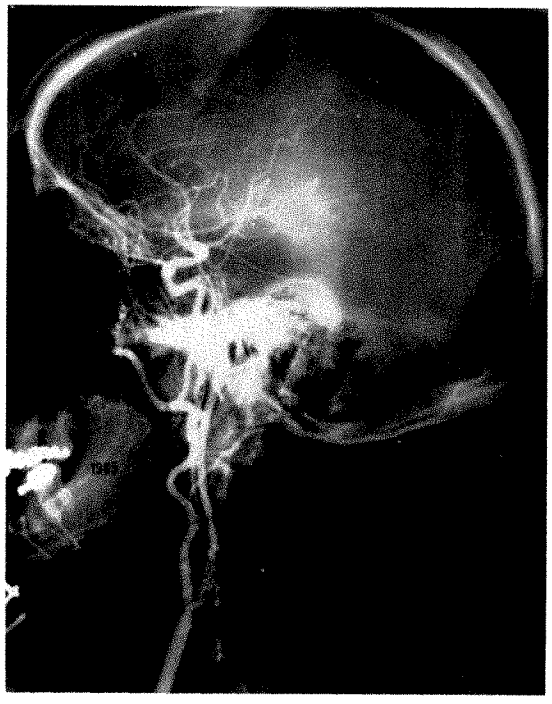


FIG. 3. Lateral roentgenogram in early arterial phase of typical Minnesota patient. There is a significant narrowing of the origin of the internal carotid artery. The proximal first centimeter of this vessel shows more than 50 per cent reduction in lumen diameter. The lesion extends distally an additional 2 centimeters but this segment is less severely constricted. Also involved to a lesser extent are the distal common carotid and proximal external carotid arteries. Except for minor irregularity in the region of the carotid siphon, the intracranial arteries appear normal.

"normal." There was no statistically significant difference between the two racial groups in the number of "normal" angiograms.

When the lesions were classified by location as either extracranial or intracranial, a significant difference was encountered. In the Minnesota group, the 42 patients with lesions involving more than 25 per cent of the lumen diameter of the vessel showed 85 such lesions. Seventy-six of the lesions were extracranial (Fig. 3) and only 9 were intracranial (Table II). In the Kyushu group, 35 patients had 73 lesions occluding more than 25 per cent of the lumen diameter. However, only 30 of these 73 lesions (43 per cent) were extracranial. Expressed from a slightly different aspect, intracranial lesions of possible or likely anatomic significance represented 58 per cent of the total intra- and extracranial lesions in the Japanese group but only 11 per cent of the American group (Fig. 4, *A* and *B*).

If we consider only lesions producing more than 50 per cent narrowing of the diameter of the arterial lumen, only 5 patients in the Minnesota group exhibited lesions intracranially which satisfied this criterion. However, 16 patients in the Kyushu group had intracranial disease falling into this category (Table III). To further confirm this difference, arteries with complete occlusion were tabulated. Only one complete occlusion was found extracranially in the Japanese stroke patients. However, 14 such cases were found in the Minnesota study, chiefly at or near the carotid bifurcation (Table IV).

## INVESTIGATOR RELIABILITY

The results described above are based on interpretation of the angiograms by the Minnesota investigators. A possible factor of variability would be the difference in interpretation between the Japanese and American physicians. Accordingly, a reliability test was designed. Twenty-eight Minnesota angiograms and thirty Kyushu

TABLE II

TOTAL NUMBER OF LESIONS (>25%)

| | Kyushu | Minnesota |
|---|---|---|
| *Extracranial* | | |
| Subclavian | 2 | 12 |
| Vertebral | 12 | 18 |
| Common carotid | 0 | 4 |
| Carotid bifurcation | 16 | 42 |
| | 30 | 76 |
| *Incracranial* | | |
| Anterior, posterior, middle, siphon | 28 | 5 |
| Vertebral intracranial | 12 | 3 |
| Basilar | 3 | 1 |
| | 43 | 9 |

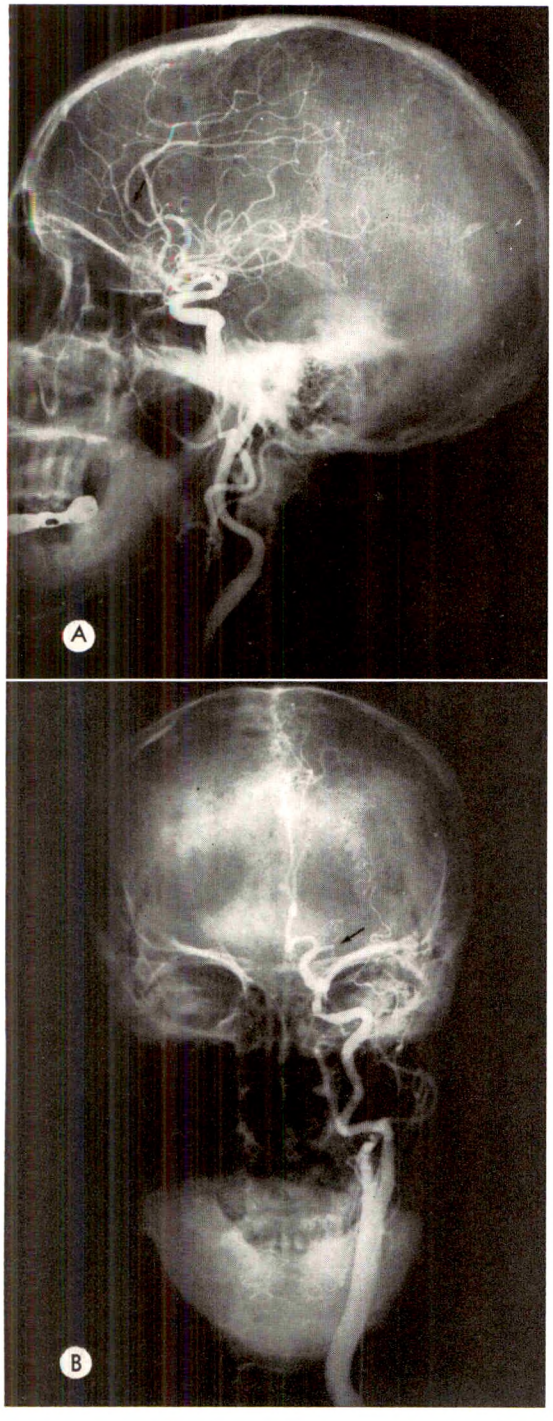



Fig. 4. Representative lesions from the Japanese series. (*A*) Lateral roentgenogram in arterial phase demonstrates diffuse intracranial small vessel involvement. Arrow points to narrowing of anterior cerebral artery. Distal continuation of this vessel is also diffusely involved as are several middle cerebral branches. (*B*) Anteroposterior view in another patient shows complete occlusion of proximal middle cerebral artery (arrow). The carotid arteries in the neck are tortuous but demonstrate no apparent occlusive lesions in either plane.

Table III

50% occlusion

| | Kyushu | Minnesota |
|---|---|---|
| Extracranial, unilateral | 4 | 16 |
| Bilateral or more than 1 vessel | 1 | 13 |
| | 5 | 29 |
| Extracranial and intracranial | 9 | 1 |
| Intracranial only | 7 | 4 |
| | 16 | 5 |
| Intracranial disease (per cent)* | 25 | 5 |
| Extracranial disease (per cent) | 21 | 35 |

* Percentages refer to the total number of patients studied at the respective universities.

angiograms were selected at random and read independently by one Minnesota investigator (K.A.) and one Japanese investigator (Y.T.). The readings were grouped into intervals of 25 per cent as outlined above. The results of the comparisons are shown in Table v. In the set of angiograms from Minnesota, the investigators agree in classification of 23 of the 28 studies. In the other 5 lesions, the disagreement is by 1

Table IV

100% occlusion

| | Kyushu | Minnesota |
|---|---|---|
| Extracranial | 0 | 14 |
| Extracranial+1 intracranial | 1 | 0 |
| Intracranial | | |
| 1 vessel | 6 | 4 |
| 2 or more vessels | 2 | 0 |
| | 9 | 18 |
| Intracranial disease (per cent)* | 15 | 4 |
| Extracranial disease (per cent) | 1 | 14 |

* Percentages refer to the total number of patients studied at the respective Universities.

TABLE V

INVESTIGATOR RELIABILITY

A. *Minnesota Angiograms*

| K.A. / Y.T. | <25% | 26–50% | 51–75% | 76–100% |
|---|---|---|---|---|
| < 25 | 9 | 3 | | |
| 26–50 | 1 | 5 | | |
| 51–75 | | | 2 | 1 |
| 76–100 | | | | 7 |

B. *Kyushu Angiograms*

| K.A. / Y.T. | <25% | 26–50% | 51–75% | 76–100% |
|---|---|---|---|---|
| < 25 | 5 | 1 | | |
| 26–50 | 3 | 10 | 2 | |
| 51–75 | | | 2 | |
| 76–100 | | | | 7 |

category only. The Kyushu angiographic set shows agreement in 24 cases and disagreement in 1 category in 6.

It would seem, therefore, that investigator reliability and consistency is good, and that this would not be a factor in the evaluation of differences between the two populations.

## COMMENT

Cerebrovascular disease is the leading cause of death in Japan. Epidemiologic studies reveal that the incidence of stroke in Japan is among the highest of the record-keeping nations of the world.[3] It has generally been believed that hemorrhagic stroke is the major contributor to this problem.

However, recent investigations suggest that the majority of cases of stroke in Japan are due to thrombosis rather than hemorrhage.[4] Phair *et al.*[5] studied the incidence of cerebrovascular disease in Hiroshima and Nagasaki and found that cerebral infarction was a more common finding than hemorrhage at autopsy.

One of the authors (J. A. R.) has been engaged in a comparative autopsy study of atherosclerosis in the arterial circle of Willis.[6] A total of 5,035 specimens from Minnesota and 1,035 from Japan were reviewed. The prevalence of cerebral atherosclerosis appears at least as great in Japan as in the Minnesota population. Actually, in any given decade, the frequency of atherosclerotic involvement of the circle of Willis is somewhat higher among the Japanese.

In spite of the fact that we are dealing with a comparatively small series, our results appear to indicate that atherosclerotic occlusive vascular disease in the Caucasian population is more commonly demonstrated in the extracranial vessels than in the Japanese population. In agreement with generally accepted statistics for the United States, occlusive disease in the Minnesota group was most frequent at the carotid bifurcation. In Japan, on the other hand, atherosclerotic plaques in this region are relatively uncommon, and changes were more frequent in the small intracranial vessels.

It must be emphasized that many other factors not evident on angiography play a part in the production of the clinical picture of stroke. Therefore, we can draw no definite conclusions regarding the role that these roentgenologically demonstrated lesions played in the production of the stroke syndrome.

## SUMMARY AND CONCLUSIONS

A comprehensive long term study of cerebrovascular disease is being carried out at the University of Minnesota and at Kyushu University in Japan. Four vessel angiography utilizing selective catheterization for maximal detail with roentgenograms in 2 planes has been employed in these patients. This report points out one significant difference between the Japanese and American populations of stroke patients. The incidence of extracranial lesions demonstrable on angiography in the American patients is significantly higher than in

the Japanese group. Conversely, intracranial lesions are much more frequently demonstrated in the Japanese group.

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# EPIDURAL HEMATOMAS*

## VARIED ANGIOGRAPHIC SIGNS

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EPIDURAL hematoma is a well known neurosurgical emergency. Until the advent of cerebral angiography, clinical evaluation was the only reliable diagnostic approach. Plain skull roentgenograms frequently illustrating a fracture over a meningeal vessel and a displaced pineal body were ancillary roentgen signs of intracranial bleeding or edema. Roentgenologic delineation of the type, as well as the location of the intracranial process, could not be made.

More recently, associated adjacent, remote, or contralateral cranial injuries, as well as verification and location of an epidural hemorrhage, have prompted the study of most suspects with emergency cerebral angiography. In Hancock's[4] series of 34 epidural hematomas, 6 would not have been revealed by conventionally placed burr holes. Indeed, he stated that epidural hematomas as close as 1 centimeter to the burr hole may not be apparent. Clinical judgement must be exercised, however, as to the effects of some delay in surgery during the cerebral angiography.

In a large city hospital head trauma is common, and even its more unusual complications are not rare. During the past several years, we have attempted to study the majority of patients with severe head trauma before surgical exploration. Twenty-five patients demonstrating 27 epidural hematomas (2 patients had bilateral epidural hemorrhages) on angiography provide the material for this report. Of a total of 27 epidural hematomas, 11 were located over the parietotemporal convexity, 8 beneath the temporal lobe, 4 in the anterior cranial fossa, and 4 in the occipital epidural space. Cases of subtentorial epidural hematomas are excluded from this report.

TABLE I

ANGIOGRAPHIC SIGNS OF EPIDURAL HEMATOMA
(25 cases, 27 epidural hematomas)

| | No. |
|---|---|
| 1. Lenticular shaped avascular zone | 15 |
| 2. Medial displacement of the middle meningeal artery | 8 |
| 3. Amputation of the middle meningeal artery | 2 |
| 4. Middle meningeal artery extravasation into | |
| a. Fracture line and/or subgaleal fascia | 4 |
| b. Arterial venous sheath | 1 |
| c. Meningeal vein(s) | 0 |
| d. Epidural space | 1 |
| 5. Pseudoaneurysm middle meningeal artery | 0 |
| 6. Displacement of sagittal (or other) sinuses | 1 |
| 7. Extravasation from sinus | 0 |
| 8. Nonspecific avascular mass | 13 |

### ANGIOGRAPHIC SIGNS OF SUPRATENTORIAL EPIDURAL HEMATOMAS

Table 1 shows the various angiographic signs which may be seen with epidural hematomas and their frequency in our series. Some cases exhibited more than one sign. These will be commented upon individually.

1. *Lenticular Shaped Avascular Zone.* Norman[8] differentiated the avascular space of an extracerebral hematoma, originally described by Lohr,[7] into two distinct shapes; the semilunar collection was said to be characteristic of an acute subdural hematoma while the lenticular avascular zone was described in a chronic subdural hema-

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toma. In agreement with others,[14] however, we have not uncommonly found the semilunar zone to be a chronic subdural hematoma rather than an acute one. A lenticular zone of avascularity, on the other hand, is almost always a chronic subdural or an epidural hematoma (Fig. 1). Oblique views may be necessary to delineate the collection, the true shape of which is best visualized in the capillary-venous phase of the angiogram (Fig. 2). Although we know of no definite way to differentiate the shape of an epidural hematoma from that of a chronic subdural hematoma, epidural hematomas may not have a sharp inner margin and frequently have several vessels overlying the bare zone (Fig. 3).

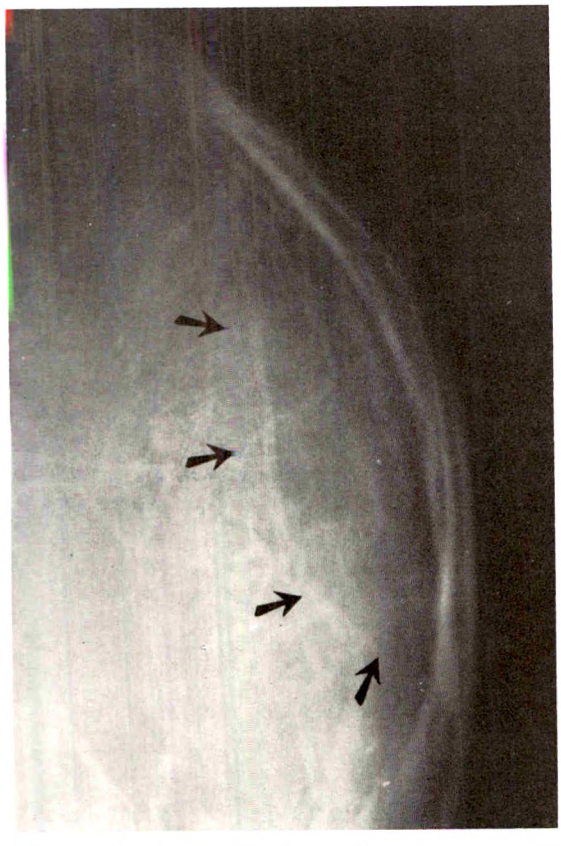


Fig. 1. Anteroposterior view, left common carotid angiogram. The lenticular shaped avascular zone, best appreciated in the venous phase of the angiogram, represents the classical appearance of a convexity epidural hematoma. Arrows point to medial margin of the collection.

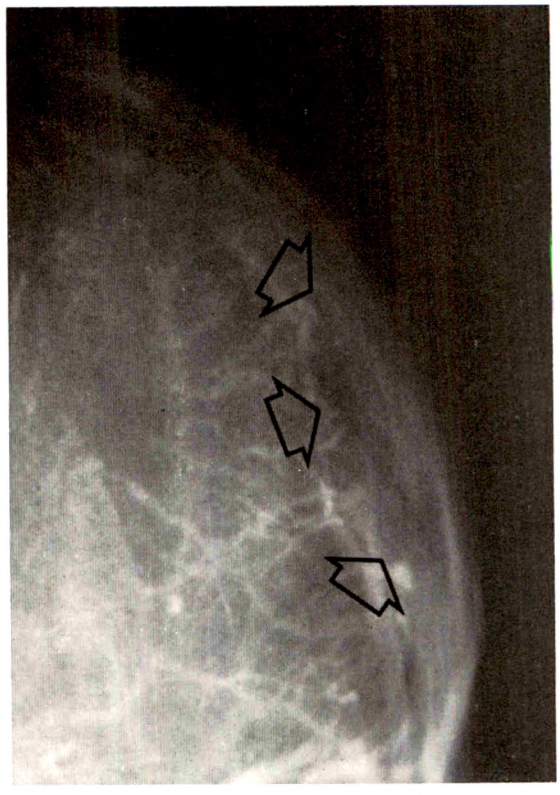


Fig. 2. Oblique view, left common carotid angiogram. Anteroposterior view of this case did not show any extracerebral mass. Posterior oblique view illustrates a lenticular collection over the occipital lobe (arrows) representing an epidural hematoma.

2. *Displacement of the Middle Meningeal Artery*. The epidural hematomas of the middle cranial fossa tend to displace the middle meningeal artery inwards. This vessel is not always easy to visualize on common carotid angiography, but can be clearly seen with selective external carotid studies. In subtemporal epidural collections, the middle meningeal artery may be displaced upwards from the floor of the middle cranial fossa. It is important to be certain that it is the low convexity segment of the middle meningeal artery which is displaced inwards (Fig. 4). High up on the convexity (*i.e.* posterior), a branch of the middle meningeal artery normally courses medially.

3. *Amputation of the Middle Meningeal Artery*. Interruption of the lacerated mid-

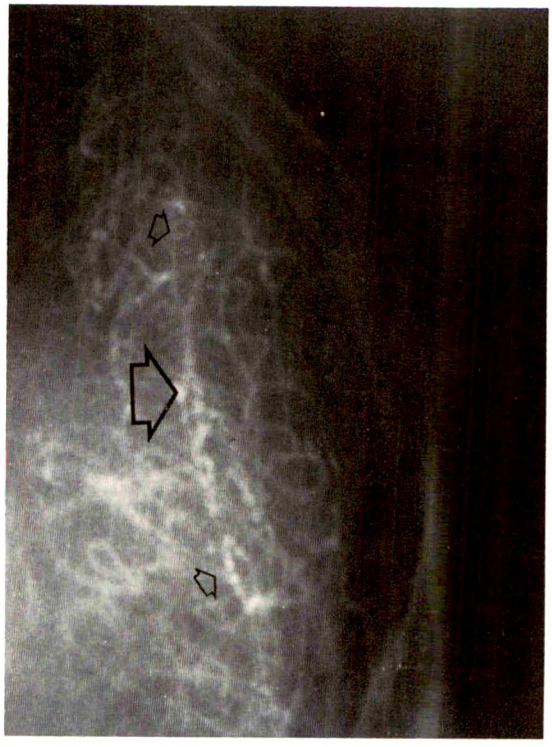


FIG. 3. Anteroposterior view, left common carotid angiogram. Arrows demarcate the middle meningeal artery displaced inwards, *i.e.*, the medial border of an epidural hematoma. Note that there are many vessels overlying the biconvex collection. There is extravasation of contrast medium along the course of the middle meningeal artery (compare with Figure 6*A*).

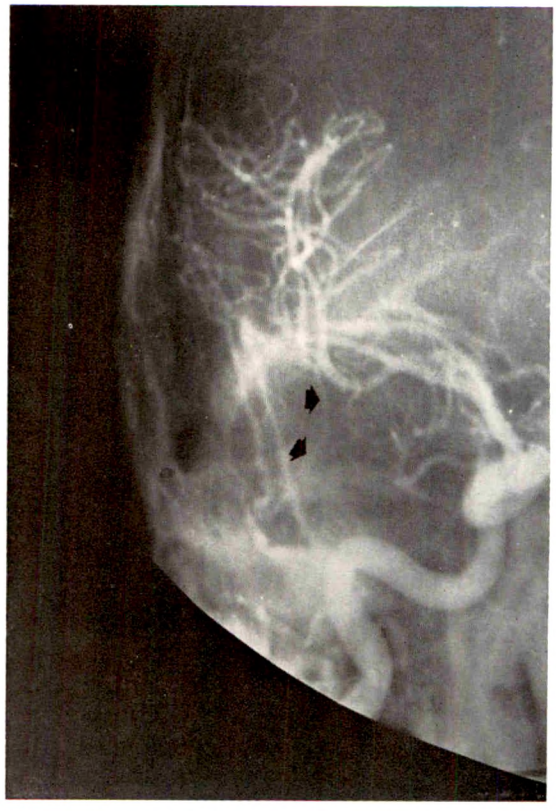


FIG. 4. Anteroposterior coned view of right common carotid angiogram. There is medial displacement of the low convexity segment of the middle meningeal artery (arrows) indicating an epidural process. A large middle cranial fossa epidural hematoma was drained in this case.

dle meningeal artery may be an important sign of an epidural hematoma. We have seen 2 cases in which the posterior division of the middle meningeal artery showed interruption without extravasation on angiography (Fig. 5). This point, the site of bleeding, was verified surgically to be completely transsected in each case. The reason for lack of extravasation of the contrast material in these 2 cases is not clear. Similarly, the same angiographic pattern has been seen by us in complete avulsion of the internal carotid artery. Intermittent spasm at the site of transsection is the most likely explanation for the pseudo-obstructed appearance in both of the situations described.

4. *Middle Meningeal Artery Extravasation.* A tear of the middle meningeal artery may be diagnosed by extravasation of contrast material. Contrast material may be

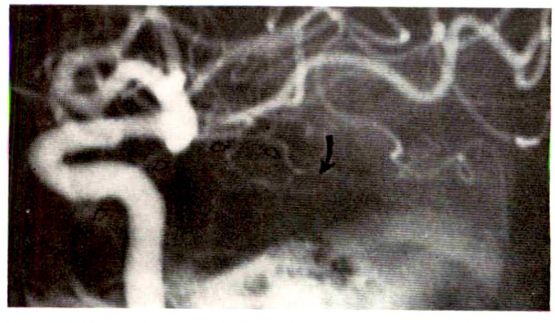


FIG. 5. Lateral view, left common carotid angiogram. Clear black arrows point to the posterior division of the middle meningeal artery. Solid black arrow indicates the site of apparent obstruction of this vessel. At surgery this was the site of complete transsection resulting in a massive epidural hemorrhage.

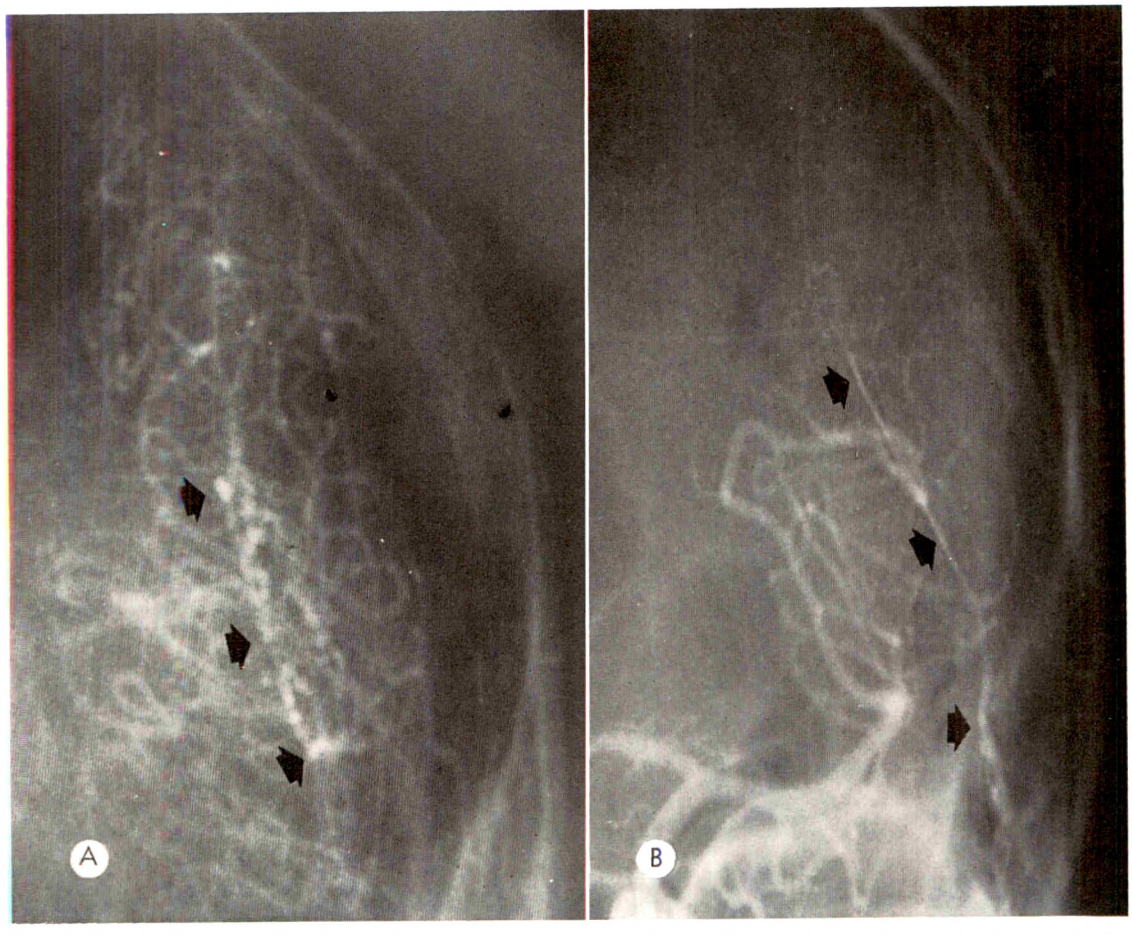



Fig. 6. (*A*) Anteroposterior view, right common carotid angiogram (same case as Figure 3). Arrows depict berry-like collections of contrast material along the course of the medially displaced middle meningeal artery. Multiple lacerations of this artery were found at surgery. This case represents extravasation of contrast medium into the epidural space. (*B*) Anteroposterior view, left common carotid angiogram. Arrows point to extravasation of contrast material along the course of a medially displaced middle meningeal artery in a case of a large epidural hematoma. The contrast medium in this case lies within an associated fracture.

seen in the epidural space, fracture line, diploic space or beneath the subgaleal fascia.[10] Various combinations of any of the above may occur. Figure 6, *A* and *B* illustrates several examples of this phenomenon.

A more dramatic example of extravasation is the development of a fistula between the middle meningeal artery and the paired meningeal veins.[6] There may or may not be significant bleeding into the epidural space in the above situation. As a matter of fact, middle meningeal artery-vein(s) fistula have been described in which surgery disclosed no epidural collection at all.[14] Fincher[3] reported an arteriovenous fistula between the middle meningeal artery and the superior petrosal sinus with no associated epidural hematoma. Leslie *et al.*[6] described a "tram track" appearance as being due to arteriovenous fistula of the middle meningeal vessels. We have seen a similar case (Fig. 7). On our angiogram, the appearance is more likely due to extravasation into the sheath containing the artery and paired meningeal veins rather than a fistula.

5. *Pseudoaneurysm of the Middle Menin-*

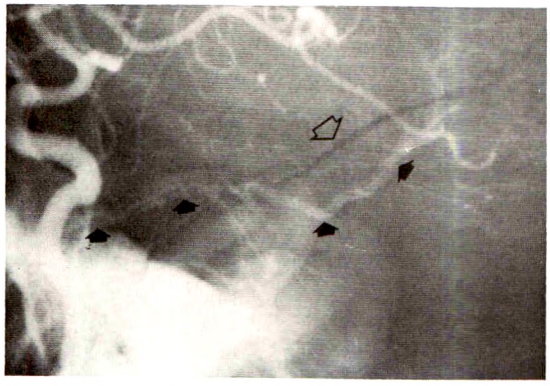


FIG. 7. Anteroposterior view, left common carotid angiogram. The small black arrows follow the course of the middle meningeal artery. There is a "tram track" appearance to the vessel. This appearance persisted for 6 seconds during serial angiography, suggesting, therefore, that we are seeing contrast material in the sheath of the middle meningeal artery and paired veins, rather than an arteriovenous fistula. The radiolucent paired defects on either side of the central linear opacity represent the silhouette of the paired meningeal veins.

*geal Artery*. Most aneurysms of the middle meningeal artery are pseudoaneurysms, *i.e.*, a hematoma communicating with an arterial lumen.[5,11] A true aneurysm of the vessel is exceedingly rare. The encasement of the middle meningeal artery in a deep bony groove of the skull almost isolated from the endosteum accounts for the containment of the hematoma and pseudoaneurysm formation. The same mechanism, according to Schechter *et al.*[10] accounts for the lack of significant bleeding in many cases showing extravasation. None of our cases illustrated this phenomenon.

6. *Displacement of a Dural Sinus*. Displacement of a dural sinus first described by Wickbom[12] indicates an epidural process. This applies to any dural sinus but is more commonly appreciated at the sagittal sinus level (Fig. 8). Rotation, vault asymmetry, and lack of a midline position of the sagittal sinus can account for an erroneous interpretation of displacement.[1] Venous washout from the cerebral circulation opposite to the injected side may prevent good sagittal sinus fill and simulate displacement (Fig. 9).

7. *Extravasation from a Dural Sinus*. Ruggiero *et al.*[9] were the first to describe extravasation from a dural sinus in an epidural hematoma. We have not seen this angiographic sign. Actual sinus bleeding is unusual, however; arachnoidal granulation tears are the usual course of epidural hematomas adjacent to the dural sinuses.[13]

8. *Avascular Mass*. When located in the floor of the middle cranial fossa, epidural hematomas present as avascular masses. The extracerebral location as evidenced by elevation of the anterior choroidal artery and lack of temporal artery draping do not differentiate them from subdural collections, avascular meningiomas, or a mass in

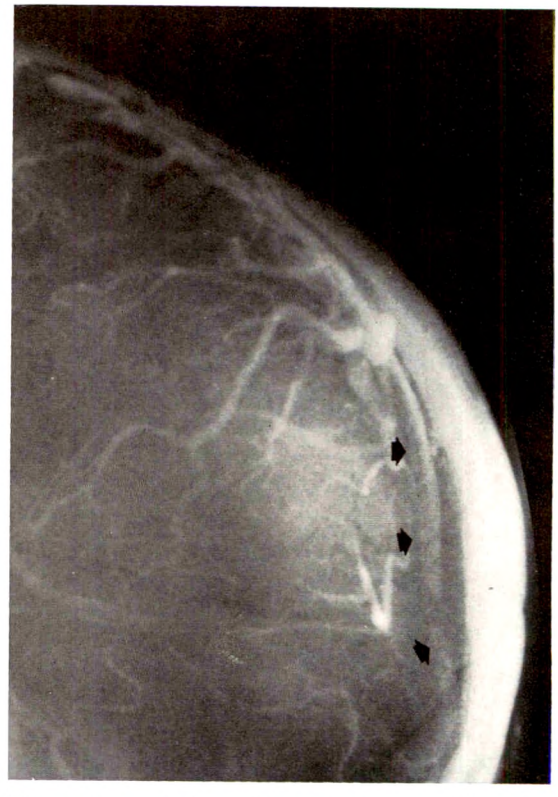


FIG. 8. Lateral view, right common carotid angiogram. The occipital portion of the sagittal sinus (arrows) is displaced forward. At surgery, a large epidural collection of blood was found between the calvarium and the sinus.

the inferior temporal gyrus. We have seen this appearance in several frontal and parieto-occipital epidural hemorrhages (Fig. 10). Usually in the latter two locations, oblique views will show an extracerebral avascular collection similar to the temporal convexity collections.

## DISCUSSION

Angiography has produced many signs which are suggestive, and in some cases diagnostic, of an epidural hematoma.

Extravasation from the middle meningeal artery into the epidural space or extravasation from a dural sinus is pathognomonic of epidural bleeding. On the other hand, spillage of the contrast material into the diploic space, subgaleal space, or into the middle meningeal veins may be a protective mechanism and frequently is associated with very little or no bleeding into the epidural space.[10] The formation of a pseudoaneurysm may be seen with very little epidural bleeding.

Displacement of the middle meningeal vessels or a dural sinus only indicates the epidural location of a process without differentiating blood, infection or neoplasm.

A lenticular avascular zone with slightly irregular margins and often several overlying vessels cannot be considered pathognomonic. Subdural empyema, chronic subdural hematomas, arachnoid cysts, and other disease processes may present a similar angiographic pattern.[2]

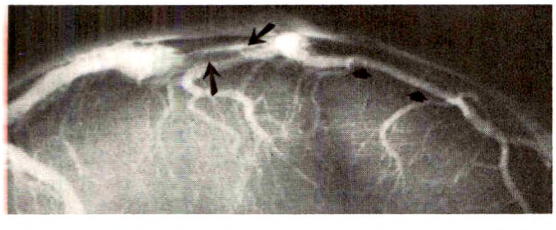


Fig. 9. Lateral view, left common carotid angiogram. The venous phase of the angiogram shows apparent downward displacement of a portion of the sagittal sinus (large arrows). The frontal segment of the sinus does not fill from this side. Small arrows indicate a draining parasagittal vein. The apparent displacement of the sinus is probably secondary to contralateral venous washout as a carotid angiogram on that side showed normal filling and position of the entire sagittal sinus.

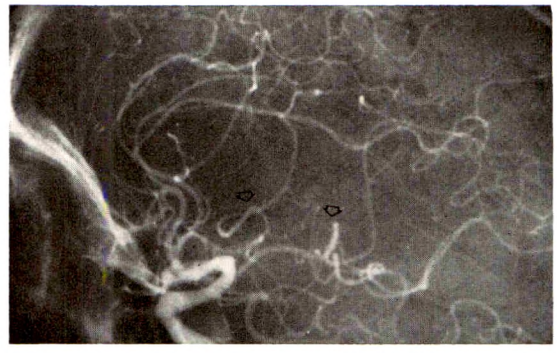


Fig. 10. Lateral view, right common carotid angiogram. The vessels of the insula (arrows) are pushed downwards. Above the arrows, in the region of the frontal operculum, the frontoparietal group of vessels are stretched; the findings are consistent with a lateral frontal mass. Oblique views in this case failed to show the extracerebral location of this mass which proved to be an epidural hematoma. On plain skull roentgenograms there was evidence of a fracture situated low in the long axis of the ipsilateral frontal bone.

Amputation of the middle meningeal artery, we would assume, could be occlusive disease, and may not necessarily imply a transsection of this vessel; hence, this sign must be considered as suggestive, but not diagnostic of an epidural hematoma.

Lack of appropriate contralateral shift of the anterior cerebral artery has been suggested as an ancillary sign.[1] This perhaps is related to the contained smaller volume of an epidural hematoma as compared to an acute subdural hematoma, which usually spreads freely over the cerebral convexity. This sign, we feel, is of limited value and we have not been able to verify its validity in our series.

Circulatory slowing, secondary to increased intracranial pressure, is frequently seen but lacks specificity.

## CONCLUSIONS AND SUMMARY

The angiographic manifestations of epidural hematomas in 25 patients are presented. A lenticular shaped extracerebral

zone and an avascular mass were the two most common angiographic findings, followed by medial displacement of the middle meningeal artery. Amputation of the middle meningeal artery, varied patterns of extravasation, and dural sinus displacement were also seen.

Extravasation from the middle meningeal artery into the epidural space or extravasation from a dural sinus are pathognomonic of epidural bleeding.

Awareness of the signs mentioned and others noted in Table 1 is important for prompt diagnosis, location and early treatment of epidural hematomas.

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VOL. 101, NO. 1

# THE CORONAL TANGENTIAL VIEW FOR THE DEMONSTRATION OF EXTRACEREBRAL HEMATOMAS*

*By* KEITH RABINOV, M.D.,† *and* PETER LAVENDER, M.R.C.P., F.F.R.‡
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THE presence of localized subdural or epidural hematomas on the convexity may occasionally not be demonstrated in routine views and the precise localization or extent of other such collections may not be clearly shown. As pointed out by Zingesser, Schechter and Rayport,[2] oblique roentgenograms obtained by rotating the head towards or away from the side under investigation will usually show frontal and occipital collections. The true situation may be revealed by an apparently little used view. Benedetti, Constantini and Ravasini[1] reported use of a view obtained by tilting the head 30 degrees away from the incident roentgen-ray beam in the lateral projection. We have been employing a modification of this "coronal tangential view."

## TECHNIQUE

Common carotid artery puncture and angiography are performed in the usual way. Anteroposterior, lateral and oblique views are obtained, with the addition of a coronal tangential view.

For examination of the right side (Fig. 1, *A* and *B*) the head is tilted toward the left and the tube angled craniad so that the roentgen-ray beam strikes the skull just above the ear at an angle of about 35 degrees with the sagittal plane. For examination of the left side, the head is tilted toward the right.

Alternatively, the base view may be used for each side of the head, with films in the anteroposterior box. If this position is employed, the grid must be rotated so that it is parallel with the roentgen-ray beam. The exposure factors are the same as for the lateral view.

## RESULTS

The appearance of the normal carotid angiogram in the coronal tangential view is shown in Figure 2, *A–C*. The middle cerebral artery branches and the cortical veins are seen adjacent to the cranial vault extending from the frontal to the occipital region. The middle meningeal artery is noted in its groove in the bone. Examples of subdural collections are shown in Figures 3, *A–C*; and 4, *A–D*.

## DISCUSSION

The majority of subdural and epidural hematomas are demonstrated in the standard anteroposterior, oblique and lateral views. There remain cases, however, where the collection is far anterior or posterior, or very localized, so that separation of the cortical vessels from the skull vault may not be evident. In these cases, the presence and distribution of these collections may be better visualized in the coronal tangential view. In addition, the middle meningeal artery in its groove is shown tangentially, which may aid in distinguishing between subdural and epidural collections. Since separation of vessels from the cranial vault may also occur in the presence of brain atrophy, avascular brain tumors or brain infarcts, the differential diagnosis between these entities and extracerebral hematomas may at times prove difficult and require careful angiographic as well as clinical assessment.

## SUMMARY

A little used coronal tangential view is described to better demonstrate the position and extent of extracerebral collections.

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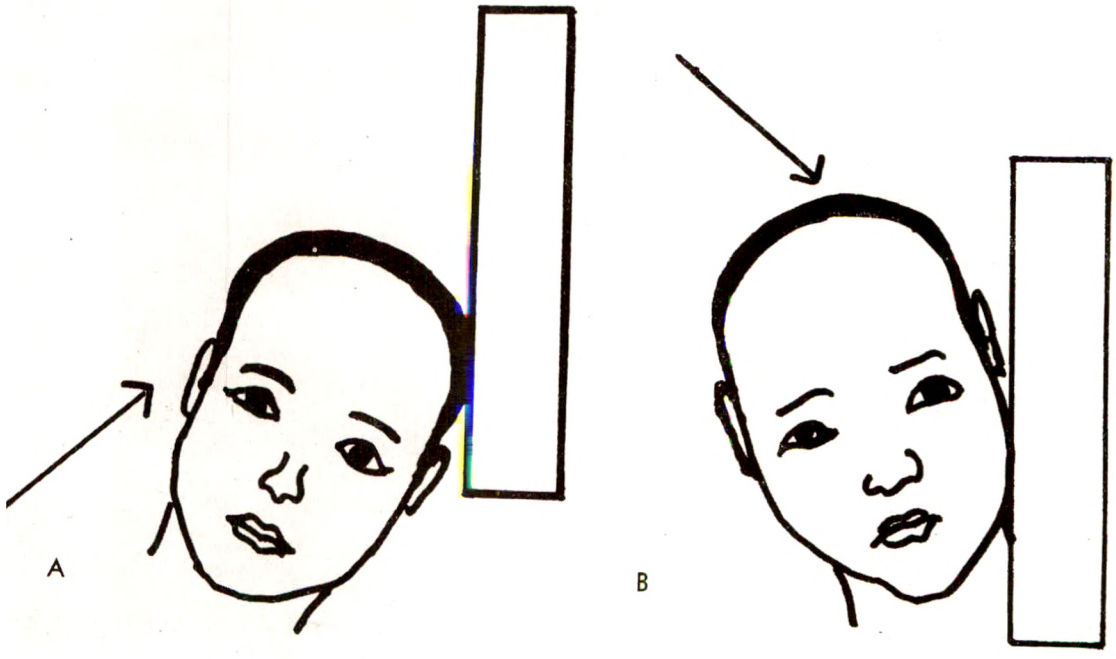
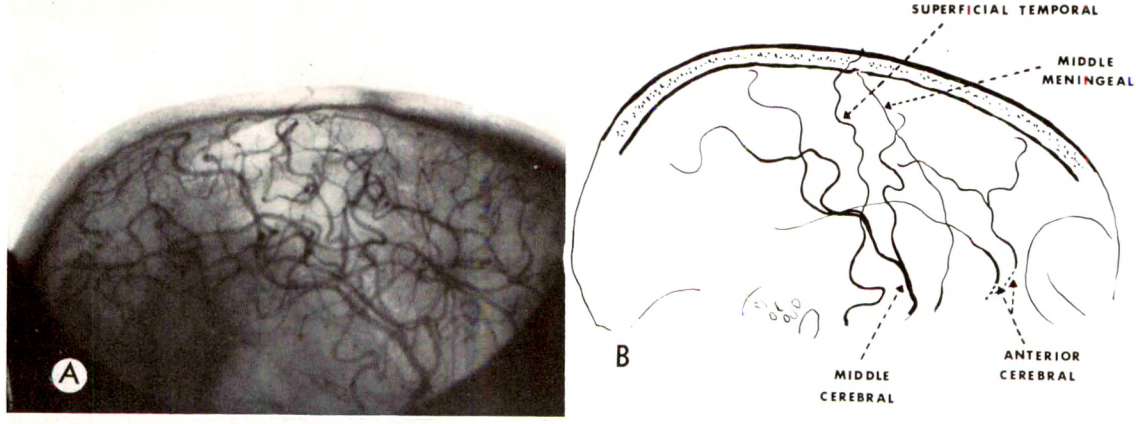



FIG. 1. (*A* and *B*) Position of the head in relation to the film and incident roentgen-ray beam. The roentgen-ray beam should be at an angle of approximately 35 degrees to the sagittal plane of the skull.



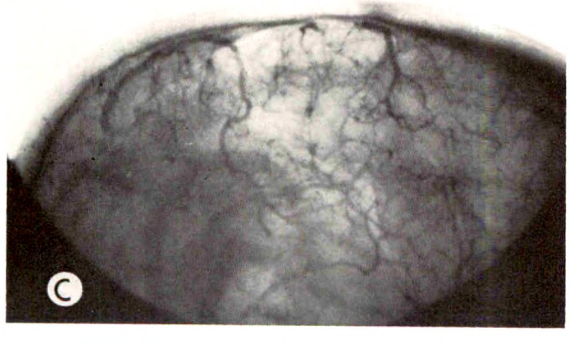




FIG. 2. (*A*) Coronal tangential view showing arterial phase using a common carotid artery injection. Note the close approximation of the cortical branches to the inner table of the skull and the position of the anterior branch of the middle meningeal artery. (*B*) Line drawing of *A*. (*C*) Venous phase.

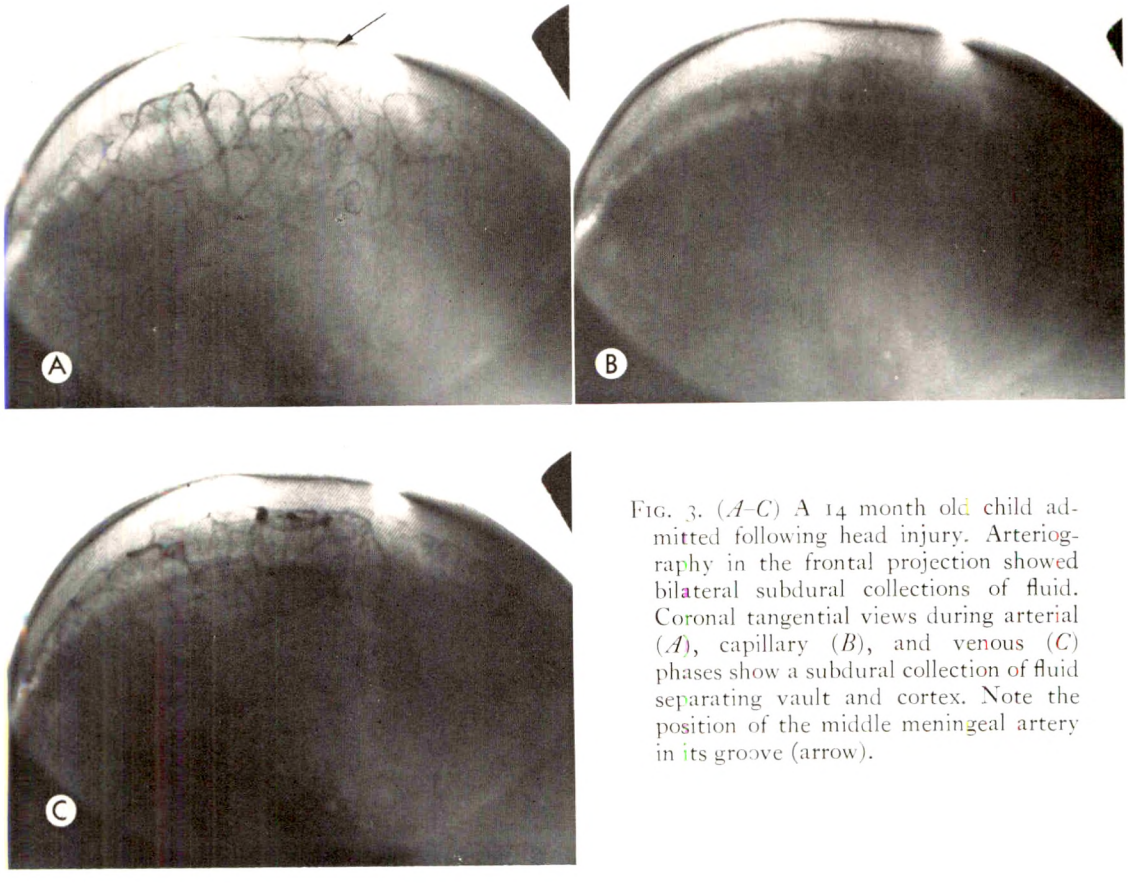



FIG. 3. (*A–C*) A 14 month old child admitted following head injury. Arteriography in the frontal projection showed bilateral subdural collections of fluid. Coronal tangential views during arterial (*A*), capillary (*B*), and venous (*C*) phases show a subdural collection of fluid separating vault and cortex. Note the position of the middle meningeal artery in its groove (arrow).

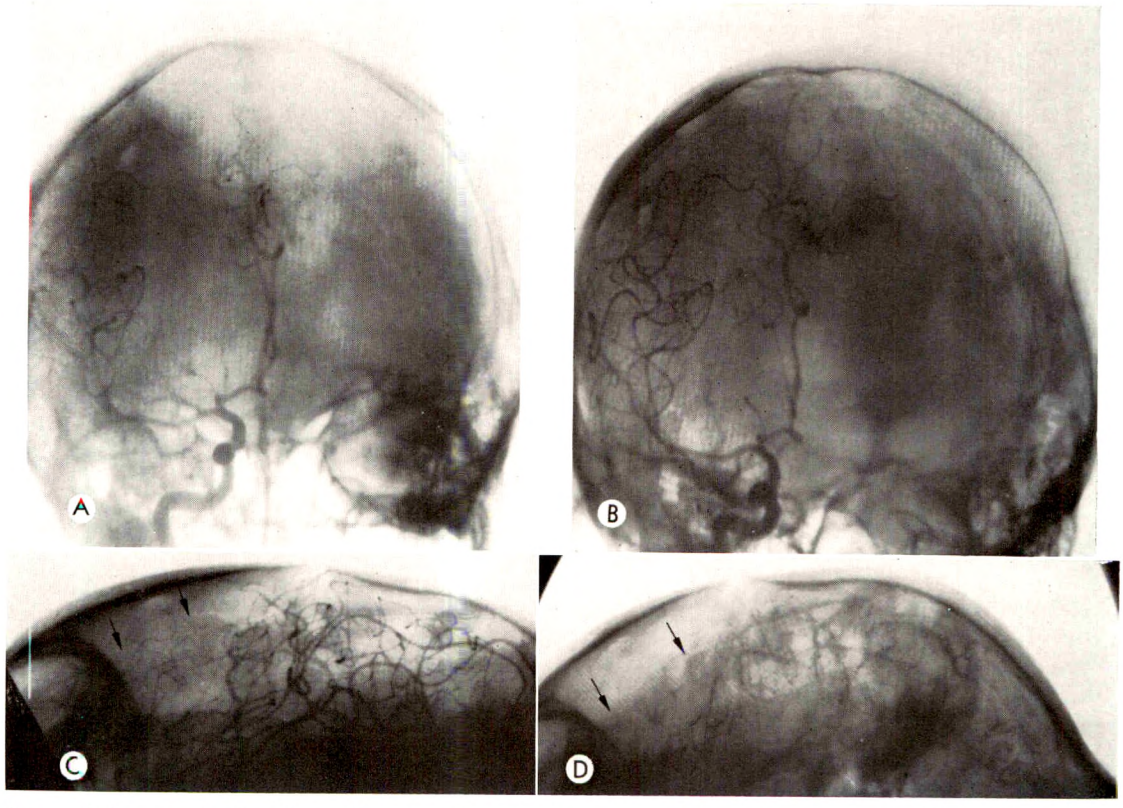



FIG. 4. A 65 year old physician suffered recurrence of symptoms following drainage of a subdural hematoma resulting from trauma. (*A*) Anteroposterior postoperative carotid arteriogram shows a displacement of the anterior cerebral artery but no hematoma is seen in this view. (*B*) Anteroposterior oblique carotid arteriogram shows the fronto-parietal region with minimal separation of the cortical vessels from the vault. (*C*) Coronal tangential view shows a 2 cm. separation of cortex and vault in the frontal region. The arrows indicate the cortical branches separated from the vault above the orbit. Note the superimposed midline branch of the calloso-marginal artery. (*D*) The venous phase delineates the well localized hematoma (arrows).

This view may also demonstrate collections not seen in standard views and may help to differentiate between subdural and epidural collections of fluid.

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# THE HANGING HEAD POSITION FOR DETECTION OF SITE OF INTERNAL CAROTID ARTERY OCCLUSION*

*By* ROBERT L. KOCH, M.D., WILLIAM P. BIEBER, M.D., *and* MALCOLM C. HILL, M.B.
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ANGIOGRAPHIC diagnosis of occlusion of the internal carotid artery is frequently uncertain. The findings at postmortem examination do not always agree with those of the roentgen examination.[1–6]

Newton and Couch[4] have implicated certain possible causes of error in the diagnosis of occlusion of the internal carotid artery. These are subintimal injection, subintimal hematoma, extravascular injection, external carotid puncture, distal internal carotid occlusion and increased intracerebral pressure.

The value of delayed roentgenography in apparent internal carotid artery occlusion has been emphasized by Murphey and Shillito.[3] With this technique, however, they experienced success only when the block was above the ophthalmic artery, which maintained a slight flow in the internal carotid artery.

Illustrations in several articles[1–3,5] have shown a tapering column of contrast medium ending in the most dependent portion of the internal carotid artery, usually near the first cervical vertebra. In more recent reports[1,5] this tapering was recognized as a layering phenomenon.

By means of a simple variation in the technique of carotid angiography, differentiation between cervical and cranial occlusion is possible. We call it the "hanging head" position.

The hanging head position is achieved by elevating the thorax and shoulders above the table top with sponges. This elevation allows the head to fall back in full extension (Fig. 1, *A* and *B*) and maintenance of a needle in the carotid artery is not difficult. Few roentgenograms are needed, and the timing should be delayed.

The following case report illustrates the use of this technique.

## REPORT OF A CASE

A 45 year old woman who had had mitral valve disease since the age of 8 years, arrived in the emergency room of the San Francisco General Hospital shortly after falling from a chair. She was unable to speak and was semicomatose, and a left Babinski sign was noted. Hemiparesis was thought to be present on the left. Mitral murmurs and atrial fibrillation were additional findings; the left popliteal pulse was absent.

A right carotid embolus was suspected and immediate roentgenograms were obtained. A No. 7 French teflon catheter was passed into the aortic arch from the right axillary artery. The roentgenograms obtained on arch injection (Fig. 2*A*) showed a patent left carotid artery and normal vertebral arteries. The right internal carotid artery appeared to be obstructed near the first cervical vertebra. The right external carotid artery did not fill. The catheter was withdrawn into the right subclavian artery and a reflux injection of renografin 60 was made. On the 12 second roentgenogram, the column of contrast medium again appeared to end in a tapered configuration near the first cervical vertebra (Fig. 2*B*). This was thought to represent a layering phenomenon rather than the actual site of obstruction, and the hanging head position was used for the first time. On repeated injection the column advanced slowly to the cavernous portion of the internal carotid artery, appearing as a layering on the 12 second roentgenogram (Fig. 2*C*).

Although the exact site of obstruction of the cranial internal carotid artery was not reached, obstruction in the neck was ruled out. The patient was treated conservatively. The severe neurologic deficits continued and were associated with convulsions.

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FIG. 1. (*A*) Routine position on carotid angiography. (*B*) The hanging head position.

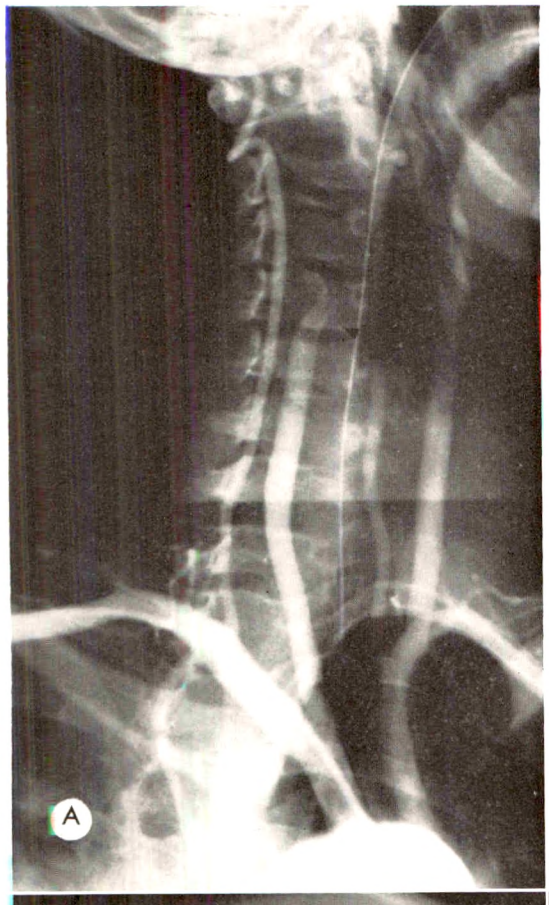
## MATERIALS AND METHOD

A phenomenon as basic as layering should be easily reproducible in the laboratory. To investigate the validity of the roentgenographic impression described in the case report, we constructed a simple model of the carotid circulation in which saline in an enema bag was suspended 150 cm. as the source of flow. A glass Y connector formed the common carotid bifurcation. A flow of 3 cc. per second through the tube representing the external artery was maintained during all roentgen exposures. The distal end of the tube representing the internal carotid artery was occluded by a closed stopcock. The usual technique was simulated by introducing, with a hand injector, 11 cc. of 60 per cent renografin through an arteriography needle. The roentgenograms obtained (Fig. 3, *A–D*) are a graphic demonstration of the layering phenomenon and how it can be overcome by positioning.

The study was repeated with a No. 25 needle replacing the closed stopcock. Flow of 1 drop per second was used to simulate a patent ophthalmic artery. As expected, the column of contrast medium advanced very slowly, approximately 2 cm. per minute. The head of the column resembled that seen in Figure 3*B*.

## DISCUSSION

In their study of intracranial aneurysms, Schechter and Elkin[6] showed that in a slow-flow system, the contrast medium will separate from the blood. A layering is then seen in the most dependent part of the aneurysm. This phenomenon is based upon the greater weight of the contrast agent; *i.e.*, the specific gravity of renografin 60 is 1.33, whereas that of blood is 1.06. The authors concluded that a false evaluation of







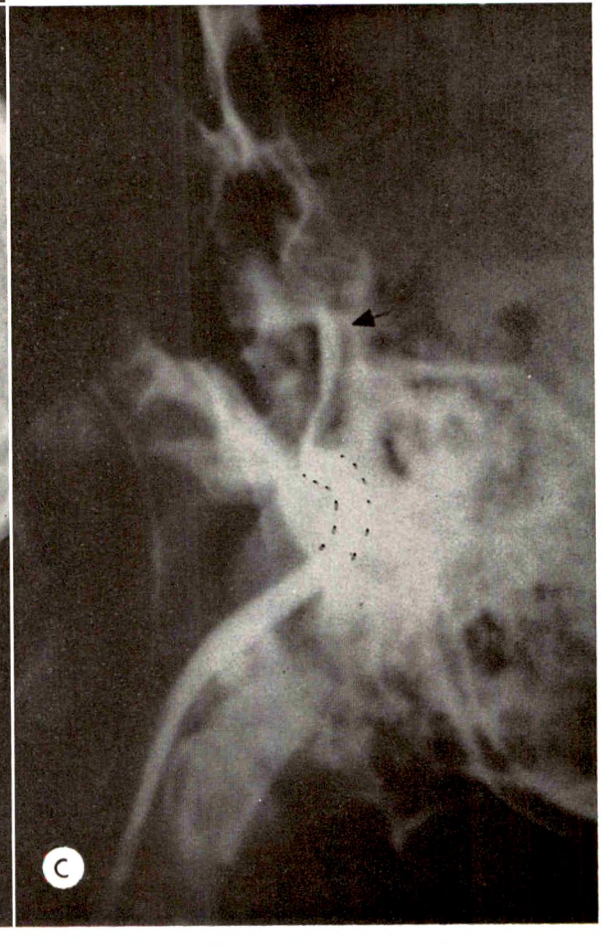



FIG. 2. (*A*) The early phase after injection into the aortic arch. The arrow indicates the slowly advancing column of contrast medium. (*B*) A 12 second roentgenogram, right subclavian injection in the standard position. The contrast column ends in the neck (arrow). (*C*) A 12 second roentgenogram, right subclavian injection, in the hanging head position. The column ends in the cavernous portion of the internal carotid artery (arrow). The image of the petrous portion is enhanced.

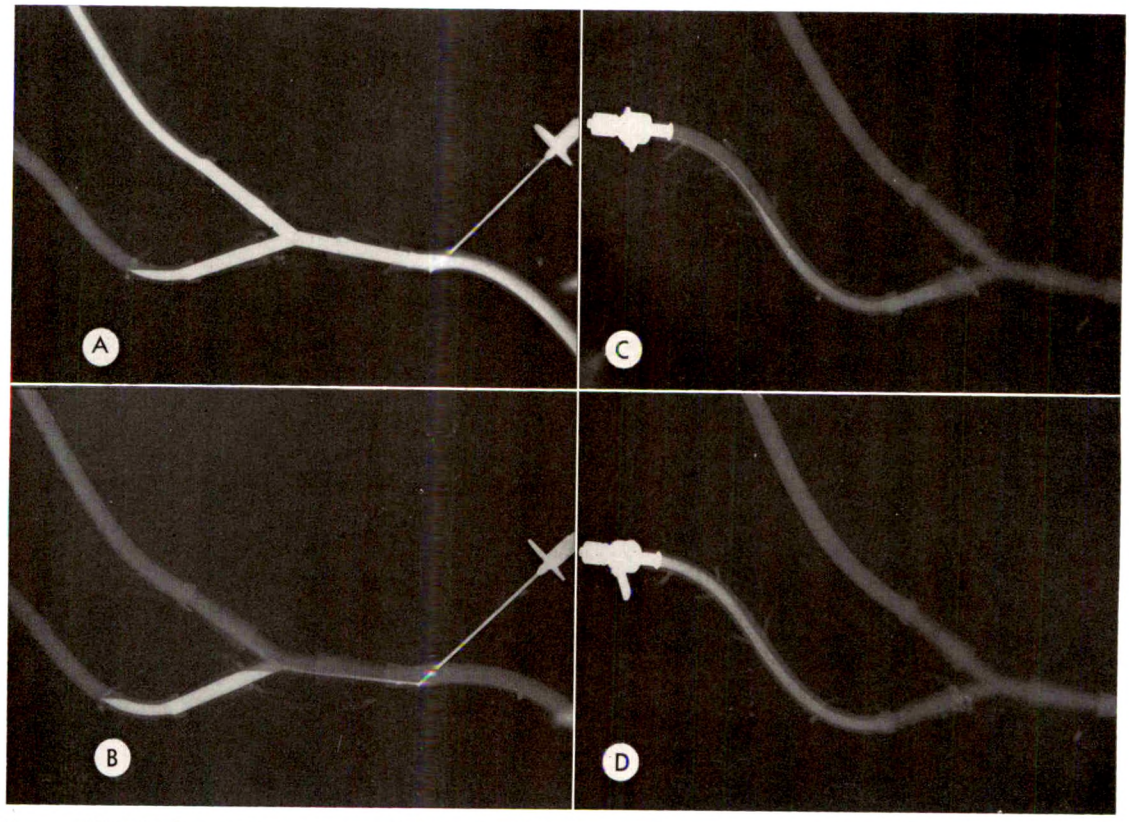



FIG. 3. Model of carotid circulation. (*A*) End of injection in the standard position. The contrast medium flows through the "external carotid artery." (*B*) One minute after injection in the standard position. Pseudo-obstruction of the "internal carotid artery" from layering. (*C*) A 10 second roentgenogram in the hanging head position 50 degrees off the horizontal. Contrast medium is flowing toward the obstruction. (*D*) At 20 seconds in the hanging head position, contrast medium has flowed by gravity to the point of obstruction.

aneurysm could be avoided by changing the patient's position.

This same phenomenon will occur in any part of the vascular system where flow is greatly retarded or absent. The internal carotid artery, without branching in its cervical portion and turning upward in the standard arteriographic position, is especially subject to stratification when occluded distally.

We have not had occasion to use the hanging head position in pseudo-occlusion of the internal carotid artery secondary to increased intracranial pressure. Neither have we tried the prone position, which would be technically possible with transfemoral carotid artery catheterization. This position may also be valuable and would allow roentgenography in the frontal view.

SUMMARY

A case report and an experimental model are presented to demonstrate how layering of contrast medium may stimulate an occlusion of the cervical internal carotid artery, and how the hanging head position prevents false localization.

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# A TECHNIQUE OF SIMULTANEOUS TWO PHASE CAROTID ANGIOGRAPHY*

*By* TAKASHI IWABUCHI, M.D.,† ZENTARO ITO, M.D.,† *and* JIRO SUZUKI, M.D.‡
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AN ANGIOGRAPHIC technique was devised to visualize simultaneously the arterial and venous phases with a single exposure in carotid angiography. This technique has been applied in patients with various intracranial disorders. It proved valuable in enhancing diagnostic accuracy.

## TECHNIQUE

The preparations and precautions as regards angiographic complications are similar to those used in ordinary carotid angiography. For adults, in general, intravenous pentobarbital anesthesia is employed. Twenty milliliters of urografin 76 per cent are warmed up to body temperature in a syringe. The common carotid artery is punctured percutaneously. A first 10 ml. of the contrast material is rapidly injected and after 6 seconds the remaining 10 ml. is injected. As in ordinary carotid angiography, the film is exposed after 8 ml. of the second injection. Smaller doses are utilized in infants and children. By this technique, both arterial and venous phases can be visualized simultaneously during one single exposure, with the use of a relatively small amount of contrast material.

## MATERIAL AND RESULTS

A total of 36 cases, 24 males and 12 females, were subjected to the examination. The ages of the patients ranged from 1 year and 2 months to 61 years.

Sixteen cases with mild head injury or migraine had no abnormal findings of ordinary carotid angiography or neurologic examination. The simultaneous two phase carotid angiograms of these patients were considered to be normal patterns.

A normal vascular pattern is illustrated in Figure 1, *A* and *B*. The appearance rate of each tributary of the cerebral vessels in the lateral view of the simultaneous two phase carotid angiograms was evaluated. Each branch of arteries, both superficial and deep veins, and the superior sagittal sinus could be well visualized in most cases. The inferior sagittal sinus and transverse sinus, however, failed to be visualized in 6 of 16 cases (Table I).

Twenty cases were proved to have intracranial disease such as brain tumors, subdural hematomas, etc. (Table II). In 18 of the 20 cases the simultaneous two phase carotid angiography revealed abnormal patterns. Some of these abnormal patterns are illustrated in the following representative case reports.

## REPORT OF CASES

CASE I. This patient was a 32 year old woman with a meningioma in the right parietal region. The vascularity around the tumor and the tumor stain were shown more clearly and comprehensively by the simultaneous two phase carotid angiography than by the ordinary angiography (Fig. 2, *A* and *B*).

CASE II. This patient was a 33 year old woman with sphenoidal ridge meningioma on the left side. Different tumor stains were shown in the arterial or venous phase by ordinary carotid angiography (Fig. 3, *A* and *B*). Concomitant visualization of both arterial and venous phases in a single film resulted in a sharper demonstration of the tumor stain (Fig. 3*C*).

CASE III. This patient was a 48 year old man with chronic subdural hematoma. Ordinary carotid angiography failed to show displace-

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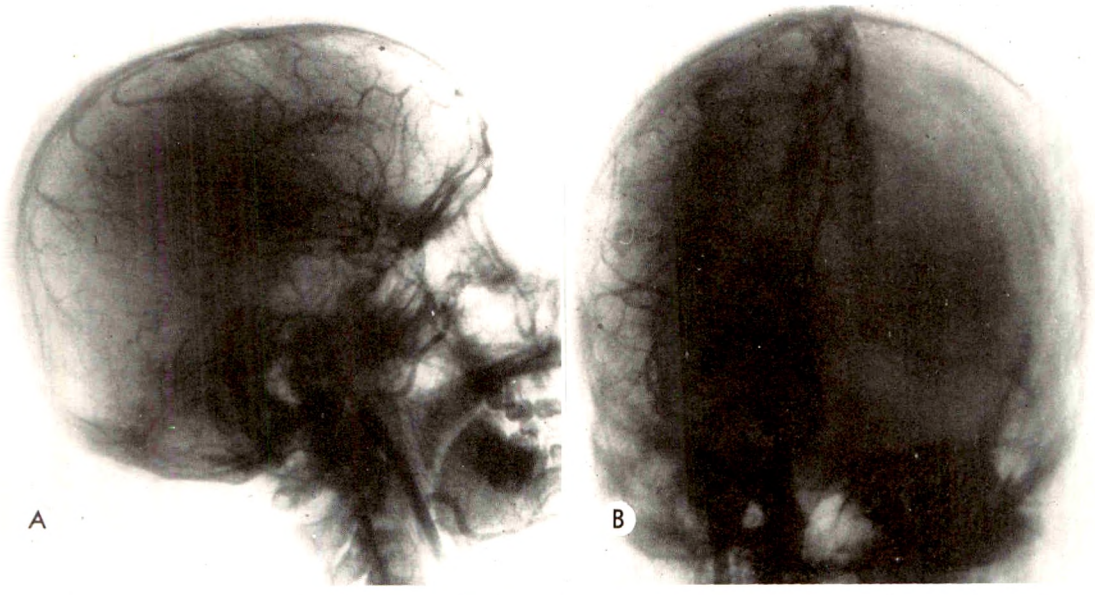



FIG. 1. A normal vascular pattern of simultaneous two phase carotid angiography. (*A*) Lateral view. (*B*) Oblique anteroposterior view.

ment of the anterior cerebral artery and it was difficult to detect the presence of an avascular area. This area, however, was well visualized in the simultaneous two phase carotid angiogram (Fig. 4).

## DISCUSSION

In general, dilatation of the lateral ventricles can be fairly well shown by the ordinary carotid angiography. The lateral roentgenogram in the arterial phase usually demonstrates tension arch-like displacement along with elevation of the anterior cerebral artery, whereas in the venous phase it shows dilatation of the lateral ventricles by changes of small veins running on the surface of the anterior portion of the lateral ventricles.[4,5] The simultaneous two phase carotid angiogram reveals more distinctly these abnormal patterns in a single film.

In cases with arteriovenous fistula, a

TABLE I

CEREBRAL VESSELS VISUALIZED BY THE SIMULTANEOUS TWO PHASE CAROTID ANGIOGRAPHY
(analysis of 16 cases)

| Name of Vessels | No. of Cases |
|---|---|
| Carotid syphon | 15 |
| Anterior cerebral artery | 14 |
| Middle cerebral artery | 15 |
| Superficial veins | 16 |
| Deep veins | 15 |
| Superior sagittal sinus | 14 |
| Inferior sagittal sinus | 10 |
| Transverse sinus | 10 |

TABLE II

CLINICAL DIAGNOSES OF 36 CASES SUBJECTED TO THE SIMULTANEOUS TWO PHASE CAROTID ANGIOGRAPHY

| Clinical Diagnoses | | No. of Cases |
|---|---|---|
| Normal | | 16 |
| Brain tumor | | 12 |
| Astrocytoma | 3 | |
| Meningioma | 3 | |
| Glioblastoma | 2 | |
| Oligodendroglioma | 1 | |
| Pinealoma | 1 | |
| Pituitary adenoma | 1 | |
| Lindau's disease | 1 | |
| Arteriovenous fistula | | 3 |
| Chronic subdural hematoma | | 3 |
| Arachnoiditis | | 1 |
| Cerebral palsy | | 1 |
| Total | | 36 |

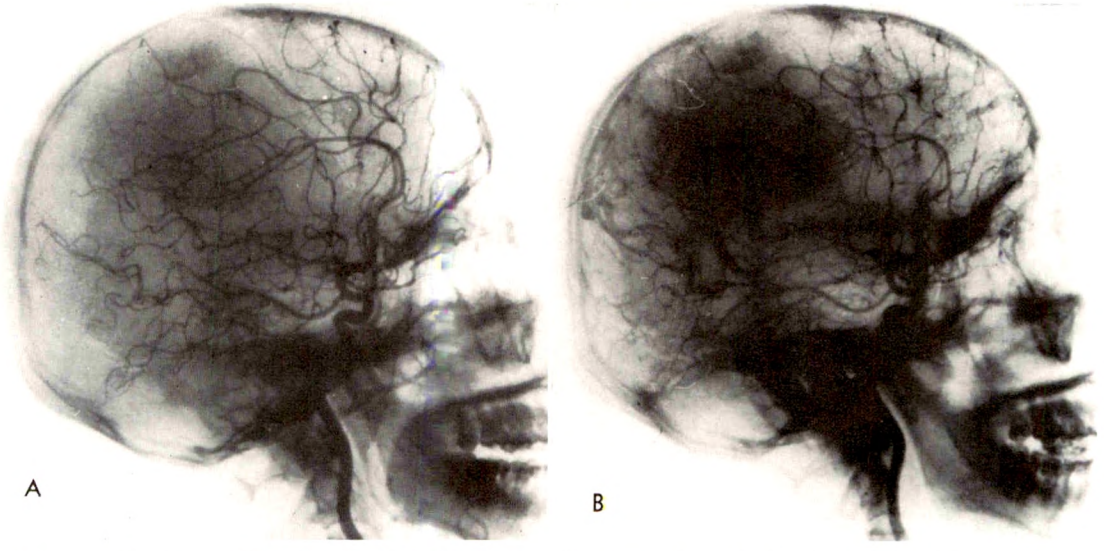



FIG. 2. Angiograms of Case 1, a 32 year old female with meningioma in the right parietal region. (*A*) Arterial phase of ordinary carotid angiography. (*B*) Simultaneous two phase carotid angiogram.

large amount of contrast material rapidly bypasses the fistula and, therefore, it is difficult to visualize the venous phase well by using our method. When our method was compared with that of the rapid serial angiography in 3 cases with arteriovenous fistula in the present series, the serial angiography proved superior in visualizing the feeding vessels and outlining the tract of the fistula. When compared to ordinary carotid angiography, however, somewhat better visualization of the arteriovenous fistula was obtained by our method. In all cases an accurate diagnosis was made also by the simultaneous two phase carotid angiography.

## COMMENT

Greitz[1] has indicated that in rapid serial angiography cerebral circulation time and appearance rate of each cerebral vessel are somewhat variable depending upon the chemical structure, concentration and amount of contrast material used or the pathologic condition of patients. It is widely accepted through the works of Grote,[2,3] Schiefer,[6] Tönnis,[7,8,9] and Wickbom[10] that cerebral circulation time, and appearance rate and pattern of vessels on carotid angiography, are characteristic of the type of brain tumor. Some kinds of tumor are visualized usually in the arterial phase, while others are well outlined only in the venous phase. It seems that the presence of a tumor can not be missed in the majority of cases by concomitant visualization of both arterial and venous phases. Accordingly, we have devised a simultaneous two phase carotid angiography, arterial and venous, as previously mentioned. Clinical application of this method of angiography confirmed its value in the diagnosis of various intracranial lesions. Angiograms obtained by this method appear at a glance to be complicated because of the superimposition of the arterial and venous phase vessel patterns. The main arterial and venous trunks are, however, easily identified, because the cerebral arteries and veins run not parallel but separately following different courses.

The simultaneous two phase angiography is of special value in detecting dilatation of the lateral ventricles and tumor stains and in outlining the tumor and minor avascular areas due to small intracranial hematomas.

The technique is simple, reliable, in-

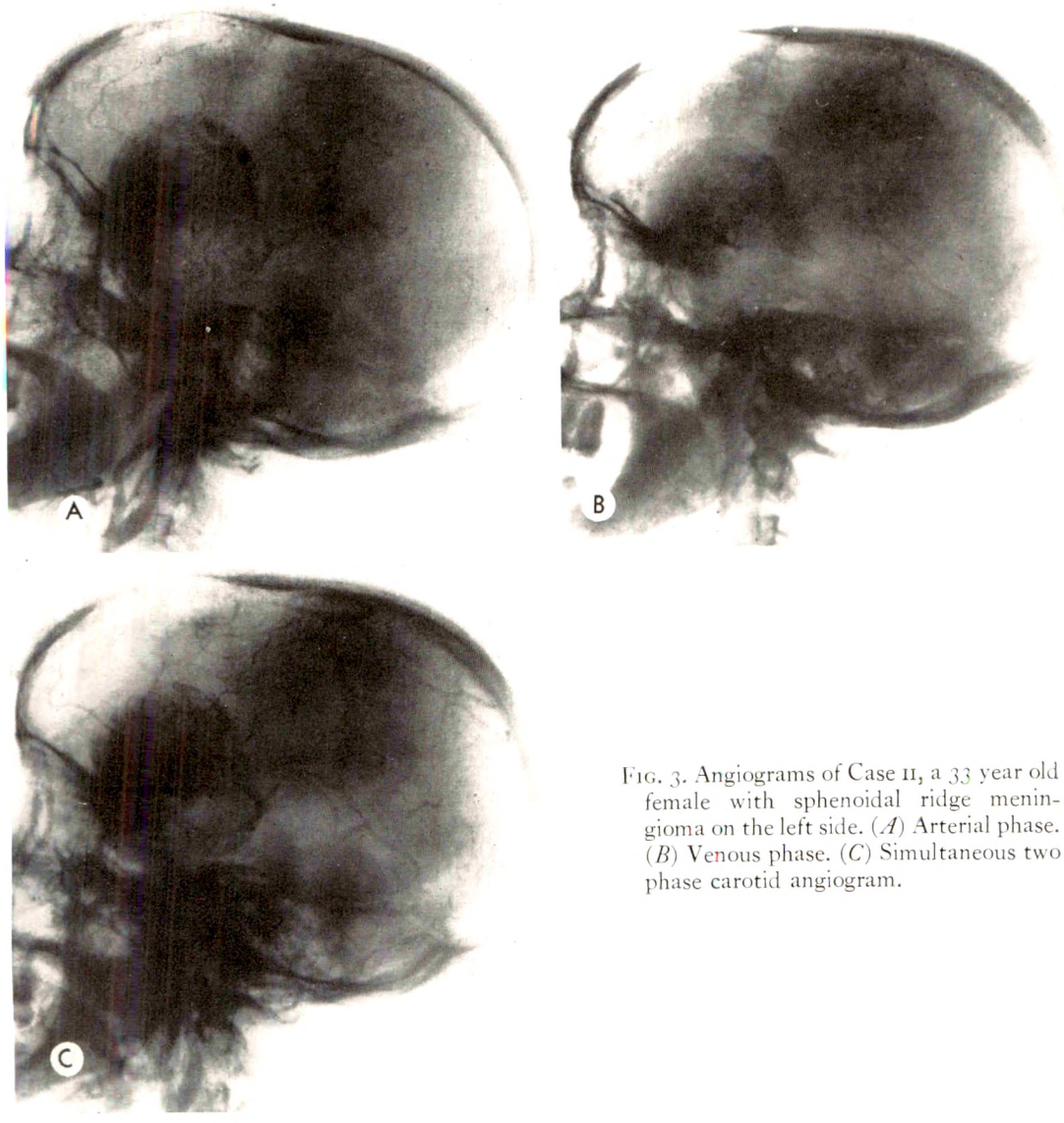



Fig. 3. Angiograms of Case 11, a 33 year old female with sphenoidal ridge meningioma on the left side. (*A*) Arterial phase. (*B*) Venous phase. (*C*) Simultaneous two phase carotid angiogram.

nocuous and does not require any special instrumentation.

## SUMMARY AND CONCLUSIONS

1. In simultaneous two phase carotid angiography, contrast material is injected twice at a 6 second interval. Immediately before the completion of the second injection, an angiogram is made encompassing the arterial and venous phases on a single film—hence, the name of "simultaneous two phase carotid angiography."

2. This technique was used in 36 patients whose ages ranged from 1 year and 2 months to 61 years. Comparative study was made of the appearance rate of each portion of the cerebral vascular trees in the normal and in cases with various intracranial lesions. The figures were compared with those of the separate visualization of the arterial and venous phases in the ordinary carotid angiography in all cases and of rapid serial angiography in a few.

3. The simultaneous two phase carotid angiography is superior to ordinary carotid angiography in revealing dilatation of the



FIG. 4. Simultaneous two phase carotid angiogram of Case III, a 48 year old male with chronic subdural hematoma.

lateral ventricles, tumor staining and/or outline of the tumor, or an obscure avascular area in head injury.

4. The technique is simple, reliable, nonocuous, and does not require any particular instrumentation.

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VOL. 101, NO. 1

# TECHNIQUE OF CEREBRAL ANGIOGRAPHY IN CHILDREN*

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CEREBRAL angiography is now an established part of the investigation of many neurologic conditions in the adult. It is of particular value in the management of cases with space occupying lesions of the brain and in cerebrovascular disease. In the field of pediatric neurology, however, the use of cerebral angiography has been more restricted, mainly because the usual techniques are difficult to apply in children and also because there is a "feeling," unsupported by fact, that the procedure carries an added risk in children. It is the purpose of this paper to describe and discuss the technique used by the authors for cerebral angiography in children which is felt to be technically simple, safe and to give excellent visualization of the total cerebral circulation.

## TECHNIQUE

The technique consists of percutaneous retrograde right brachial injection using Rochester plastic needles and a pressure injector.

*Anesthesia.* The procedure is preferably done under general anesthesia, although with older children local infiltration with 2 per cent xylocaine (lidocain U.S.P.) coupled with suitable premedication is sometimes sufficient.

*Puncture.* The arm is extended and attached to a wooden arm board with a piece of adhesive tape around the wrist. The brachial artery is then palpated at the elbow and is punctured and transfixed by the Rochester needle. The needle, which consists of an inner metallic needle and an outer plastic catheter, is then gradually withdrawn until a flow of blood is obtained, when it is again advanced and the inner metallic portion is removed. The small polyethylene catheter is then slowly withdrawn while the operator watches for a free pulsatile flow of blood indicating that the tip of the catheter is lying within the lumen of the artery. At this point, it is usually easy to advance the catheter along the arterial lumen. In the case of any difficulty, a guide wire with a soft flexible end can be introduced into the artery through the plastic catheter and the catheter advanced over the wire which is then withdrawn. The catheter is firmly connected to a hard plastic connecting tube and flushed every 2 or 3 minutes with normal saline. Satisfactory visualization of cervical and intracranial vessels may be obtained with 60 per cent methylglucamine diatrizoate (renografin 60). The bolus consists of 20 to 30 cc. for each injection depending on the age of the child.

*Roentgenographic Technique.* Anteroposterior and lateral serialograms are obtained using an automatic Amplatz pressure injector at 120–150 pounds per square inch of pressure. The anteroposterior view requires a 20–25 degree tilt of the central roentgen beam caudally. The head is either rotated 90 degrees to the side or a horizontal roentgen-ray beam is used for the lateral view. A satisfactory arterial, capillary and venous series can be obtained by exposing the first 6 films at the rate of 2 per second followed by a pause of 1–2 seconds, then exposing the remaining films at the rate of 1 per second.

## RESULTS

This technique has been used in 12 children ranging from 5 to 15 years of age. In all cases, there was excellent visualization of

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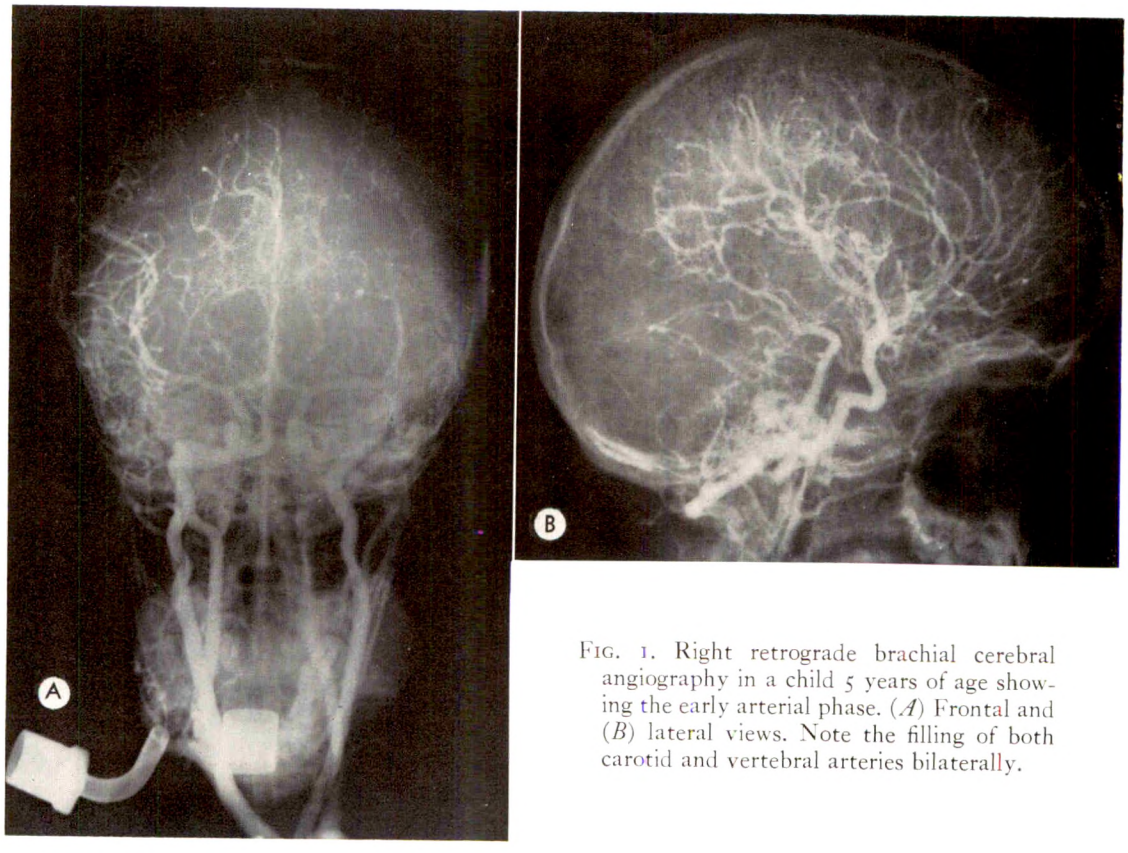



FIG. 1. Right retrograde brachial cerebral angiography in a child 5 years of age showing the early arterial phase. (*A*) Frontal and (*B*) lateral views. Note the filling of both carotid and vertebral arteries bilaterally.

both carotid arteries and of the right and usually the left vertebral arteries with total opacification of intracranial vessels (Fig. 1–4). There were no serious complications. There may be some mild discomfort due to extravasation of a small amount of blood at the puncture site.

## DISCUSSION

The first cerebral arteriography in a child was performed by Moniz[8] in 1928, using direct puncture of the carotid arteries. This method has been used widely in adults but few reports can be found in the literature dealing with its use in children. Torkildsen and Koppang[17] published the first report on percutaneous carotid angiography in 33 children, the youngest 2 years of age. Picaza[11] reported his experience in 50 children ranging in age from 9 days to 15 years. He used an open method of carotid arteriography in most cases and outlined the indications for and the complications of the procedure in his report. The first detailed paper on cerebral arteriography in children was published in 1955 by Poser and Taveras[12] who used the percutaneous carotid method. They described their technique, compared its morbidity with pneumoencephalography under similar conditions and discussed the complications. The retrograde brachial route was first suggested by Gould, Peyton, and French[5] as a method for vertebral arteriography in infants and children. They felt that the methods described at that time; namely, radial artery catheterization,[13] percutaneous puncture in the neck,[16] and percutaneous puncture at the base of the skull[14] were difficult to apply to children. They exposed the brachial artery in the distal 1/3 of the arm under general anesthesia and then injected 8–18 cc. of radiopaque contrast medium through a 15–18 gauge needle while the artery was clamped distally beyond the needle. The right ca-

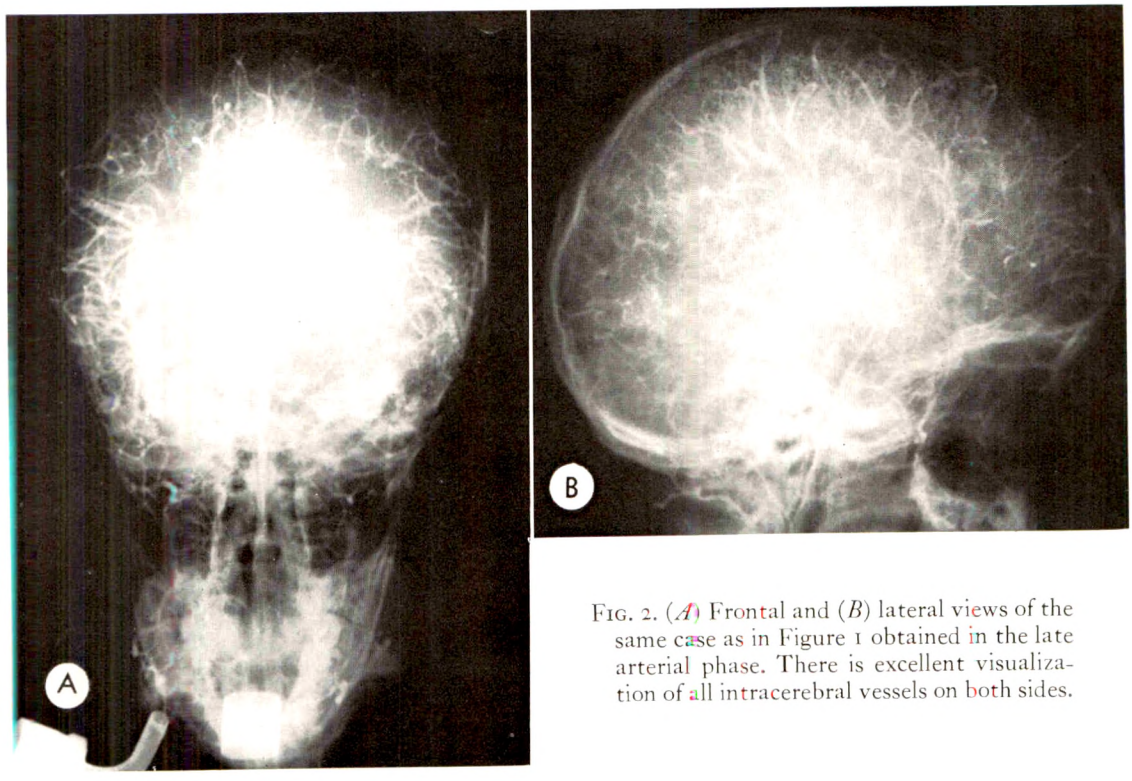



Fig. 2. (*A*) Frontal and (*B*) lateral views of the same case as in Figure 1 obtained in the late arterial phase. There is excellent visualization of all intracerebral vessels on both sides.

rotid and vertebral systems were outlined in all 7 cases examined. Following this preliminary report, the method of open retrograde brachial injection was also recommended by Kuhn[6] who described the technique in detail and discussed its merits. Carrea and Schuster[1] reported the results of 128 open retrograde brachial injections in infants and children up to the age of 5 years. Filling of both carotid arteries and at least one vertebral artery was obtained in 90 per cent of normal children under 1 year of age, but the incidence of total cerebral angiography decreased sharply in older children.

A different approach to carotid arteriography was proposed by Weiner, Azzato, and Mendelsohn.[18] They exposed the superficial temporal artery in the preauricular region, ligating it distally and then passed a plastic catheter proximally along the lumen for 12–15 cm. until it reached the common carotid artery. Anteroposterior and lateral views were obtained following injection of 6–10 cc. of contrast medium for each view. The authors commented on the possible application of this technique for cerebral angiography in children and used it successfully in 1 infant, 10 months of age. Later, Oller *et al.*[9] reported the use of combined open retrograde right brachial and left temporal arterial catheterization in both adults and children to obtain total cerebral and extracerebral angiography at one operative session. They found the temporal catheter angiography "a safe and reliable method for carotid angiography and an excellent technique for pediatric patients."

Cerebral angiography following the intravenous injection of radiopaque contrast material was recommended by Pheline *et al.*[10] in infants and also by Steinberg *et al.*[15] This has not provided satisfactory visualization in the hands of others and carries potential complications from injection of a large bolus of contrast material, such as an anaphylactic reaction.

The use of total aorto-cervico-cerebral angiography after retrograde femoral cath-

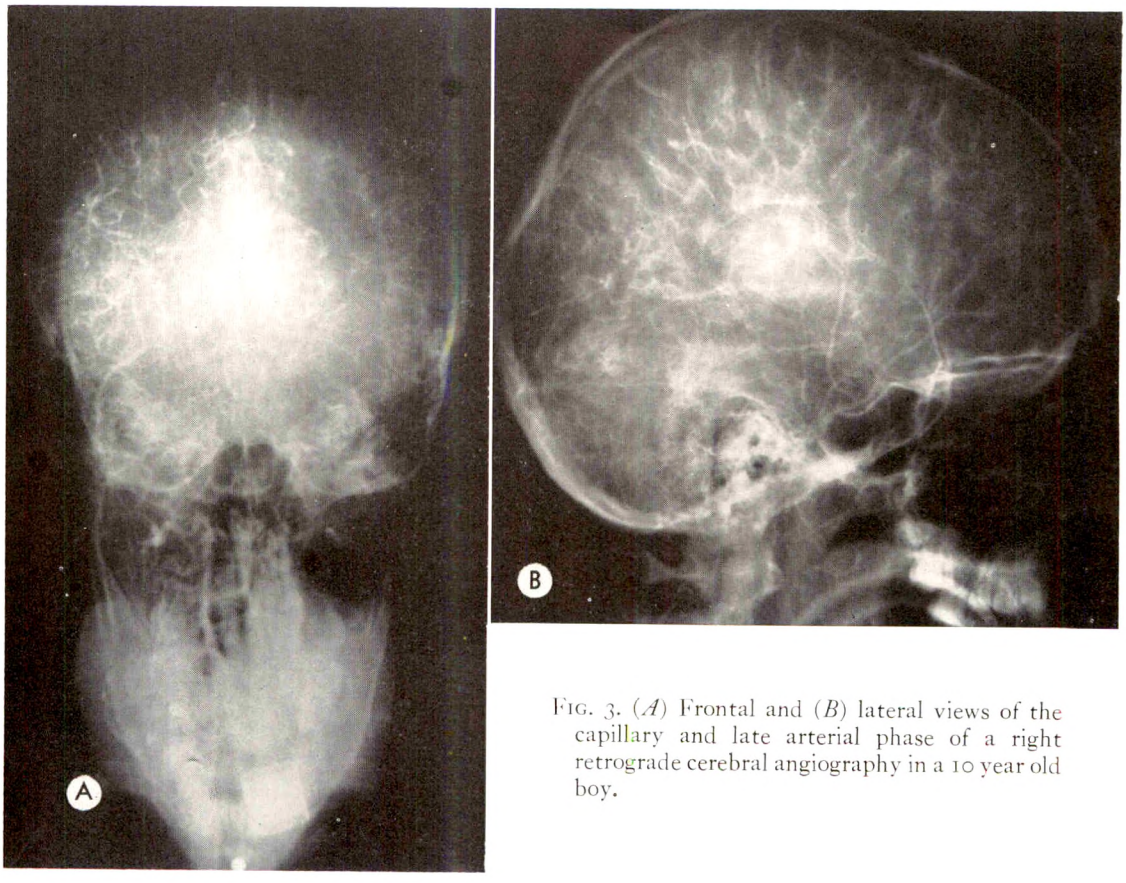



FIG. 3. (*A*) Frontal and (*B*) lateral views of the capillary and late arterial phase of a right retrograde cerebral angiography in a 10 year old boy.

eterization was first introduced by Gensini and Ecker.[4] The application of this method in children with selective opacification of each of the carotid and vertebral arteries was reported by Lurie *et al.*[7] Francisco *et al.*[2] reported their results in 7 children, $3\frac{1}{2}$–12 years of age, using this method and discussed its merits.

Cerebral angiography is now receiving wider acceptance in the field of pediatric radiology. The initial reluctance to use the procedure because of complications in this particular age group has been shown to be unwarranted. Poser and Taveras[12] reported that the complications appear to be less frequent and less severe in children. They studied the morbidity rate judged by temperature elevation, increased pulse rate and fall in blood pressure in children ranging from $5\frac{1}{2}$ months to 13 years of age, following cerebral angiography and pneumoencephalography. The group who had arteriography showed a 20 per cent morbidity which was significantly less than the 49 per cent morbidity rate in the children undergoing pneumoencephalography. Both procedures were performed under general anesthesia. All the complications were transient in nature indicating that cerebral angiography in children is a relatively safe procedure.

It is often necessary to visualize the whole cervicocerebral circulation for adequate diagnosis. If this is done by the direct method, it entails four separate punctures of both carotids and both vertebral arteries. Carotid puncture is more difficult in children than in adults due to the small size of the carotid artery and the shortness of the neck. In addition, it is desirable to avoid direct puncture of a vessel which may be the site of the pathologic condition under investigation, *e.g.*, in cases of acute infantile hemiplegia due to carotid thrombo-

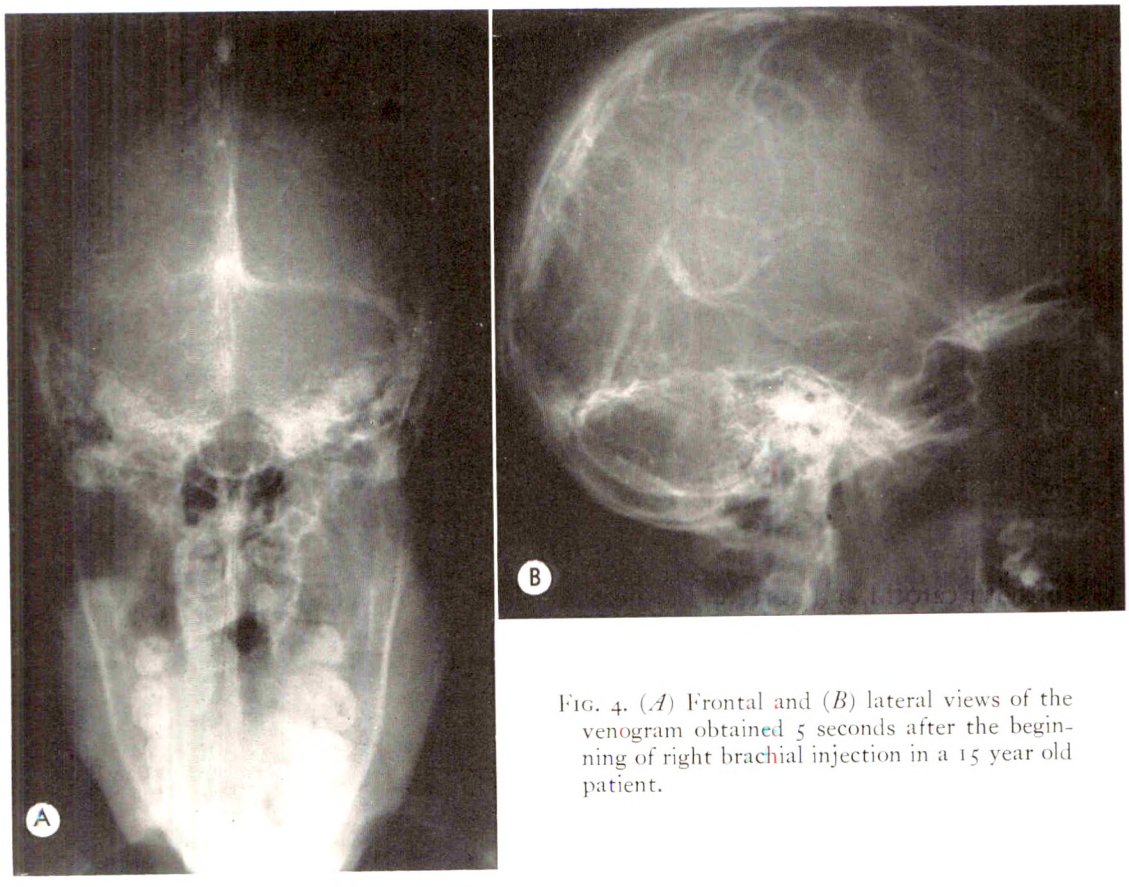



Fig. 4. (*A*) Frontal and (*B*) lateral views of the venogram obtained 5 seconds after the beginning of right brachial injection in a 15 year old patient.

sis.[3] Direct vertebral puncture is much more difficult and is frequently impossible in small children.

### THE MERITS OF THE PROCEDURE

The technique described in this paper was found to fulfill the requirements needed for total visualization of the cervicocranial vessels. The merits of this procedure can be summarized as follows:

I. It is technically simple.
   (a) The percutaneous puncture of the brachial artery at the elbow is much easier than carotid or vertebral artery punctures in children.
   (b) Passage of the small Rochester catheter into the brachial artery is undoubtedly easier than retrograde femoral catheterization. It does not require any preliminary test roentgenogram or screening to check its position and ensures safe injection of the contrast medium into the lumen of the artery. The technique also obviates the danger of subintimal injection or extravasation which may occur following direct injection through percutaneous needles.
   (c) Movement of the head and neck into a satisfactory position for roentgenography can be performed without the risk of dislodging the arterial needle which sometimes occurs with direct punctures in the neck.
   (d) Preliminary cut down and arterial suture is not needed and the procedure is consequently shorter in duration.

II. The method has been shown to be safe with lack of complications apart from

mild soreness and mild extravasation of blood at the elbow which may cause some slight transient discomfort. The absence of major complications is undoubtedly due to avoidance of direct carotid or vertebral puncture and the direct injection of the contrast medium into these arteries. In addition, the procedure does not require the introduction of a catheter into the arch of the aorta which is necessary in the retrograde femoral artery method.

III. There is excellent total opacification of the intracranial and extracranial vessels using this technique. The use of the automatic pressure injector consistently produced good visualization of both carotid and vertebral systems in all cases. The contrast obtained is much superior to that obtained following the bilateral intravenous injection of radiopaque contrast medium for aortography. It is also better than the results of transfemoral aortic catheterization, particularly in opacification of the intracranial circulation.

IV. The visualization of the whole cerebral vasculature has definite advantages:

(a) It obviates the necessity for another procedure to complete the investigation. There is no need to resort to the left temporal artery catheterization to supplement the right brachial as advocated by Oller *et al.*[9]

(b) Many of the conditions in pediatric neurology are either bilateral (*e.g.* bilateral subdural hematomas) or involve the brain as a whole (*e.g.* diffuse cerebral atrophy). In some cases clinical findings are insufficient to allow localization to one area of the brain.

(c) The arterial, capillary and venous phases of cerebral circulation of the brain are well seen in the serial angiograms which permit comparison of the two sides and more accurate diagnosis.

(d) The size of the lateral ventricles can be estimated from the appearance of the anterior and middle cerebral arteries and the deep cerebral veins permitting the diagnosis of ventricular dilatation.

## SUMMARY

The technique of percutaneous retrograde brachial injection using Rochester needles and an automatic pressure injector is described. The evolution of the methods used for cerebral angiography in children has been reviewed. The advantages of the technique recommended here are that it is simple and safe. It also allows total visualization of the extracerebral and intracerebral circulation which has particular advantages when performing cerebral angiography in children.

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# THYROCERVICAL STEAL*

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SUBCLAVIAN artery "steal" resulting from obstruction of the subclavian artery proximal to the vertebral artery origin has been well documented.[1] Classically, cerebral ischemia results as blood, tapped from the contralateral vertebral circulation, is diverted from the brain and flows down the vertebral artery to the subclavian artery beyond its obstruction. The degree of shunt varies with the degree of subclavian artery obstruction and with the oxygen demand of the upper limb. Other combinations of obstruction and shunt involving brachiocephalic vessels have been described.[2]

Recently the authors encountered a patient with bilateral subclavian artery obstruction in whom there was a theft of arterial blood from the head for the upper extremities but the sites of obstruction and the routes of collateral flow were unusual.

## ILLUSTRATIVE CASE

M. O., a 34 year old white female, was well until 1954 when she began to experience episodes of weakness, fatigue and exertional dyspnea. Her hands blanched and became painful when exposed to cold. She complained of easy fatigability of the upper extremities. There was concomitant history of migratory arthritis involving the major joints. Digitalis and steroid therapy provided temporary relief.

In August of 1961, because of further diminution of circulation in the right upper extremity, a subclavian obstruction was removed and an arterial graft inserted. A pseudoaneurysm developed at the graft site which was removed later the same year.

At the time of recent admission, she complained of intermittent severe headaches associated with blurred vision and episodes of dizziness. Fatigability of the upper extremities had progressed to become incapacitating.

*Physical examination.* The patient was an alert, cooperative, white female who looked older than her stated age. Blood pressures were unobtainable in the upper extremities by auscultation. By palpation, blood pressure was 60/0 on the left. There was no palpable pulse on the right. Both hands were pale and cool. The lower extremity blood pressures were 150/70 on the right and 150/60 on the left.

Examination of the eyes revealed slight enophthalmia and minimal arteriosclerotic changes in the retinae. A low-pitched systolic murmur was heard best along the right sternal border. A low-pitched diastolic murmur was heard in the aortic area. The remainder of the examination was non-contributory.

*Laboratory findings.* Routine laboratory procedures as well as L. E. preparations, serology, sedimentation rate, spinal fluid examination and an electroencephalogram were within normal limits. An electrocardiogram was normal except for changes compatible with left ventricular hypertrophy.

*Roentgenologic findings.* The percutaneous transfemoral route was used for arch angiography and selective catheterization of the major aortic arch branches.

Each subclavian artery was obstructed just distal to the origin of the vertebral artery (Fig. 1). The right vertebral artery supplied the

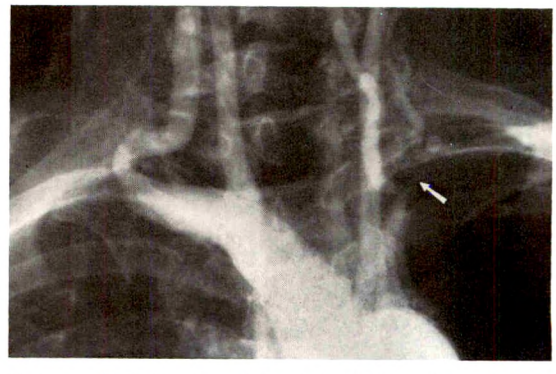


FIG. 1. *Arch injection.* There is obliteration of both subclavian arteries just beyond the vertebral artery orifices. On the left, a line of contrast medium (arrow) delineates a slender extension of the main arterial channel which extends to the thyrocervical trunk.

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basilar artery. The left vertebral artery terminated in the left posterior inferior cerebellar artery. Both vertebral arteries had extensive perispinal collateral branches through which contrast medium poured rapidly into ascending branches of the homolateral thyrocervical trunk.

On the left, a major branch of the thyrocervical trunk (the transverse scapular artery) supplied the upper extremity (Fig. 2). On the right, arterial supply to the upper extremity traversed a network of collateral branches belonging to the thyrocervical and costocervical trunks (Fig. 3). There was no evidence of contrast substance in either subclavian artery beyond the site of obstruction indicating total rather than segmental lumen obliteration.

Bilateral carotid angiograms and the abdominal aortogram were unremarkable.

SUMMARY

A case of bilateral "thyrocervical steal" is presented. The subclavian arteries were totally obliterated beyond the vertebral artery orifices. Arterial blood to the upper extremities flowed via the vertebral arteries, spinal collaterals, homolateral thyrocervical branches in the neck, the thyrocervical trunks *per se* and finally via lateral branches from the trunks to the arms. On the right side, the costocervical trunk participated as well. The carotid angiograms were normal.

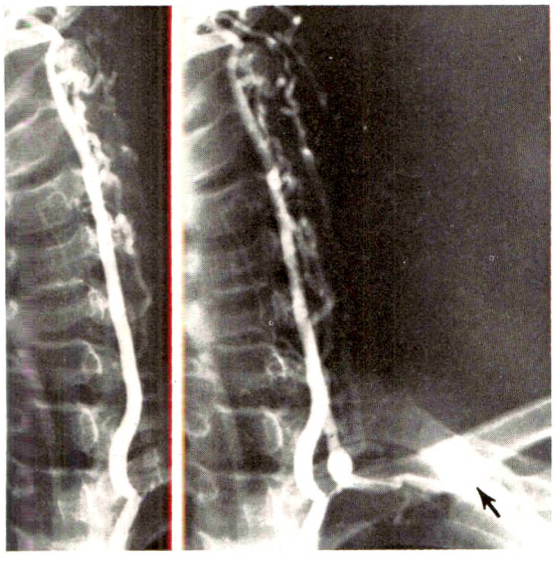


FIG. 2. *Left subclavian injection.* As in Figure 1, there is demonstrated a minute channel connecting the subclavian artery with the thyrocervical trunk. However, the main flow is up the vertebral artery, across spinal collaterals, and down to the thyrocervical trunk. Since the subclavian artery lumen is completely obliterated, the blood then flows to the arm via the transverse scapular branch (arrow) of the thyrocervical trunk.

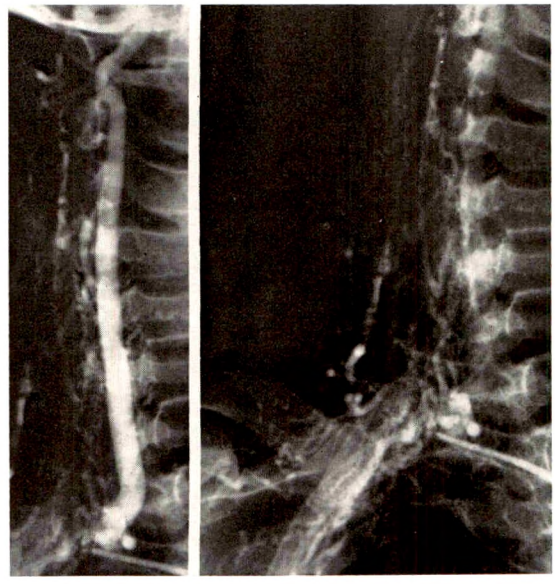


FIG. 3. *Innominate injection.* The findings in the arterial phase are similar to those seen on the left side. The thyrocervical and costocervical systems are both part of the collateral circulation and the channels are more numerous. The trunks *per se* are not well defined.

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# THE DIAGNOSIS OF TUMORS OF THE SPLENIUM OF THE CORPUS CALLOSUM*

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IN THE course of one year the authors had an opportunity to study 5 patients with gliomas of the splenium of the corpus callosum at the Stanford Medical Center. The accurate diagnosis of each case was made only by roentgenographic methods and in each instance was confirmed by autopsy.

## CLINICAL FINDINGS

The age, sex, and clinical chronology of the patients are presented in Table I. In Case II, a previous history of seizures and gradual personality change made it difficult to determine the onset of symptoms. However, an acute change in the patient occurred 2 weeks prior to her admission. In Case IV, memory loss had preceded the sudden onset of symptoms by 2 years; whether due to the patient's tumor or to senile changes is uncertain. In the remaining 3 cases, however, a short interval occurred between the onset of symptoms and the first hospitalization and the subsequent course of the patient was relatively rapidly downhill.

## PRESENTING SYMPTOMS

Headache, nausea, and vomiting were present initially in 3 patients. In 4 patients, including all but the youngest, mental changes were prominent presenting symptoms. These included memory loss, confusion, decrease in intellectual ability and disorientation. In 2 patients there was some unsteadiness of gait and in 1 of these there was some difficulty in seeing to the left. In Case I, the presenting complaints included some weakness of the left leg and some difficulty using the left hand without specific weakness. He also complained of numbness and paresthesias in the left hand.

## INITIAL PHYSICAL FINDINGS

Papilledema was found in 3 patients, 2 of whom had presented with mental changes. Three patients had partial or complete homonomous hemianopia; 1 of them also had a left sixth nerve palsy. A fourth patient had an alternating strabismus with nystagmus. Unilateral pyramidal tract signs were present initially in 3 patients and developed shortly after admission in a fourth. Decreased ability to perform rapid alternating movements was noted in 2 patients. There was decreased joint position sensation in 2 patients and a mild astereognosis in a third. When there were multiple neurologic findings, the findings were referable to one hemisphere except that 1 patient had increased reflexes bilaterally and a second patient had bilateral upgoing toe responses in the presence of otherwise unilateral pyramidal tract abnormalities.

## ELECTROENCEPHALOGRAPHY

Three patients had electroencephalography with skin electrodes. In 1 patient, bilateral frontal and temporal rhythmic delta bursts were interpreted as suggestive of a midline lesion. The records in the other 2 patients were diffusely abnormal with delta slowing and sharp waves in the left frontal region in one of them.

## AUTOPSY FINDINGS

In each case, a massive infiltrating tumor was found centered in the splenium of the corpus callosum (Fig. 1 and 2). The lesions extended forward into the septum pellucidum and laterally into the hemispheres. Occasionally extension to one or the other side was more prominent, though in no case was the extension unilateral. Microscopic examination revealed a glio-

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TABLE I

SUMMARY OF CASES

| Case No. | Age (yr.) | Sex | Onset of Symptoms to First Admission | Cerebrospinal Fluid | Radiation Therapy | First Admission to Death |
|---|---|---|---|---|---|---|
| I | 17 | M | 6 weeks | | 5,600 rads in 5 weeks | 7 months |
| II | 43 | F | 6 years—seizures<br>4 years—gradual personality changes<br>2 weeks—sudden mental change | 78 mg. per cent protein, otherwise normal | | 3 months |
| III | 63 | F | 4 months | | 1,750 rads in $2\frac{1}{2}$ weeks | 5 weeks |
| IV | 70 | M | 2 years—gradual memory loss<br>2 months—sudden mental change | | | 3 weeks |
| V | 30 | F | 1 month | 75 mg. per cent protein, otherwise normal | | 2 days |

blastoma multiforme in each case; in Case 1, on a background of a pilocytic astrocytoma.

## ANALYSIS OF RADIOLOGIC FINDINGS

### SKULL ROENTGENOGRAPHY

In 1 of our 5 cases there was atrophy of the sella turcica, indicating increased intracranial pressure (Case v). Otherwise, the routine skull roentgenograms were normal.

Previous reports have indicated that roentgenographic evidence of increased intracranial pressure was uncommon in corpus callosum tumors.[4] Other findings on skull roentgenograms that have been mentioned in the literature but were not seen in our cases were downward and forward displace-



FIG. 1. Case IV. Coronal brain section at the level of the splenium of the corpus callosum showing enlargement and complete replacement of this structure by neoplasm.

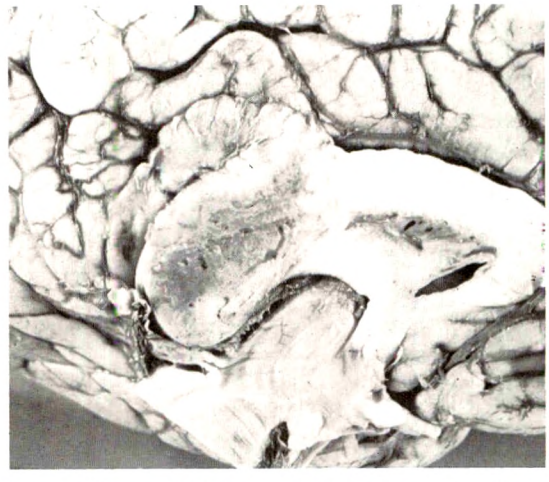


FIG. 2. Case V. Midline sagittal brain section showing glioblastoma multiforme replacing an enlarged splenium of the corpus callosum and extending into the mesial inferior parietal cortex.

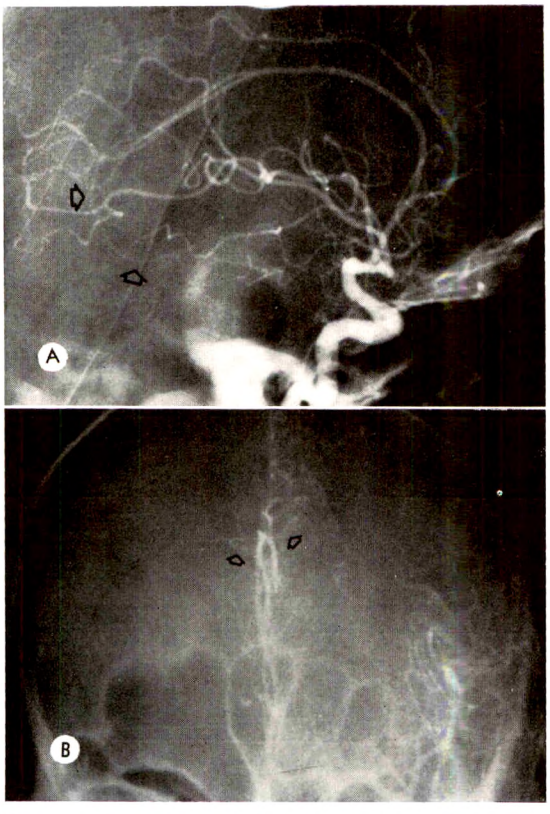



FIG. 3. Case IV. (*A*) Lateral projection, arterial phase, right carotid arteriogram. The anterior cerebral artery shows a smooth bowing suggesting enlargement of the ventricles. Extending from the distal portion of the pericallosal artery, a vessel, indicated by the 2 arrows, forms a wide curve, convex posteriorly. This vessel is the posterior pericallosal artery and outlines the enlarged splenium of the corpus callosum. Compare with Figure 2. The middle cerebral vessels are crowded anteriorly, a sign of a posterior mass. (*B*) Anteroposterior projection, arterial phase, left carotid arteriogram. There is a slight distal shift of the pericallosal arteries toward the right, indicated by the step-like deformity as the vessels cross beneath the falx. The arrows indicate the plexus of vessels on the superior surface of the corpus callosum. The left side is distinctly elevated when compared with the right. This finding, in conjunction with the shift, indicated asymmetric extension of the tumor into the left hemisphere.

ment of the pineal gland, or displacement of a calcified glomus of the choroid plexus.[20]

### ARTERIOGRAPHY

All 5 patients had carotid arteriography. In general, the vessel displacement seen in the arterial phase was nonspecific. In only 2 of our cases was there any midline shift of the arteries, and this consisted of a slight shift in the distal portion of the pericallosal artery (Fig. 3*B*). In 2 patients the sweep of the pericallosal artery was widened suggesting hydrocephalus (Fig. 3*A*). A ventriculogram in one of these patients and postmortem study in both revealed that the ventricles were relatively small. The bowing of the anterior cerebral vessels may have been due to forward displacement of brain by the huge mass rather than hydrocephalus. Minor irregularities in the course of the pericallosal vessels as seen in the lateral projection were not of diagnostic value since there is a great degree of normal variation.[24] In Case IV, elevation of the left side of the plexus on the dorsal surface of the corpus callosum,[12] as well as a slight distal shift of the pericallosal artery, indicated greater extension of the tumor into one hemisphere (Fig. 3*B*). In one patient (Case V) downward displacement of the posterior cerebral artery revealed tentorial herniation of the temporal lobe.

In 3 patients specific findings were present in the arterial phase. In 2, this consisted of a tumor stain supplied from the posterior portion of the pericallosal artery and located in the region of the splenium (Fig. 4*A*; and 5*A*). In one of these, early filling of draining veins was seen in the late arterial phase. The stain was seen in only a small portion of the tumor, indicating that these tumors were predominantly avascular roentgenographically. Other reported cases have shown a similar relatively small area of abnormal vessels.[23,28] In one other patient visualization of a branch which originated from the posterior portion of the pericallosal artery and swept around an enlarged splenium of the corpus callosum enabled a specific diagnosis to be made (Fig. 3*A*). This artery has been called either the posterior pericallosal artery,[9,10,25] or the artery of the splenium.[17] It usually arises from the posterior cerebral artery and may function as an anastomotic channel between the anterior cerebral and posterior cerebral vessels.

The anteroposterior projection showed only a slight shift of the midline deep veins in 2 of our 5 cases; in the other 3 they were midline. While this in itself was not specific, it was important in indicating the midline nature of the tumor. The lateral venous phase was, by far, the most important clue to the diagnosis as has been noted in the past.[6,13,14,19,23,25,26] The arc of the great vein of Galen was widened and the descending portion of the internal cerebral vein was focally flattened or concave so that it continued the curve of the vein of Galen (Fig.

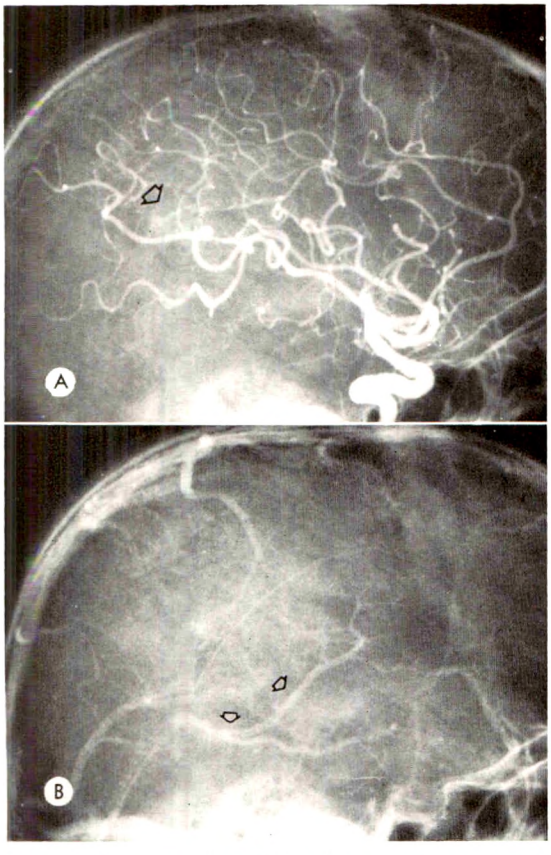



FIG. 4. Case III. (*A*) Lateral projection, arterial phase, right carotid arteriogram. The configuration of the vessels is normal. The arrow indicates a cluster of tumor vessels supplied by the pericallosal artery. This blush is in the posterior portion of the corpus callosum, just inferior to the pericallosal artery. (*B*) Lateral projection, late venous phase, right carotid arteriogram. Posterior portion of the internal cerebral vein (upper arrow) is flattened and displaced anteriorly and inferiorly. The arc of the vein of Galen (lower arrow) is widened forming a continuous curve with the internal cerebral vein. The septal veins and anterior ependymal veins can be seen quite clearly and do not indicate significant ventricular enlargement. The inferior sagittal sinus is seen as a thin line parallel to, but posterior and superior to, the internal cerebral vein.

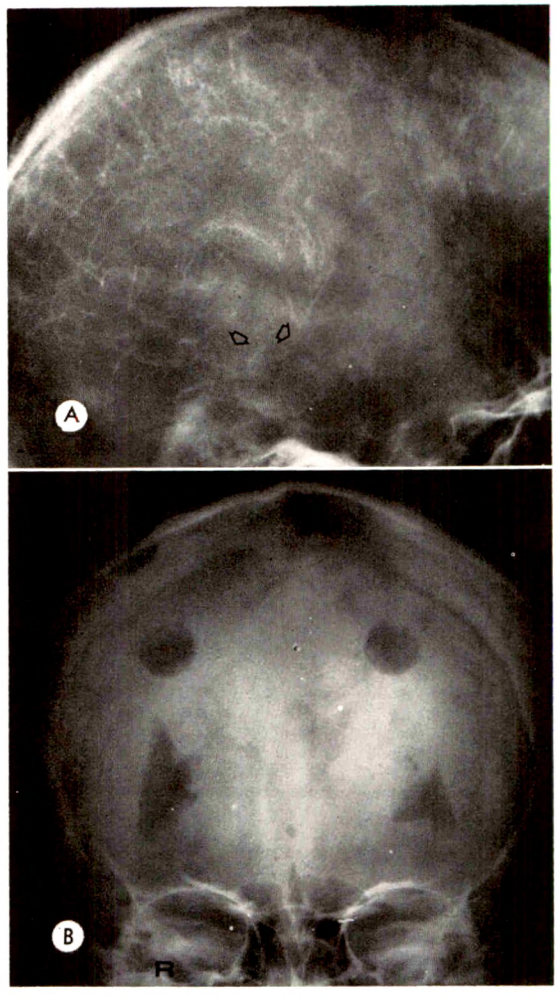



FIG. 5. Case V. (*A*) Lateral projection, late arterial phase, right carotid arteriogram. A fine arcuate blush of tumor vessels can be seen in the central portion of the head. These drain by a fan-like group of veins into the vein of Galen which is indicated by the two arrows. The arc of the vein of Galen is widened. (*B*) Posteroanterior projection, brow-down position, ventriculogram. Two round parietal burr holes are seen just above the center of the roentgenogram and one frontal burr hole high on the right side. The triangular structures below the burr holes represent the atria of the ventricles widely separated by the midline mass. The mass bulges into the superior medial surface of both atria producing a concave deformity. Compare with Figure 1.

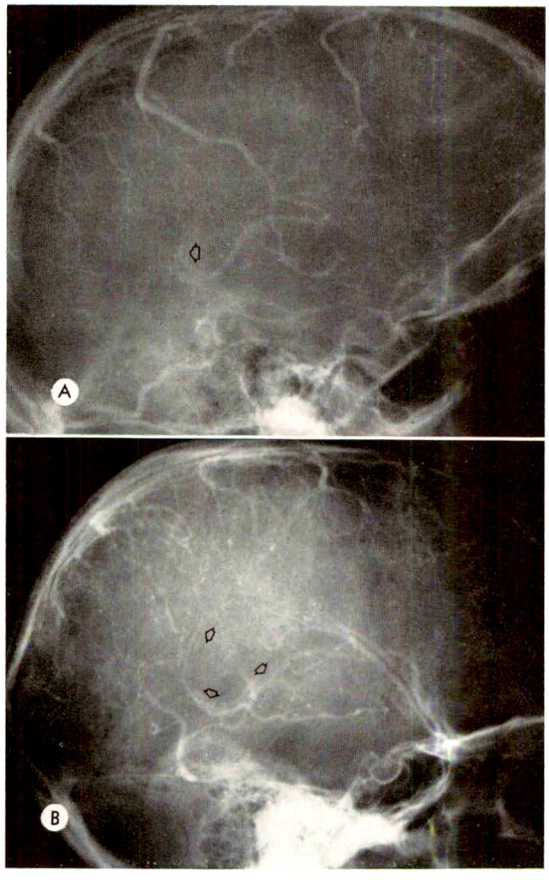



FIG. 6. (*A*) Normal deep veins in lateral projection. The arrow indicates the posterior callosal vein. The curve formed by this vein, the vein of Galen, and the posterior descending portion of the internal cerebral vein indicates the size of the splenium of the corpus callosum. (*B*) Case 1. Lateral projection, late venous phase, right carotid arteriogram. The posterior portion of the internal cerebral vein is slightly flattened and displaced anteriorly and inferiorly. It forms a wide curve with the vein of Galen (lowest arrow), which also has a widened arc. The posterior callosal vein (uppermost arrow) drains around the splenium into the vein of Galen. This vessel marks the upper border of the corpus callosum. The arc made by it, the vein of Galen, and the internal cerebral vein, indicates the enlarged splenium.

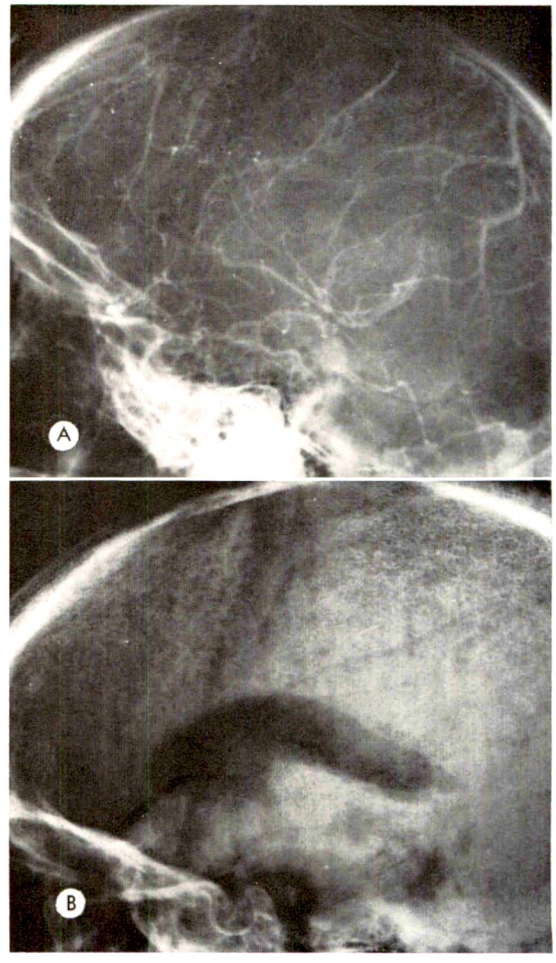



FIG. 7. Case II. (*A*) Lateral projection, late venous phase, left carotid arteriogram. The internal cerebral vein is flattened and displaced anteriorly and inferiorly. There is a focal concave deformity in its posterior portion. The arc of the vein of Galen is increased, forming, with the posterior portion of the internal cerebral vein, a grossly enlarged curve. The septal veins and anterior subependymal veins are seen through the overlying cortical vessels and do not indicate significant enlargement of the lateral ventricle. The subependymal veins in the region of the atrium appear distinctly depressed in conjunction with the widened curve of the deep midline veins. This depression corresponds with the mass seen in the pneumoencephalogram. (*B*) Lateral projection, brow-up position, lumbar pneumoencephalogram. The roof of each ventricle is slightly flattened in the parietal region and the hemispherical defect which can be seen in the roofs was shown to be in the midline in the anteroposterior projection. This focal deformity produced by the tumor of the corpus callosum corresponds well with the depression of the atrial veins seen in the venous phase of the arteriogram.

4*B*; 6*B*; and 7*A*). An angle may form in the midportion of the internal cerebral vein where the vein returns to its normal course (Fig. 6*B*; and 7*A*). This configuration has been termed "ballooning" or the "cradle" deformity of the deep veins of the brain.[19,26]

The vein of Galen may not accurately de-

line the curve of the splenium of the corpus callosum particularly when it enters into the straight sinus at either a right angle or an obtuse angle.[16] Similarly, the distance between the internal cerebral vein and the inferior sagittal sinus may not be an indication of the thickness of the corpus callosum as the position of the inferior sagittal sinus varies with the height of the falx.[13] In the venous phase, the splenium can be accurately defined and enlargement detected when the posterior callosal vein (also called the posterior cerebral vein) is visualized (Fig. 6*A*). This vein originates on the superior-posterior surface of the corpus callosum and passes around the splenium to enter into the vein of Galen.[13,16,28]

In one of our patients (Fig. 7*A*), the subependymal veins draining the medial wall of the atrium were depressed which indicated a mass protruding into the medial portion of the atrium.[27]

Vertebral arteriograms were not obtained in our patients. Since the vascular supply to the posterior corpus callosum is usually derived from the branches of the basilar artery, they might have provided additional information. The posterior pericallosal artery can be visualized in more than 60 per cent of the vertebral arteriograms.[10] The posterior callosal vein should also be visualized in a majority of examinations.

### PNEUMOGRAPHY

Tumors of the splenium of the corpus callosum share some features with tumors of the corpus callosum generally. These include separation and asymmetric distortion of the lateral ventricles and the presence of a sharp defect in the dorsal margin of one or both lateral ventricles.[5,25] More specifically, lesions in the splenium widen the distance between the medial walls of the atria of the lateral ventricles and may cause a slight depression of the posterior portion of the third ventricle with flattening of the aqueduct.[20] Since the ventricles normally deviate laterally as they form the atrium, early separation may be difficult to appreciate.

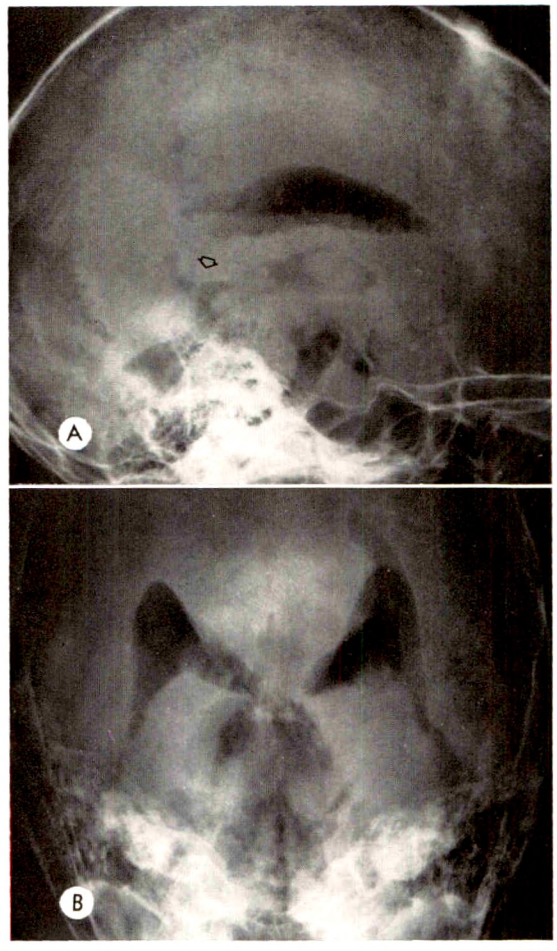



FIG. 8. Case III. (*A*) Lateral projection, erect position, lumbar pneumoencephalogram. The arrow indicates a mass bulging downward into the cistern of the quadrigeminal plate. The quadrigeminal plate, aqueduct, and possibly the posterior third ventricle are displaced anteriorly and inferiorly. A faint suggestion of a mass indenting the medial portion of the roof of each atrium can be seen. (*B*) Half axial projection, brow-down position, lumbar pneumoencephalogram. A mass produces a concave deformity in the medial wall of both atria which are displaced laterally.

Four of our 5 patients had pneumography, 2 by lumbar encephalography and 2 by ventriculography. All 4 patients showed the characteristic separation of the atria of the lateral ventricles in the anteroposterior projection (Fig. 5*B*; and 8*B*: compare with Figure 1). In one (Case V) the separation was extreme, resulting in marked narrowing of this portion of the ventricles. Three of the 4 patients showed a focal depression convex downward in the

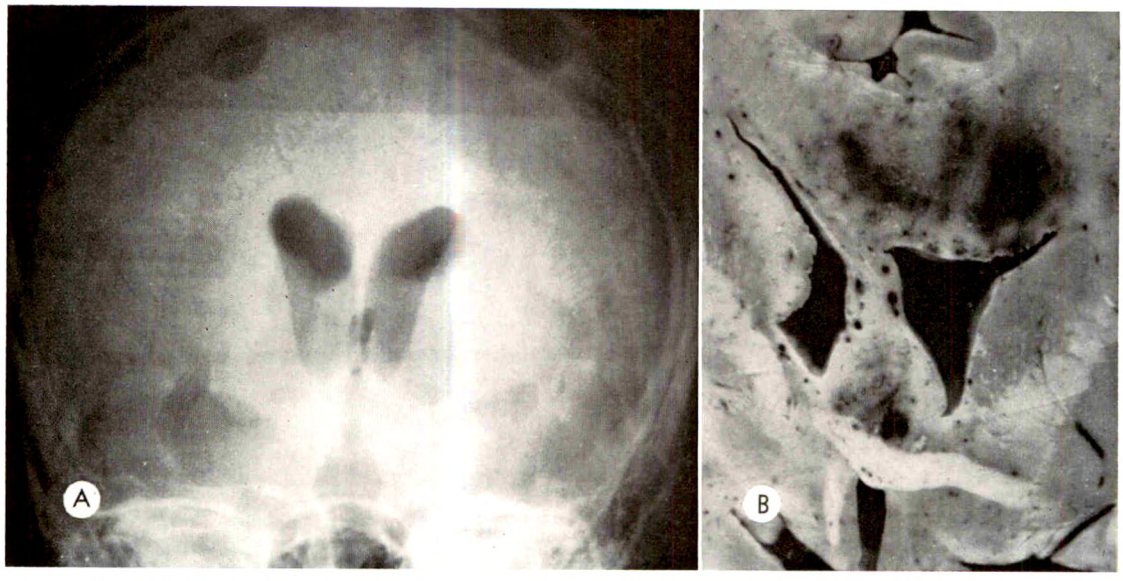



FIG. 9. Case 1. (*A*) Anteroposterior projection, brow-up position, ventriculogram. The septum pellucidum is widened. The thickening is somewhat asymmetric and irregular and extends more to the right side. There is slight irregular scalloping of the lateral margin of the right anterior horn. This was subsequently shown to be tumor extending forward along the lateral margin of the ventricle. (*B*) Coronal brain section (7 months later) at level of anterior commissure showing forward extension of glioma within the corpus callosum, invasion and thickening of the septum pellucidum, and extension of neoplasm into the lateral wall of the right anterior horn.

roof of the posterior portion of the ventricles in the lateral projection (Fig. 7*B*; and 8*A*). Erect roentgenograms were available on 1 of the 2 patients studied by lumbar encephalography. These demonstrated a mass protruding from above into the cistern of the quadrigeminal plate (Fig. 8*A*). Associated with this was flattening of the curve of the aqueduct.

All 4 patients had a widened septum pellucidum. In 1 patient gross asymmetric thickening of the posterior portion of the septum pellucidum was seen in the brow-down projection. In the brow-up projection, irregular asymmetric widening was seen in this patient and in two others (Fig. 9, *A* and *B*). This widening took the form of an inverted triangle with the base towards the vertex. Irregular widening of the septum pellucidum of the type seen in these patients is diagnostic of a malignant infiltrating tumor.[15,22] This finding clearly differentiates an infiltrating tumor of the brain from an extracerebral mass such as a falx or tentorial meningioma which may mimic some of the pneumographic signs of a tumor of the splenium. In one reported series, roentgenographic widening of the septum pellucidum occurred in 28 of 181 cases of gliomas and 9 of these were in tumors originating in the corpus callosum.[22] In another report widening was found in 18 out of 36 tumors of the corpus callosum.[24] In the fourth patient (Case v) a smooth widening of the septum pellucidum with parallel walls was visualized in the brow-up projection which was indistinguishable from a non-communicating cyst. There was insufficient air in the ventricles in the brow-down projection to visualize the posterior septum pellucidum. Such a smooth-bordered widening of the septum, indistinguishable from a non-communicating cyst, has been reported to be due to tumor invasion.[15,22]

The ventricles were not enlarged in the 4 patients. In 1 patient (Case v) the large size of the tumor prevented insertion of a ventricular cannula through posterior parietal burr holes. Preliminary evidence of a posterior midline lesion should discourage attempts at posterior ventriculography.[20]




Fig. 10. Case 11. Anterior (*A*) and right lateral (*B*) photoscans of the head following intravenous injection of chlormerodrin $Hg^{203}$. The solid arrow in (*B*) indicates the anterior direction. An area of abnormal uptake is seen in the center of the head which corresponds to the area of the splenium of the corpus callosum. Compare with Figure 7, *A* and *B*.

### Gammaencephalography* (Brain Scanning)

Four of our 5 patients had brain scanning as part of their initial diagnostic evaluation. Three were examined using chlormerodrin $Hg^{203}$ and 1 with technetium 99m pertechnetate. Three (Cases 11, 111 and 1v) were found to have an abnormal area of isotope concentration in the posterior midline region appropriate for a lesion of the splenium (Fig. 10, *A* and *B*). In 1 case in which both an anterior and a posterior scan were obtained, the lesion was better visualized on the posterior scan. The fourth patient (Case 1) had been studied with chlormerodrin $Hg^{203}$. The initial scan was interpreted as showing an equivocal area of uptake in the right posterior temporal region. Evidence obtained several months later indicated that the tumor extended into this region, suggesting that the original scan might have been a true positive. The scan, however, had failed to identify the primary focus of the lesion. A second scan of this patient using technetium 99m pertechnetate was made 7 months later. Despite intervening radiotherapy and a reduction in the size of the posterior portion of the tumor, the technetium 99m scan was positive for a posterior midline lesion, suggesting that if the original scan had been made with this material it might have been positive. The number of our cases does not allow a prediction of the success rate of gammaencephalography in detecting gliomas of the splenium. The finding of 3, and possibly 4, positive scans in a lesion generally considered difficult to diagnose is quite encouraging. This finding is in keeping with the expectation that the more invasive gliomas, which are the usual lesions found in the splenium, will yield a high percentage of positive brain scans.[11,18]

* All of the scanning procedures were performed in the Radioisotope Laboratory under the supervision of Dr. Joseph Kriss.

### Differential Diagnosis

In the majority of cases the diagnosis of a posterior corpus callosum tumor can be established by carotid arteriography. In some cases a tumor blush will be visualized in the appropriate location. In the absence of such a stain, the appearance of the typical ballooning or cradle deformity of the deep veins of the brain in the absence of significant degree of midline shift of the arteries or deep veins is quite diagnostic.

An appearance of a widened curve of the vein of Galen with flattening of the internal cerebral vein but without focal deformity must be interpreted with caution. Such an appearance can be a normal variant in a dolichocephalic skull with a narrow falx

cerebri.[16] This may be suggested by an unusually steep course to the straight sinus and a high position of the inferior sagittal sinus. A similar appearance can result from posterior falx and midline tentorial meningiomas and from severe hydrocephalus. In the absence of distinguishing arterial displacements the extracerebral lesions can be differentiated by visualization of the posterior pericallosal artery or vein. If these vessels are not seen on carotid arteriography, a vertebral study would be indicated. If these vessels show a widened curve of the splenium, then the differentiation from marked hydrocephalus is the only one of importance. If other findings did not indicate hydrocephalus, the diagnosis of a tumor of the splenium would be established. If, however, there is evidence of hydrocephalus, supplemental information is necessary to make the diagnosis. In most cases a positive gammaencephalogram when correlated with the arteriographic findings will be sufficient. If not, an air study will be necessary. Under these circumstances, *i.e.*, the possibility of marked hydrocephalus, a ventriculogram would be most suitable. This should logically be approached via frontal burr holes as has already been discussed. The findings should lead to the correct diagnosis.

When an air study is the first examination, the differential diagnosis of a tumor of the splenium will include: (a) false appearance of separation of the atria due to inadequate filling; (b) pinealoma; (c) aneurysm of the vein of Galen; (d) meningioma of the posterior falx or the midline tentorium; and (e) a superior vermis tumor which has extended upward through the tentorial incisura.[20,23] These lesions can often be distinguished by their slightly different location and direction of growth. Evidence of tumor invasion into the septum pellucidum permits an accurate diagnosis of an infiltrating glioma. Often angiography will be necessary to complete the diagnosis.

Gliomas of the occipital or parietal region will often involve the posterior corpus callosum and may extend across to the opposite hemisphere. These lesions will not be a diagnostic problem on either air study or an arteriogram as they produce a greater degree of lateral shift of the cerebral structures.

A positive gammaencephalogram may be the initial roentgenologic study. In that case, an arteriogram would still be indicated to distinguish a deep midline meningioma from a glioma of the splenium as both of these lesions might produce the same distribution of isotope uptake. The distinction between a glioma of the splenium and a glioma of one hemisphere invading the splenium is of less clinical importance.[11]

We have encountered 1 case in which a benign cyst subsequent to a hematoma in the parietal parasagittal area extended into the posterior corpus callosum. The roentgenographic features on carotid arteriograms and pneumoencephalograms were indistinguishable from those of an infiltrating tumor of the splenium but there were no tumor vessels and only equivocal widening of the septum pellucidum. A gammaencephalogram was negative. The onset of the patient's disease with a subarachnoid hemorrhage and the benign course for a year were distinctly different from those of the patients described above. The diagnosis of a benign cyst was made at biopsy, and the extent of the cyst was demonstrated by injecting it with air.

## DISCUSSION

In 1884, Bristowe[3] described 4 cases of corpus callosum tumor and attempted to correlate their signs and symptoms with the gross findings at autopsy. The characteristic features were as follows: (1) a gradual progressive worsening of the patient's condition; (2) a progressive hemiparesis with vague motor symptoms on the contralateral side; (3) mental symptoms, characterized by apathy, extreme drowsiness, a puzzled inquiring look, difficulty in swallowing, and cessation of speech; (4) the absence of cranial nerve palsies; and (5) the slightness of headache and vomiting. Bristowe considered it unlikely that this clinical state was due to direct involvement of

the corpus callosum but thought that the signs and symptoms were produced by increased intracranial pressure and invasion of the neoplasm into the adjacent portion of each cerebral hemisphere. He felt that aphasia could be produced by extension to appropriate areas of the dominant hemisphere and that seizures might also occur.

Akelaitis,[1] 25 years ago, demonstrated that in man the corpus callosum could be completely sectioned without producing any specific dysfunction in praxis, gnosis, language and orientation.

The experience of clinicians has been disappointing in establishing with reasonable certainty the diagnosis of a corpus callosum tumor on the basis only of signs and symptoms.[7,8,21] Many of the patients had headache as their only prominent complaint and showed evidence of papilledema. A significant number of cases had abnormalities in the cerebrospinal fluid, consisting of pleocytosis and increased protein content. Left-handed apraxia, especially in the presence of a right hemiplegia, was accepted as a valid sign of such neoplasms, although there was considerable variation in its reported incidence.[2]

At the present time, the clinician may suspect the presence of a neoplasm in the corpus callosum on the basis of a change in mental state, signs of bilateral pyramidal disease, apraxia of the left hand, and difficulty in walking, especially if these signs and symptoms are accompanied by evidence of increased intracranial pressure. This complete constellation, however, was not recognized in our 5 patients. An electroencephalogram showing evidence of a bilateral projected type of slow wave disturbance would provide added evidence of a deep-seated midline expanding lesion. Such a clinical picture, however, does not provide an accurate diagnosis on which one may plan surgical intervention or any other form of therapy. It has only been with the development of air contrast studies, arteriography, and radioisotope techniques that an accurate diagnosis of neoplasms of the corpus callosum has been reached.

Schlesinger[20] observed that pneumography permitted identification of a large portion of the tumor but usually failed to demonstrate the extent of the tumor into adjacent structures. He suggested that arteriography might provide more information about the extent of the lesion. Unfortunately, our experience indicates that while the origin of the tumor can be readily identified on arteriography, the extent of the lesion is not delineated.

## SUMMARY

1. Primary neoplasms of the posterior corpus callosum are usually malignant gliomas which typically show extensive invasion anteriorly into the corpus callosum, fornix, and septum pellucidum and laterally into the centrum semiovale and mesial parietal cortex of each cerebral hemisphere.

2. An accurate diagnosis of such tumors cannot be made on clinical grounds alone.

3. On carotid arteriography the appearance in the lateral projection of the typical ballooning or cradle deformity of the deep veins of the brain in the absence of any significant degree of midline shift of the arteries or deep veins is diagnostic of a mass lesion involving the splenium of the corpus callosum. When this characteristic deformity is not seen, visualization of the posterior callosal vein or of the posterior pericallosal artery may define an enlarged splenium. The presence of a tumor stain located in the appropriate region is diagnostic but is not a constant finding. Vertebral arteriography may be useful to supplement carotid arteriography. In some cases differentiation from marked hydrocephalus may require an air study.

4. Air studies may reveal increased separation of the atria of the lateral ventricles, focal deformation of the medial aspect or roof of one or both ventricles, downward displacement of the posterior third ventricle, encroachment into the cistern of the quadrigeminal plate, and a most important differential point, thickening of the septum pellucidum.

5. Gammaencephalograms were positive in 3 out of 4 cases. They are a valuable diagnostic aid but must be supplemented

by other procedures to provide accurate anatomic localization.

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Dr. James B. Golden kindly allowed us to include Case III in this report. The roentgenographic studies on that patient were performed by Dr. Howard Jones.

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# VENTRICULOGRAPHY BY DIRECT CATHETERIZATION OF THIRD VENTRICLE USING A WATER SOLUBLE SUBSTANCE

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THE purpose of the authors is to describe a method of selective visualization of the third ventricle, aqueduct and fourth ventricle, utilizing densopax (sodium salt of 3-acetylamine 2, 4, 6-triiodobenzoic acid and polyvinylpyrrolidone).* Densopax is an innocuous opaque substance, which offers high diagnostic accuracy and has proved to be nonirritating. Further advantages are its water solubility and complete absorption in approximately 15 minutes. It was first used by one of us (P.J.B.) in a patient suspected of having a cerebellopontine angle tumor.

## HISTORIC REVIEW

Iodoventriculography is one of the more controversial "contrast" radiologic procedures used in neurologic examinations. In Argentina,[1,3,5,10,19,27] Europe,[12] and other areas,[18–22,25] this procedure has been well accepted and found to be innocuous. Pardal and Pardal[27] reported 1,280 iodoventriculographies performed without risk in the Neurosurgical Department of the University of Buenos Aires in Argentina. Garland[18] stated that lipiodol may be injected safely into the subarachnoid space, as did Schüller,[32] who in 1936 investigated patients with possible tumors at the base of the brain by suboccipital or intraspinal injection of 40 per cent iodipin. However, many articles appear in the literature in which the deleterious effects of iodipin on the meninges are discussed.[11,13,16,20,21,22,24,25] Ebaugh and Mella,[13] in 1926, reported a transient aseptic meningitis following intracisternal injection of iodized oil in 13 cases. Lindblom,[21] in 1931, made a critical investigation of the effects of various iodized oils on the meninges. He found that the degree of irritation caused by the oil varied with the amount of free fatty acid being transformed into iodic fatty acid. In experimental work on animals, he found fatty granuloma in the subarachnoid cavity and pachymeningitis reactions. Fumarola and Enderle[16] reported 1 fatal case following the cisternal injection of lipiodol. Lysholm,[22] in 1937, reported that the use of lipiodol as suggested by Balado,[8] in 1928, was undesirable because of very definite irritating properties, especially on the ependyma. Furthermore, the high viscosity of the preparation occasionally caused the patient's condition to deteriorate. Marcovich *et al.*[25] reviewed the action of lipiodol on the leptomeninges and considered it to have irritating effects. In 1931, Arnell and Lidström[5] reported the first case in which abrodil was employed. Later Arnell[6] summarized his experiences. While acknowledging the merits of abrodil (good opacification, nonirritating effects, optimum absorption), the author also reported various complications following injection. Thorotrast (thorium dioxide), used for the first time by Antoni[4] in 1932, was not recommended because it was suspected to be a carcinogenic agent. Alexander *et al.*,[2] in 1934, Reeves and Stuck,[31] in 1938, and Hughes,[20] in 1953, also reported several histologic reactions of the leptomeninges when using thorium dioxide. In 1950[9] and 1959[30] pantopaque (ethyliodophenylundecylate) was used for ventriculography, apparently without irritating effects. However, Erickson and van Baaren[14] later described meningeal reaction to pantopaque,

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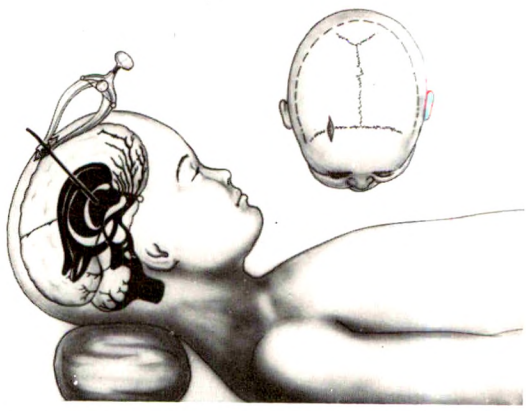


FIG. 1. Artist's drawing of central densopax midline ventriculography.

that terminated fatally with adhesive arachnoiditis of the basilar cisterns. Tarlov[33] reported a case of pantopaque meningitis discovered at operation. Reaction to pantopaque has also been demonstrated by the studies of Peacher and Robertson.[28]

## TECHNIQUE

The technique of ventriculography using densopax is similar to that employed by Azambuja *et al.*[7] and has been previously published by one of us.[34] With the patient supine and the head elevated and turned slightly to the left, under local anesthesia, a burr hole is made on the right side of the coronal suture, 2.5 cm. from the midline (Fig. 1). A ventricular needle is introduced into the frontal horn, and then is replaced by a fine, soft rubber catheter, which is inserted through the tract of this needle. After reaching the frontal horn, its insertion is continued until it touches the floor of the ventricle, causing it to naturally slide posteriorly and medially, following the curvature of the head of the caudate nucleus until it enters the foramen of Monro, stopping at the posterior inferior recess of the third ventricle. Lateral roentgenograms are made to determine the position of the tip of the catheter, which should lie between vertical lines drawn from the anterior clinoid process and from the external auditory canal in the lateral view (Fig. 6), and in the midline area in the frontal view (Fig. 7). The catheter is left in place after closing the skin and galea and 2 cc. of densopax is injected. One lateral and two frontal roentgenograms are made. Visualization of the posterior part of the third ventricle, aqueduct and the fourth

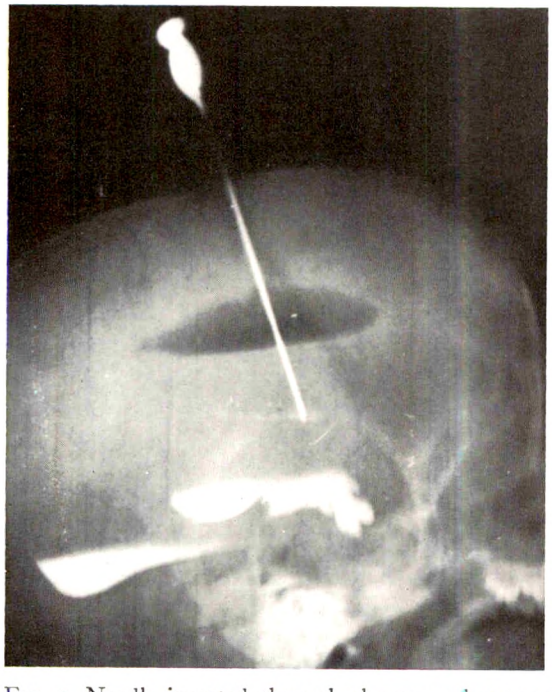


FIG. 2. Needle inserted through the coronal suture into the third ventricle. Complete aqueductal occlusion is demonstrated.

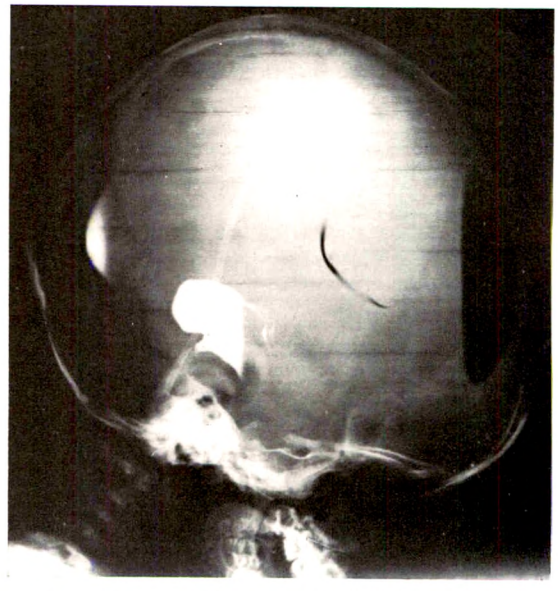


Fig. 3. Case 1. Dilatation of third ventricle and aqueductal occlusion are demonstrated.

ventricle is obtained (Fig. 4, 6 and 9). In hydrocephalic patients, 1 or 2 cc. of densopax is injected, using a No. 18 needle, through the coronal suture line into the third ventricle in conjunction with air (Fig. 2).

Using this procedure, we have been able to fill the caudal ventricular system in almost every instance. With this technique we find it unnecessary to employ fluoroscopy or serial roentgenography in order to follow the course of the contrast material through the ventricular system, as described by other authors. Illustrative case material is presented below.

## ILLUSTRATIVE CASES

Case 1. The parents of this patient gave "large head" as the chief complaint on admittance. No vomiting or convulsion was noted. Examination showed an obviously enlarged head and a bulging, tense and enlarged anterior fontanel. A central air and densopax ventriculogram demonstrated dilatation of the lateral and third ventricle with aqueduct stenosis (Fig. 3).

Case 11. L.B., a 4 year old child, was admitted to the Children's Hospital on July 29, 1965, because of headache, increasing fatigue and poor appetite. The birth and family histories were normal. Neurologic examination showed a well developed, well nourished child with second degree bilateral papilledema, impaired coordination and ataxic gait on a wide base. Laboratory studies including blood cell counts, blood Wassermann test, and a chest roentgenogram were normal. Central air and densopax ventriculography, performed 4 days prior to admission, revealed a lateral shift of a dilated aqueduct. There was good visualization of the fourth ventricle (Fig. 4). Suboccipital craniectomy under endotracheal anesthesia revealed a mass arising from the right cerebellum. Microscopic examination showed astrocytoma. After a satisfactory convalescence, therapy was instituted.

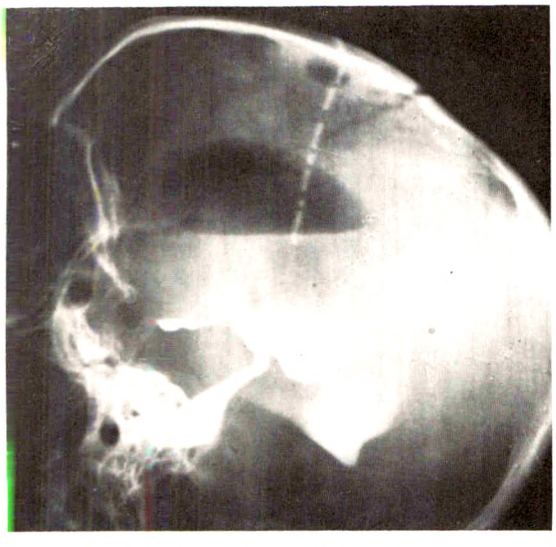


Fig. 4. Case 11. Lateral central air densopax ventriculogram showing good visualization of the third ventricle, aqueduct and fourth ventricle. Note air in dilated frontal horn.

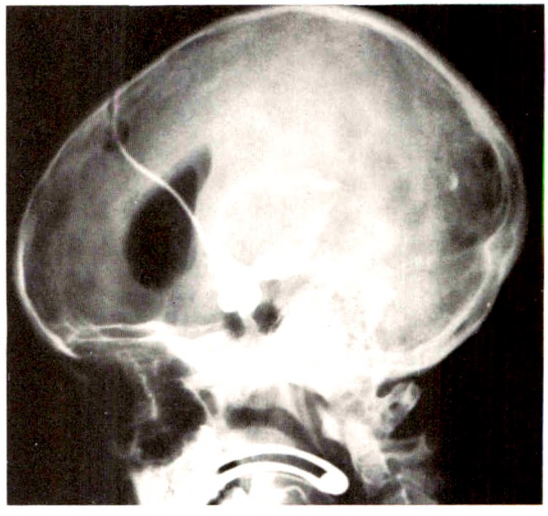


Fig. 5. Case 111. Excellent visualization of the aqueduct, third and fourth ventricles and air in the dilated frontal horn. There is a mass in the third ventricle.

Case 111. R.C.B., a 6 year old child, was admitted to the Private Clinic with chief complaints of vomiting and headache. The birth history was normal. She had been relatively well until 10 days before admission, when she developed headache and morning vomiting, and 3 days prior to admission her gait became unsteady. Neurologic examination revealed incoordination of upper extremities with ataxic gait. Laboratory studies showed essentially normal blood, serology, urinalysis, chemistry and spinal fluid. Pneumoencephalography demonstrated hydrocephalus, but there was no filling of the third ventricle, aqueduct and fourth ventricle. Hence, direct catheterization of the third ventricle through a frontal burr hole with injection

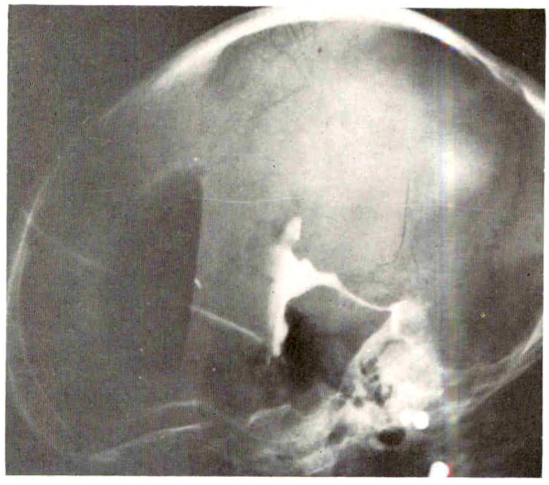


FIG. 6. Case IV. Shift of aqueduct.

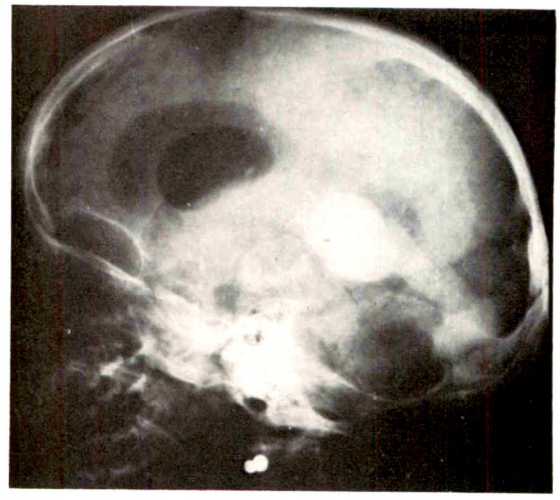


FIG. 8. Case IV. Humeral arteriogram demonstrates aneurysm of the great vein of Galen.

of densopax was performed. The roentgenogram demonstrated a mass in the third ventricle (Fig. 5). A right frontal bone flap was turned down and a vertical transcortical incision was made in the frontal lobe anterior to the motor area down into a dilated ventricle. A mass tumor was removed through the enlarged interventricular foramen. The neoplasm was considered to be a glioma; it was reported as an ependymoma.

CASE IV. A. Di S., a 1 year old female, was admitted to the Private Clinic because of an enlarged head. Examination showed a well-nourished infant with a vigorous cry. There was obvious hydrocephalus. There was cracked-pot resonance to percussion and slight horizontal nystagmus. Selective midline contrast ventriculography was performed using densopax (Fig. 6). An abnormal image with a shift of the aqueduct was visualized. On the anteroposterior roentgenogram made 5 minutes later, it was possible to see the great veins as the contrast material was reabsorbed (Fig. 7). Suspecting a malformation, humeral angiography was carried out and a deformity of the great vein of Galen was demonstrated (Fig. 8).

CASE V. R.B., a 5 year old child was admitted to the Children's Hospital on March 3, 1966 complaining of a headache, which started 2 months previously, ataxic gait and impaired



FIG. 7. Case IV. The great veins are seen after contrast material is reabsorbed.

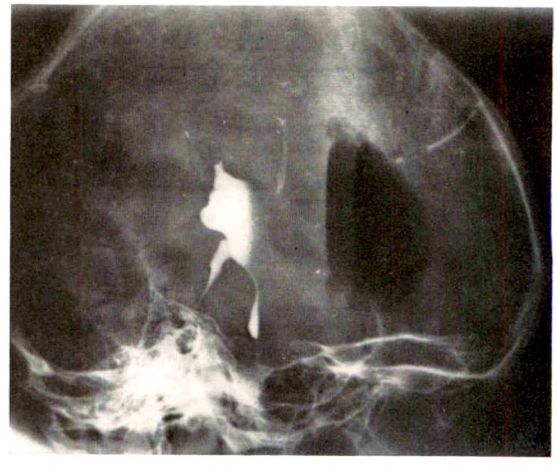


FIG. 9. Case V. Aqueductal occlusion. Fourth ventricle is not seen.

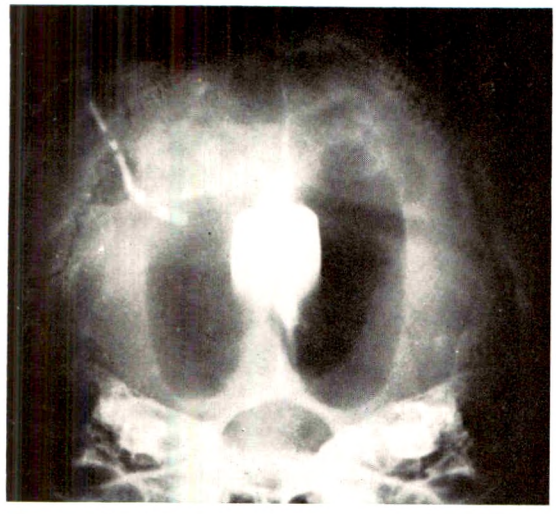


Fig. 10. Case v. Anteroposterior central air densopax ventriculogram. Note well defined dilated third ventricle and lateral shift and cut-down of aqueduct.

coordination. A roentgenogram of the skull demonstrated a slight separation of coronal sutures. There was no papilledema. A pneumoencephalogram did not demonstrate the ventricular system. Two days later, central air densopax ventriculography showed a lateral shift and cut-down of a dilated aqueduct (Fig. 9 and 10). Suboccipital craniectomy under endotracheal anesthesia revealed a cyst and a mass arising from the right cerebellum. Microscopic examination showed astrocytoma. A satisfactory convalescence followed the administration of intravenous oncovin (vincristine sulfate).

Case vi. C.A.B., a 16 month old child with meningocele appeared to be hydrocephalic. Central air densopax ventriculography was performed on March 10, 1966. Because of the child's vigorous movements, the catheter was forced out. The study revealed hydrocephalus and inferior displacement of the fourth ventricle—a deformity typical of the Arnold-Chiari syndrome (Fig. 11).


## SUMMARY

A new contrast medium, densopax (sodium salt of 3-acetylamine 2,4,6-triiodobenzoic acid and polyvinylpyrrolidone), for use in central midline ventriculography is presented. This medium is opaque, nonirritant, viscous, hydrosoluble and has optimum absorption qualities.

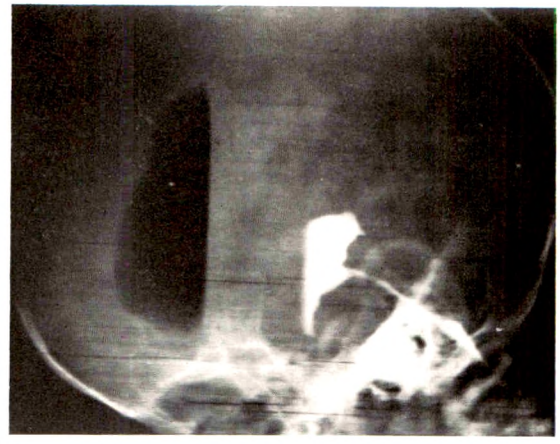



Fig. 11. Case vi. Displacement of fourth ventricle (Arnold-Chiari malformation).

The technique of central midline ventriculography, which is considered to be the procedure of choice in the roentgen diagnosis of surgical lesions of the posterior cranial fossa, is described. The procedure is also useful in outlining Arnold-Chiari malformation.

No adverse effects due to the intracranial densopax or meningeal reactions have been noted in our cases. In an inoperable case, necropsy did not show any lesion of the inflammatory type in the plexus or meninges which could be attributed to the densopax.

In 5 hydrocephalic babies, injection of the medium did not provoke any disorder in consciousness or vegetative functions, either during or after its performance.


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# EXPERIENCE WITH POSITIVE CONTRAST VENTRICULOGRAPHY*

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ADEQUATE demonstration of the structures in the posterior fossa is one of the most difficult problems in neuroradiology. It is even more difficult, and more critical, in the presence of increased intracranial pressure. Positive contrast ventriculography has been recommended either as an adjunctive procedure after air ventriculography, or as the primary study of choice.

This technique was first reported in 1928[1] but was not used generally because of the toxicity of the contrast media available.

Pantopaque was introduced in 1943, and its safety was quickly established. In 1950, Bull[2] reported 80 cases in which pantopaque ventriculography had been performed without complications. Horowitz[3] reported his experience with positive contrast ventriculography in 1956. He felt that it was not a replacement for air, but, in selected cases, it was very advantageous. Wilson and Snodgrass[8] agreed with the accuracy and safety of the procedure.

In 1960, Jefferson and Occleshaw[4] reported their large experience with positive contrast ventriculography. They demonstrated the aqueduct of Sylvius and 4th ventricle in 125 cases using pantopaque. They specifically recommended positive contrast ventriculography instead of air studies when obstruction was present.

In 1964, Robertson[7] presented a comparative evaluation of pneumoencephalography and positive contrast ventriculography. He found that air rarely filled the aqueduct of Sylvius and 4th ventricle when the intracranial pressure was elevated, and felt that positive contrast ventriculography was indicated under these circumstances.

Despite this accumulated experience, pneumography is still generally used alone in studying patients with posterior fossa symptoms and signs. Recently, we have had several cases which demonstrate the indications for pantopaque ventriculography and its usefulness.

## METHOD

Figure 1 illustrates the technique, which is quite simple. With the patient in the sitting position, a cranial burr hole is made in the usual occipital location for ventriculography. The lateral ventricle is tapped and 1 to 3 cc. of pantopaque is instilled into a lateral ventricle with the head flexed. The head is slowly extended. Serial roentgenograms or fluoroscopy are used to follow the pantopaque through the ventricular system.

## ILLUSTRATIVE CASES AND COMMENTS

CASE 1. A 57 year old white male preacher was admitted to Parkland Memorial Hospital in deep coma. He had complained of occipital headaches, ataxia, and progressive visual impairment for 6 months. He became unconscious 3 days prior to admission. The significant findings on examination were coma and severe bilateral papilledema with hemorrhages and exudates.

A pneumoventriculogram was done, visualizing only the dilated lateral and 3rd ventricles. A suboccipital craniectomy was then performed. The only abnormality found was the lack of cerebrospinal fluid drainage through the aqueduct of Sylvius. With the presumptive diagnosis of brain stem glioma, a Torkildsen shunt was carried out.

Several days later, positive contrast ventriculography was done to confirm this diagnosis (Fig. 2). There was found an area of stenosis of the aqueduct of Sylvius in both the anteroposterior and lateral ventriculograms. The 3rd

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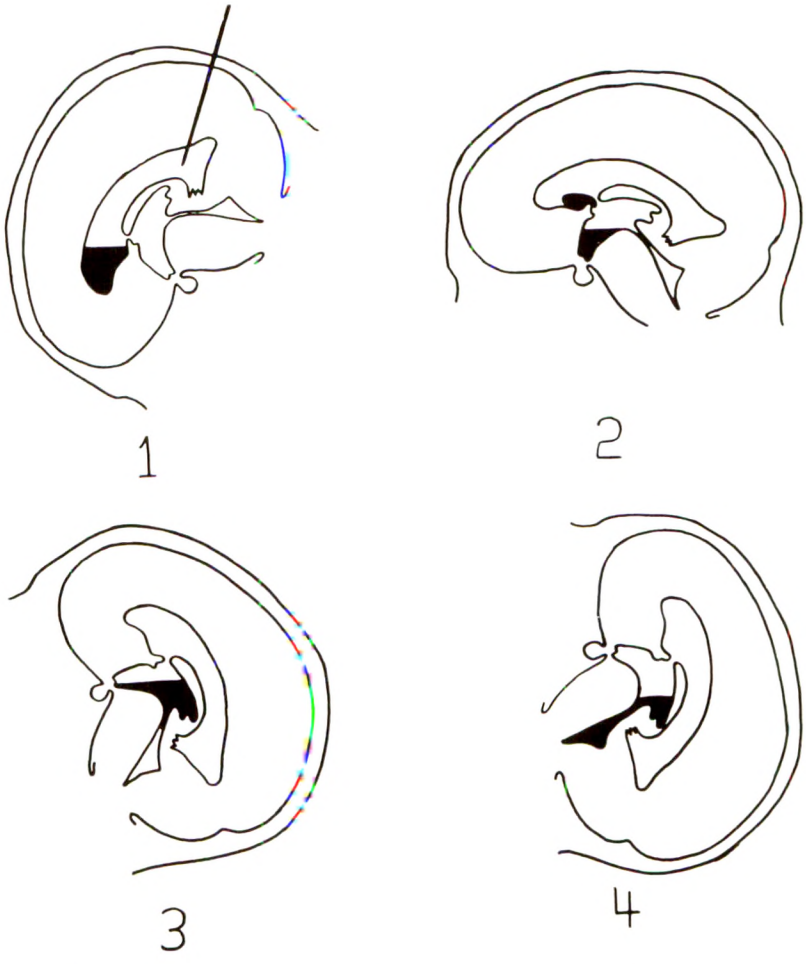



FIG. 1. Method of performing positive contrast ventriculography.

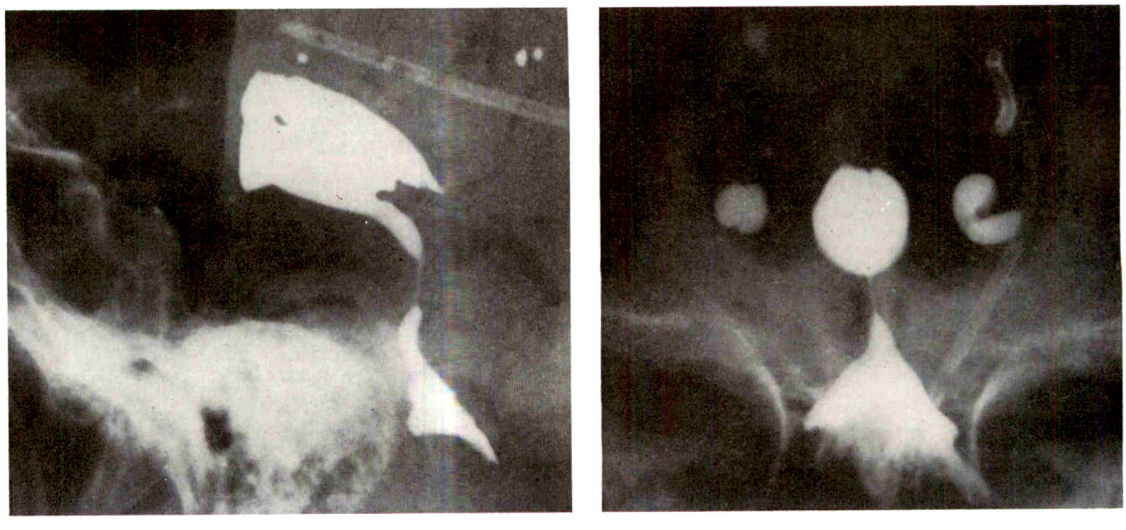


FIG. 2. Case 1.

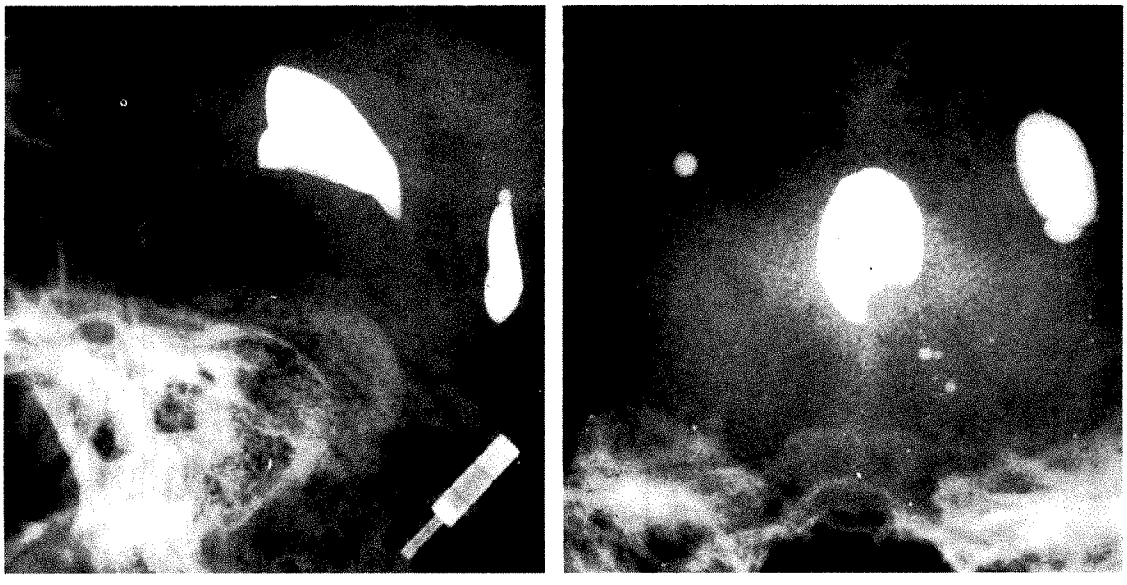


Fig. 3. Case II.

and 4th ventricles were in the midline and not deformed.

At autopsy 2 months later, an astrocytoma (Grade II) was found surrounding the aqueduct.

This patient had a surgical exploration because of the failure to fill the posterior fossa with air. A positive contrast study would have localized the tumor and a simpler operation, such as a ventriculo-atrial shunt, could have been done.

Case II. A 15 year old Negro boy was admitted with occipital headaches, staggering, and postprandial vomiting for 1 month. Neurologic examination showed bilateral papilledema and nystagmus in all fields of gaze. The gait was widebased and ataxic. His deep tendon reflexes were hypoactive.

A pneumoventriculogram revealed dilated lateral and 3rd ventricles and displacement of the aqueduct and 4th ventricle posteriorly. However, the posterior fossa shadows were so faint that localization of the mass in relationship to the 4th ventricle was difficult. A ventriculo-atrial shunt provided decompression. Figure 3 shows a positive contrast study done 1 week later, which revealed complete aqueductal occlusion. The upper limit of the tumor is seen indenting the 3rd ventricle at the origin of the aqueduct. Suboccipital craniectomy was done and a huge ependymoma of the 4th ventricle was found. This was confirmed at autopsy 1 month postoperatively.

In this case the air study was suggestive of either an intraventricular tumor or a brain stem tumor, but not diagnostic of either. The positive contrast study could have been done as the primary study in less time, with less derangement of intracranial pressure dynamics, and with more precise localization of the tumor. The limited knowledge of the upper end of the tumor being in the 3rd ventricle was useful during the operation.

Case III. A 30 year old white man presented with symptoms of increased intracranial pressure of 3 months' duration. On examination he had nystagmus, an ataxic gait, diminished tendon reflexes, Babinski plantar responses, but without lateralizing signs. Pneumoventriculography failed to fill the 4th ventricle.

Figure 4 shows the pantopaque study which demonstrated a right cerebellar hemisphere lesion. At operation, an extensive astrocytoma (Grade II) was subtotally removed. Again, the posterior fossa structures could not be demonstrated using air, but with the addition of pantopaque, an accurate localization of the tumor was accomplished.

Case IV. A 41 year old white woman was admitted with recurrent headaches and failing vision in the right eye. Three years before she had similar symptoms, and at that time ventriculography revealed hydrocephalus, felt to be secondary to aqueductal occlusion. A

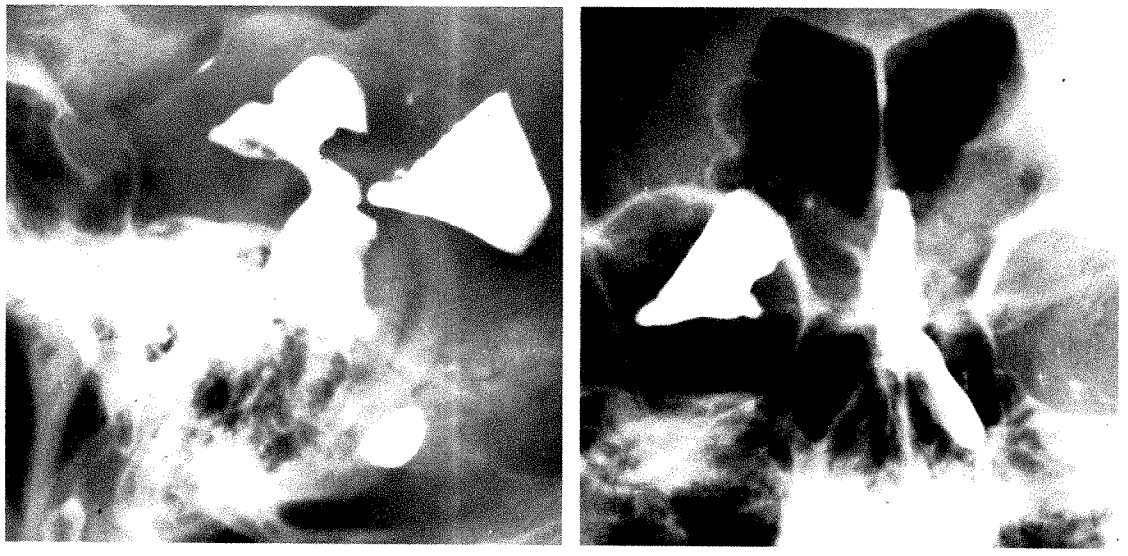


FIG. 4. Case III.

ventriculo-atrial shunt was done using a Holter valve. When she presented to us, the shunt was not functioning and she was very lethargic.

Figure 5 shows the positive contrast ventriculogram. The dilated 3rd ventricle and occluded aqueduct can be seen.

This case illustrates the advantage of using positive contrast to locate the point of blockade in obstructive hydrocephalus. This type of study has also been useful and safe in children with hydrocephalus.[6] In this case, we elected to remove the pantopaque.



FIG. 5. Case IV.

## DISCUSSION

In studying obstructive lesions of the posterior fossa, we feel that positive contrast ventriculography is preferable to pneumoventriculography to demonstrate the aqueduct and 4th ventricle. Partial or complete obstruction in the aqueduct or 4th ventricle makes filling with air very difficult. Even when filling is accomplished, the volume of these channels is so small that the roentgenographic shadows produced are very faint and easily obscured by overlying sulcal air and bone shadows. Since accurate localization of the obstructing lesion is necessary to allow sound surgical management, positive contrast ventriculography should be used to obtain this information.

It should be made clear that air study is necessary to demonstrate the lateral and 3rd ventricles. This will eliminate the possibility of a primary or secondary supratentorial lesion. The positive contrast study can then be performed to evaluate the posterior fossa. The posterior portion of the 3rd ventricle, aqueduct of Sylvius, and 4th

ventricle can be demonstrated with a minimum of manipulation of the patient. More accurate and useful information is obtained by the use of 2 to 3 cc. of pantopaque than can be obtained by air. This amount is both adequate and safe.[5]

SUMMARY

The literature on positive contrast ventriculography has been reviewed and suggests that the use of this procedure greatly improves the roentgenographic demonstration of obstructive lesions of the posterior fossa. This has been our experience. Illustrative cases are cited in which positive contrast studies aided materially in the management of the patient's disease.

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# TECHNETIUM/IRON/ASCORBIC ACID COMPLEX*

## A GOOD BRAIN SCANNING AGENT

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SEATTLE, WASHINGTON

THE scintillation camera has been used by various investigators[1,2,6,7] for several years. It was described in detail by Anger in 1964.[2] Such a camera was delivered to the Tumor Institute of the Swedish Hospital in the fall of 1964, and we are now satisfied that it is a valuable instrument for the evaluation of brain lesions when used with the appropriate radioactive material.

To date, approximately 400 brain examinations using various radioactive materials have been performed at the Tumor Institute. Radioiodinated human serum albumin, chlormerodrin tagged with mercury 197 and technetium 99m as pertechnetate have been used with disappointing results. With the development of technetium 99m/iron/ascorbic acid, an agent has been found which allows us to make full use of the potentialities of the gamma camera. Each view takes approximately 30 seconds; resolution is good and there is excellent tumor-to-brain ratio.

### METHODS AND MATERIALS

$Hg^{197}$ and $Hg^{203}$ chlormerodrin, $RI^{131}HSA$ were obtained as pharmaceuticals from Abbott Laboratory and E.R. Squibb & Sons. $Tc^{99m}$ pertechnetate is presently eluted from a generator supplied by the New England Nuclear Corporation. Earlier supplies included Nuclear Consultants and the Brookhaven National Laboratory.

The preparation of $Tc^{99m}$ pertechnetate follows the usual procedures.[13] The pertechnetate is eluted from an alumina column containing $Mo^{99}$ molybdenate by washing with saline. It is sterilized by either autoclaving or millipore filtration. The activity of the sterile pertechnetate is determined by comparison with a $Co^{57}$ standard. The level of $Mo^{99}$ contamination is determined by a 400 channel gamma spectrum analysis. Cultures are obtained to give "post facto" indication of sterility.

Technetium 99m/iron/ascorbic acid* is prepared by using a modified technique of Harper *et al.*[4] (reasons for this modification will be presented in a later report[14]). The method of preparation is as follows: place in a sterile container 1 ml. of a 1 mg./ml. $FeCl_3$ solution in 3*N* HCl; add 1 ml. of a 100 mg./ml. solution of ascorbic acid (Abbott cenolate); add the required activity of $Tc^{99m}$ pertechnetate; mix for 10 minutes by passing $N_2$ gas (water purified) through the reaction mixture. The pH at this point should be less than 1.5 (pH is determined by the use of a Beckman Model G pH Meter, and a Beckman model 39183 combination electrode). Adjust the pH to 9.0 with 1*N* NaOH and then immediately adjust it to 7.4–7.8 with 0.1*N* HCl. Sterilize by passing through a 10 or 220 millimicron filter, then assay, etc., as pertechnetate. Careless preparation usually results in an agent which has the scanning characteristics of pertechnetate.

Dosage and scanning time are as follows: 400–800 μc of $Hg^{203}$ and 800–1,000 μc of $Hg^{197}$, scanning in approximately 4–6 hours; 500 μc of $RI^{131}HSA$, scanning in 24 hours; 6–10 mc of $Tc^{99m}$ pertechnetate, scanning in 15 minutes to 3–4 hours; and 6–15 mc of $Tc^{99m}$/iron/ascorbate, scanning in 3–30 minutes (the average is 15 minutes).

* Actual structure is unknown.

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§ Chemist.

The scintillation camera used is the commercial Nuclear Chicago version of the Anger camera.[1,2] Included are a 3 inch collimator with 931 parallel holes for high energy emitters ($Hg^{203}$, $I^{131}$) and a $1\frac{1}{2}$ inch collimator with 931 parallel holes for lower energy emitters ($Hg^{197}$, $Tc^{99m}$, etc.). A homemade, 3,000 hole, technetium collimator is now being utilized. Brain scanning techniques have changed radically since the scintillation camera was first acquired. We now obtain 300,000 counts per view. The oscilloscope "dot" is somewhat defocused to blend in slight statistical variations and give a more "real"appearing scintiphotograph. A Tektronix Model 564 Storage Scope has been added as a "viewfinder."

## RESULTS

Examples of the disappointing results obtained with $RI^{131}HSA$ and $Hg^{197}$ chlormerodrin are shown in Figures 1, *A* and *B*; and 2, *A* and *B*. Scanning time with the scintillation camera and these agents was 10 minutes or more for each view, and even then the statistical levels were barely acceptable. The resulting scintiphotographs did not approach the quality of rectilinear scans.

The theoretic advantage of $Tc^{99m}$ with both conventional scanners and the gamma camera, has been discussed by several authors.[3,4,10] Initial experience with this agent and the scintillation camera was encouraging, and we did obtain clinically useful scintiphotographs. Examples of scintillation camera scans with pertechnetate are shown in Figures 3, *A* and *B*; and 4, *A* and *B*.

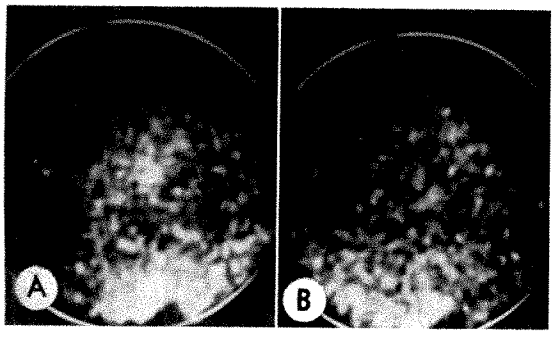



FIG. 1. T.B., 59 year old white female with episodes of unconsciousness. Scintiphotographs with $RI^{131}HSA$ were poor and time consuming. (*A*) Posteroanterior view. (*B*) Left lateral view.

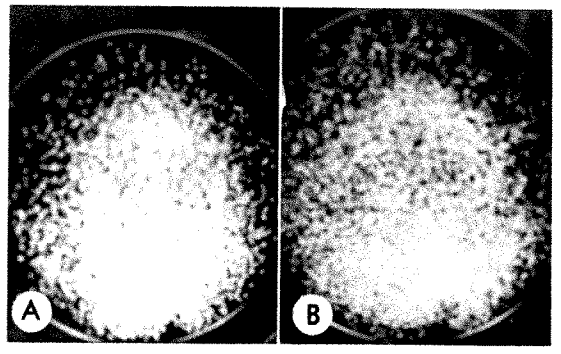



FIG. 2. K.C., 8 year old white female with headaches of sudden onset. Scintiphotographs with $Hg^{197}$ fail to demonstrate medulloblastoma later diagnosed. (*A*) Posteroanterior view. (*B*) Left lateral view.

However, it was not always possible to demonstrate a lesion using pertechnetate and the scintillation camera as definitely as when using $Hg^{203}$ and the rectilinear scanner. The fact that pertechnetate was slowly excreted provided a clue as to how the tumor-to-brain ratio might be improved. After considerable experimental work with rabbits, it was found that a complex of technetium 99m/iron/ascorbic acid was very rapidly excreted. A clinical trial comparing this complex with other agents in use demonstrated its superiority

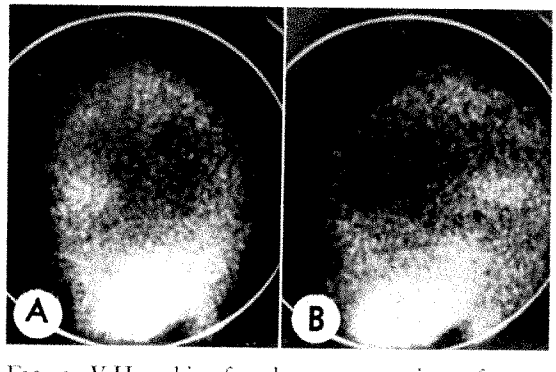



FIG. 3. V.H., white female, postoperative, after removal of meningioma with subsequent radiation therapy. Scintiphotographs with pertechnetate show left occipital lesion. (*A*) Posteroanterior view. (*B*) Left lateral view.

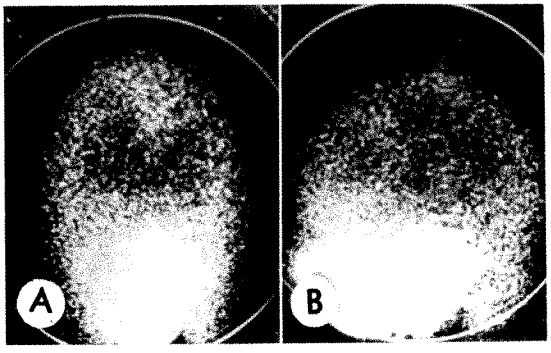



FIG. 4. S.H., white male with diagnosis of encephalopathy. Scintiphotographs with pertechnetate show left frontoparietal lesion, confirmed by arteriography. (*A*) Anteroposterior view. (*B*) Left lateral view.

to our satisfaction. Figures 7, *A–F*; and 8, *A–D* show a direct comparison in 2 patients between the pertechnetate and technetium 99m/iron/ascorbic acid complex in which the various technical factors factors are matched as closely as possible.

The technetium 99m/iron/ascorbic acid complex was initially used by the authors in November of 1965, and is now used almost exclusively. Improving the original electronics of our gamma camera has also resulted in better resolution. Now brain lesions can be demonstrated by the gamma camera with at least the same degree of certainty as by rectilinear scanning with mercurial compounds or pertechnetate (Fig. 5, *A–C*; and 6, *A* and *B*).

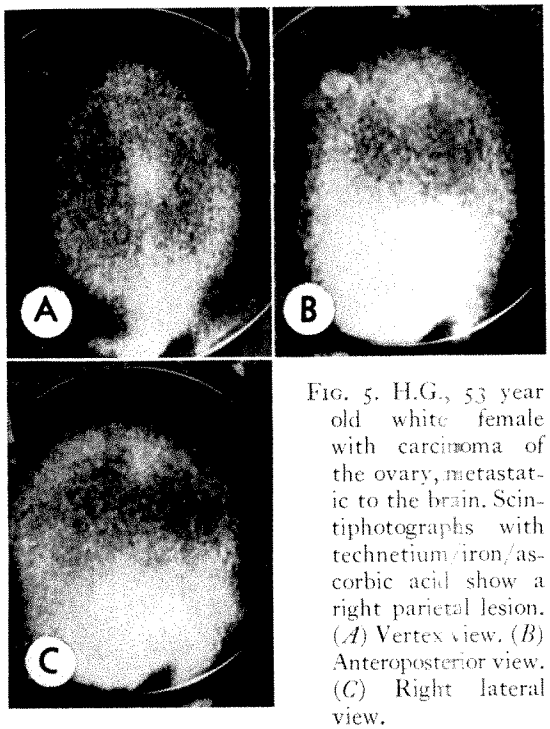



FIG. 5. H.G., 53 year old white female with carcinoma of the ovary, metastatic to the brain. Scintiphotographs with technetium/iron/ascorbic acid show a right parietal lesion. (*A*) Vertex view. (*B*) Anteroposterior view. (*C*) Right lateral view.

## DISCUSSION

The advantages of $Tc^{99m}$ over mercurial compounds have been adequately described by various authors.[7,9,11] The shorter half-life of $Tc^{99m}$ makes feasible the administration of larger doses with resulting increased counting rates and faster scanning speeds. Patient irradiation is reduced to very small amounts making repeat examinations less objectionable. $Tc^{99m}$ is particularly suitable with the gamma camera because of its monochromatic emission of 140 kev. gamma rays.

Because of its rapid blood clearance, technetium 99m/iron/ascorbic acid seems to have the same advantage over pertechnetate as did chlormerodrin over $RI^{131}HSA$. The main disadvantage of the complex is the necessity for its daily manufacture in the laboratory, a process beyond the capability of a laboratory technician at this time.

Gamma encephalography is a very useful screening test for intracranial pathology. The use of the gamma camera and technetium 99m/iron/ascorbic acid complex en-

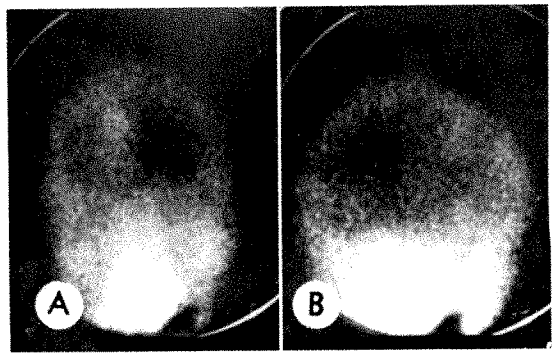



FIG. 6. M.J., 31 year old white female who had surgical removal of a brain tumor 7 months before examination. Scintiphotographs with technetium/iron/ascorbic acid show a right frontoparietal lesion near the midline. (*A*) Anteroposterior view. (*B*) Right lateral view.

ables the procedure to be carried out quickly and easily. The first pictures can be obtained if necessary 3 minutes after injection, although 15 minutes is preferred. Anterior, posterior, both lateral and a vertex scan as well as numerous repeat scans may be obtained within 20 minutes. Section scanning can also be accomplished in the same sitting. If necessary, conventional rectilinear scanning can be performed after taking the scintiphotographs. We feel that the rapidity of the examination and the ability to obtain numerous projections, coupled with excellent resolution and enhanced tumor-to-brain ratio, results in a superior radioisotope study of the brain.

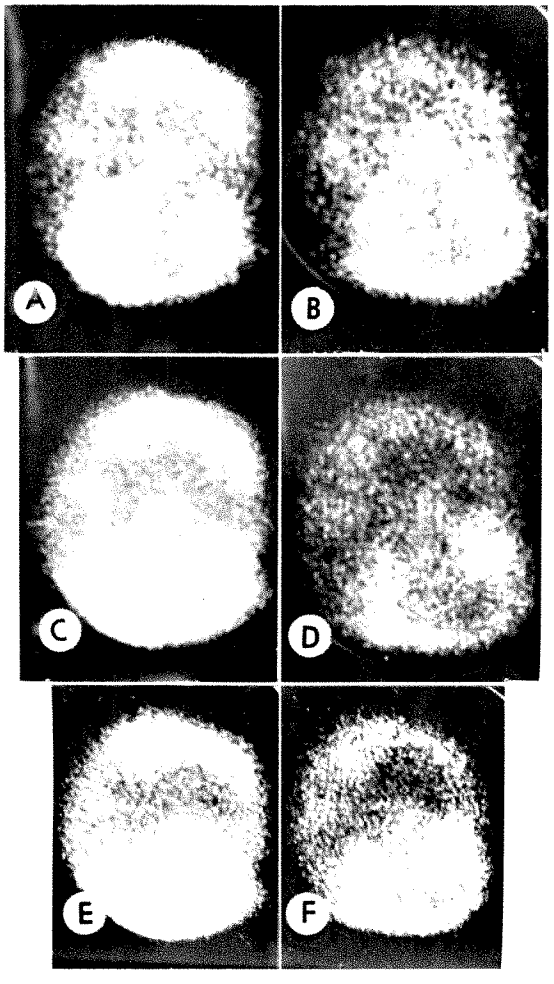



FIG. 7. C.F., 30 year old white male with synovial sarcoma. Scintiphotographs comparing technetium as pertechnetate and technetium/iron/ascorbic acid complex. At 2 minutes after injection, the tumor is easily visualized with technetium/iron /ascorbic acid complex, but is not seen with technetium as pertechnetate. The tumor continues to be better visualized at 7 minutes with the complex. (*A*) Tc/Fe/2 min. (*B*) $TcO_4^-$/2 min. (*C*) Tc/Fe/3 min. (*D*) $TcO_4^-$/3 min. (*E*) Tc/Fe/7 min. (*F*) $TcO_4^-$/7 min.

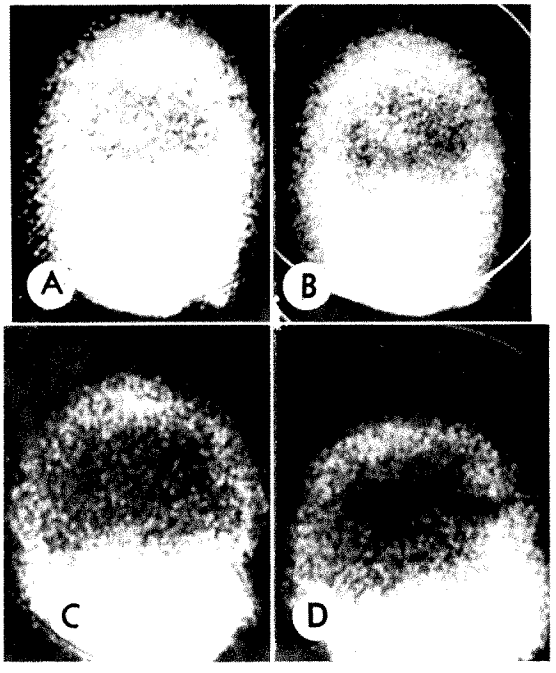



FIG. 8. E.B., 33 year old white female with primary brain tumor. Pertechnetate scintiphotographs fail to show lesion in lateral view. Technetium/iron /ascorbic acid complex scintiphotograph done 1 day later demonstrates a frontal lesion. (*A*) $TcO_4^-$ anteroposterior view. (*B*) Tc/Fe anteroposterior view. (*C*) $TcO_4^-$ right lateral view. (*D*) Tc/Fe right lateral view.

## SUMMARY

The use of various compounds in gamma encephalography with the scintillation camera is presented.

A method of preparation of technetium 99m/iron/ascorbic acid complex is described.

Advantages and disadvantages in the clinical use of the complex are discussed.

In our hands, technetium 99m/iron/ascorbic acid complex has proved to be the agent of choice for both the scintillation camera and the rectilinear scanner in the detection of brain lesions.

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# ROENTGENOLOGY OF VENTRICULO-ATRIAL SHUNTS FOR THE TREATMENT OF HYDROCEPHALUS*

*By* GERALD J. KURLANDER, M.D., *and* GONZALO T. CHUA, M.D.

HYDROCEPHALUS may be defined as ventricular enlargement, without regard to head size. The two basic categories of hydrocephalus are: (1) hypertensive hydrocephalus (obstructive hydrocephalus) and (2) hypotensive hydrocephalus (hydrocephalus *ex vacuo*).[31] Hypotensive hydrocephalus includes primary maldevelopment or secondary loss of brain substance from such causes as trauma, ischemia and infection. This latter type of hydrocephalus is not ordinarily associated with increased intracranial pressure. Hypertensive hydrocephalus, on the other hand, is associated with increased intracranial pressure and in most cases results from some obstruction in the cerebrospinal fluid pathways from the sites of origin in the ventricular system to the extracerebral sites of absorption. It is in this type of hydrocephalus that treatment is usually required to relieve or bypass the site of obstruction. Unfortunately in many instances surgical relief of the obstructive site is not possible. Treatment, then, must be directed along one of two lines:[28] (1) reduction of cerebrospinal fluid formation, and (2) shunting of excess cerebrospinal fluid away from the high pressure region usually into another area of the body such as the mastoid, ureter, thoracic duct, bone marrow, gallbladder, fallopian tube, Stenson's duct, pleural or peritoneal cavities. An effective shunting procedure currently being widely used is the ventriculo-atrial shunt.

The purpose of the authors is to point out the role of roentgenology in the placement of a ventriculo-atrial shunt and in the follow-up of an individual with such a shunt. Emphasis will be placed on the roentgen manifestations of complications of ventriculo-atrial shunts. As a basis for this report, the roentgenograms of 300 cases with ventriculo-atrial shunts have been reviewed.

## DESCRIPTION AND PLACEMENT OF VENTRICULO-ATRIAL SHUNTS

There are two types of one-way valves in common use today for shunting cerebrospinal fluid from a lateral cerebral ventricle to the right cardiac atrium. These are the Holter (Spitz-Holter) and the Pudenz (Pudenz-Heyer) valves.[18,22,28] The vast majority of ventriculo-atrial shunt operations performed at this institution have utilized the Holter valve. This valve consists of a pair of "fish-mouth" valves connected by a siliconized rubber cylinder. The low pressure valve is regulated by the manufacturer to open at a pressure greater than 10 mm. of water and the high pressure valve at a pressure greater than 50 mm. of water.[3] In addition, a medium pressure valve is available.

The valve is placed under the scalp in the mastoid region. Barium impregnated siliconized rubber catheters of very low tissue reactivity connect the valve proximally to the lateral cerebral ventricle and distally to the right cardiac atrium. The proximal catheter is performed at a right angle and is inserted into the lateral ventricle through a burr hole. The distal catheter is introduced into the right common facial vein or right internal jugular vein and passed through the right innominate vein and superior vena cava into the right atrium. If necessary the valve can be placed on the left side with the distal catheter passing through the left internal jugular vein.

The Pudenz and Holter systems are basically similar except that in the former the valve mechanism is of the "slit-type" and instead of being subcutaneous in the mastoid area, it is located distally within

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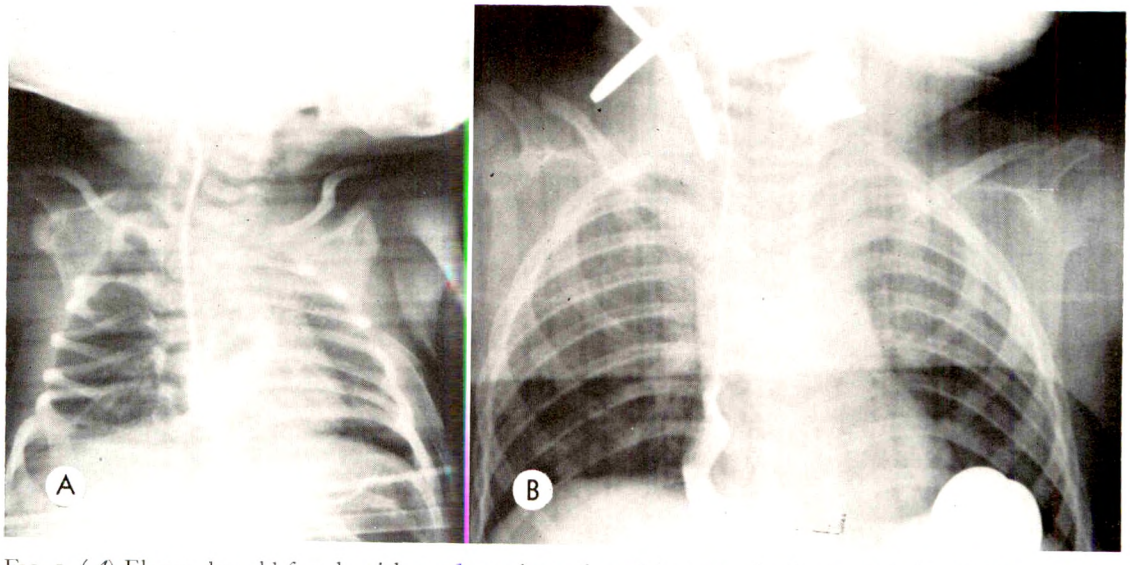



FIG. 1. (*A*) Eleven day old female with myelomeningocele and hypertensive hydrocephalus. Anteroposterior chest roentgenogram made at the time of surgery. Injection of radiopaque medium results in opacification of the left hepatic vein indicating unsatisfactory position of the catheter tip. Placement of the catheter tip on the basis of the vertebral level alone is unreliable particularly in the presence of multiple vertebral anomalies as demonstrated in this patient.

(*B*) Seventeen month old male with non-communicating hypertensive hydrocephalus. Anteroposterior chest roentgenogram made at the time of the first revision of the Holter system. Injection of radiopaque medium shows that the catheter tip has perforated the superior vena cava or right atrium resulting in opacification of the pericardiac sac.

the right atrium. Rather than the valve and tubing being radiopaque as in the Holter system, only the tips of the ventricular and cardiac catheters are impregnated with metallic powder to render them radiopaque.

Prior to surgery, a roentgenogram of the chest is made with a radiopaque ruler placed immediately adjacent to the chest. The ruler is placed a distance equal to half the anteroposterior dimension of the thorax from the table top or cassette. The distance between the suprasternal notch or medial end of the clavicle and 6th thoracic vertebra may be measured as a guide to the length of the distal catheter. At the time of surgery the catheter is introduced the predetermined distance. The position of the catheter can be verified by dripping saline solution through the tube thus converting it into an intracardiac electrocardiographic electrode.[24] The catheter can then be connected to an electrocardiograph and moved within the right atrial region until a biphasic P wave is recorded indicating the mid-atrial position of the catheter tip. It has been common practice at this institution to roentgenographically verify the position of the cardiac end of the Holter system by injecting radiopaque contrast medium through the distal tube at the time of surgery. The catheter tip can then be accurately placed in the mid portion of the right atrium. Relating the catheter tip to a thoracic vertebral body or interspace alone can be very misleading (Fig. 1, *A* and *B*). This is especially true when there are congenital anomalies of the thoracic vertebrae.

A postoperative chest roentgenogram should be obtained as a basis for future comparison since cephalad migration of the cardiac end of the system occurs with growth. A child grows an average of 6 cm. from the mastoid tip to the xiphoid process in the first 5 years of life.[22] If a very thin walled distal catheter is used, particularly in a small infant, sharp coning and excellent roentgenographic technique are necessary for catheter localization. Under these cir-

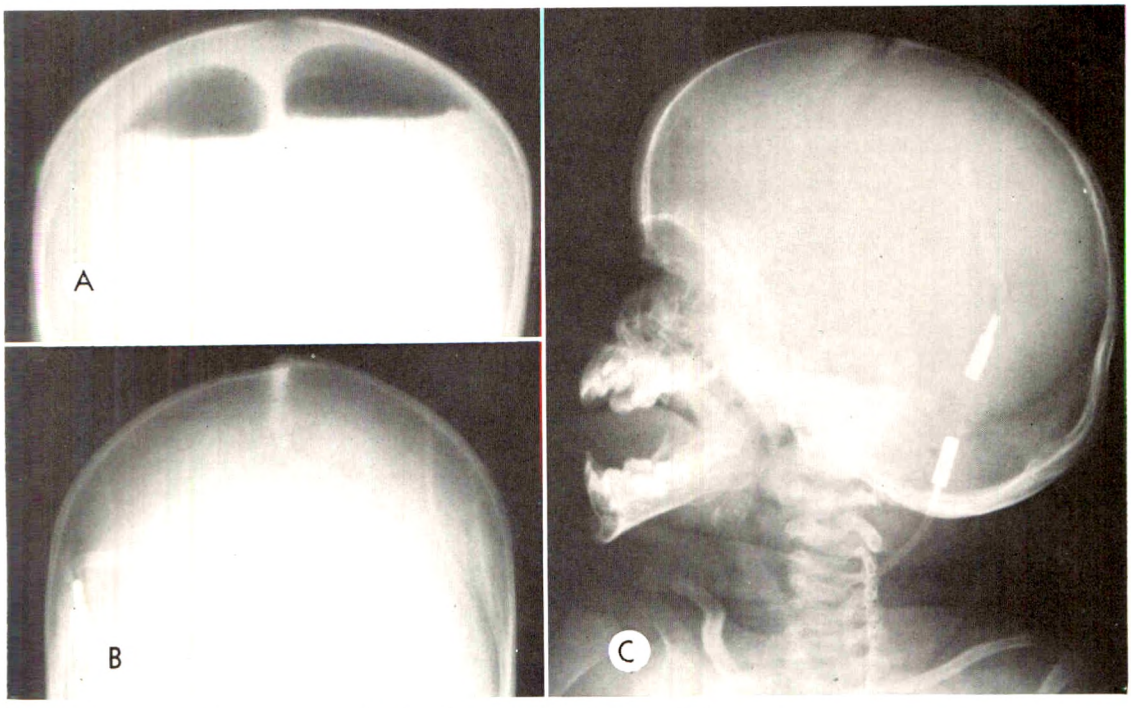



Fig. 2. Female with non-communicating hypertensive hydrocephalus.
(*A*) Upright frontal skull roentgenogram of a ventriculographic series at 4 months of age discloses marked dilatation of the lateral ventricles. (*B*) Frontal skull roentgenogram 3 months after ventricular decompression by a Holter system discloses marked overlap of the parietal bones. (*C*) Lateral skull roentgenogram made 11 months after ventricular decompression shows overdevelopment of the diploic layer of the skull.

cumstances, a mild right posterior oblique position in addition to the frontal projection is helpful in demonstrating the distal end of the catheter.

Since the Holter valve lies under the scalp in the mastoid region, the connecting tubing between the two valve components can be pumped by pressing it against the skull. The patency of the system can thus be determined. If the connecting tubing can be compressed but refilling fails to occur, a block in the proximal ventricular tube is present. On the other hand, if the valve is not compressible, there is obstruction in the distal catheter. A similar flushing mechanism is present in the Pudenz system.

## SEQUELAE AND COMPLICATIONS

### CRANIAL

After a successful ventriculo-atrial shunt has been established, there is a decrease in intracranial pressure, sometimes with apposition and overlap of the cranial bones (Fig. 2, *A–C*). Laminated thickening of the diploic portion of the calvaria may develop within several months. We have observed two instances of premature closure of the sagittal suture within 18 months of the time of insertion of a ventriculo-atrial shunt (Fig. 3, *A* and *B*). This may result from reduction of growth stress of the intracranial contents which are under low and perhaps subnormal pressure. Because of this complication some surgeons have discontinued use of the low pressure Holter valve.[1]

Rapid collapse of the ventricles can cause rupture of the bridging meningeal veins with subsequent development of subdural hematomata (Fig. 4).[7] Upward displacement of the brain stem and cerebellum secondary to shrinkage of the cerebral hemispheres is another important complication of decompression.[7] Acute pul-

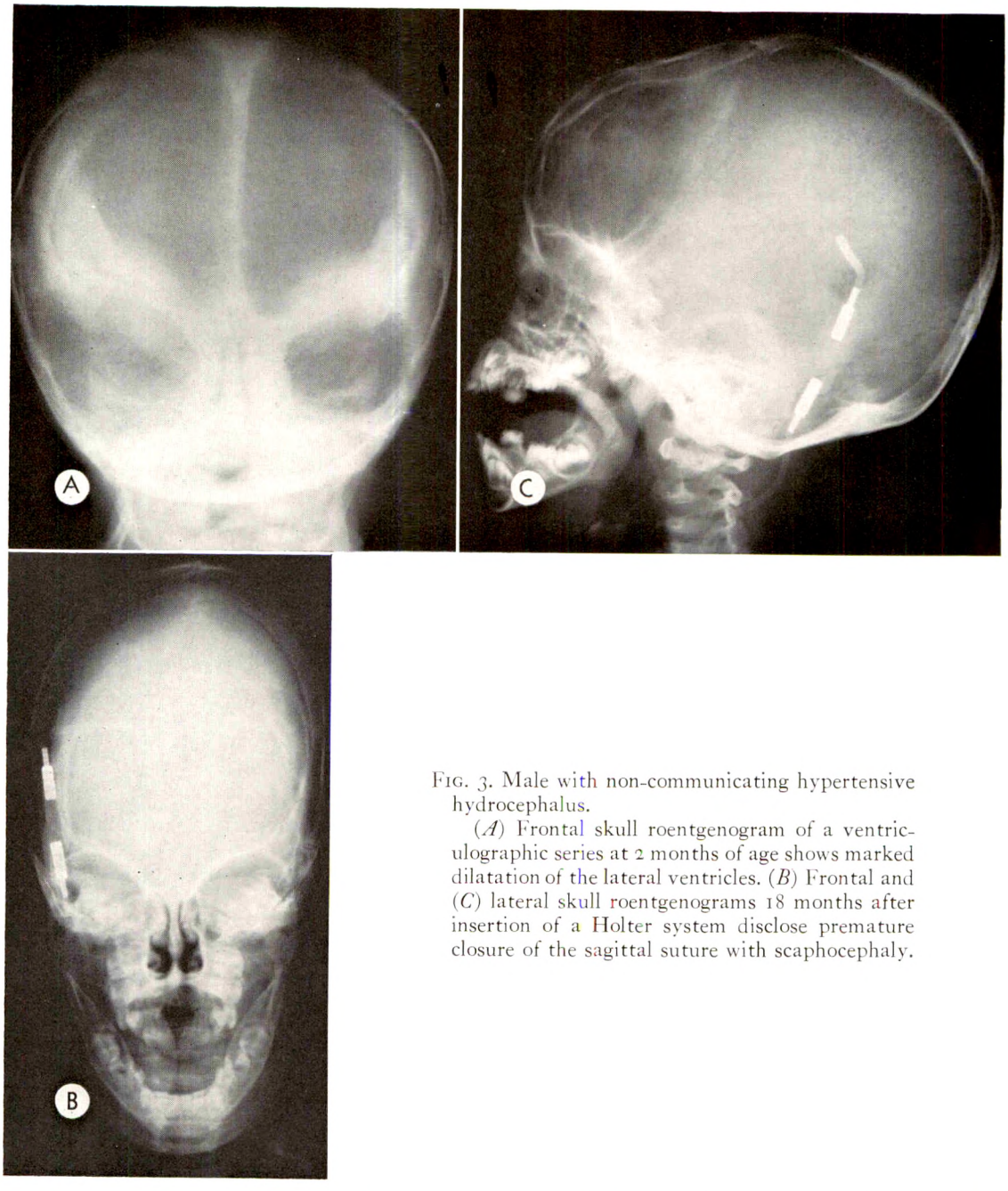



FIG. 3. Male with non-communicating hypertensive hydrocephalus.

(*A*) Frontal skull roentgenogram of a ventriculographic series at 2 months of age shows marked dilatation of the lateral ventricles. (*B*) Frontal and (*C*) lateral skull roentgenograms 18 months after insertion of a Holter system disclose premature closure of the sagittal suture with scaphocephaly.

monary edema from rapid drainage of a dilated ventricular system into the intravascular compartment is also a potential complication of ventricular decompression.[29]

When a ventriculo-atrial shunt becomes nonfunctional, a roentgen survey of the entire length of the valve and tubing in two projections should be performed. Separation of the proximal or distal tubes from the valve or a break in the tubing may be discovered. Occlusion of the proximal tubing by fragments of brain tissue or clotted blood, or the distal tubing by clotted blood may cause shunt failure but mechanical difficulty with the Holter valve itself rarely accounts for this complication.

Slight suture separation may be the first

indication of increased intracranial pressure after shunt failure has occurred. This important finding is easier to evaluate if base line skull roentgenograms had been made at some time after insertion of the ventriculo-atrial shunt.

THORACIC

The distal or right atrial catheter accounts for the majority of complications that occur with ventriculo-atrial shunts. This catheter acting as an intravascular foreign body stimulates thrombosis at its entry point into the jugular vein. The thrombus tends to propagate distally to the superior vena cava and right atrium resulting in partial or complete occlusion of the superior vena cava and its tributaries.[29,30] Thirteen of 15 cases with ventriculo-atrial shunts studied post mortem by Emery and Hilton[8] had partial or complete venous occlusion. There was thrombus formation about the distal end of the catheter in 10 of these cases and on the right atrial wall in 6. Atrial thrombosis did not occur in continuity with the distal tip of the catheter but seemed to develop at sites of trauma. In 2 cases, the thrombus extended to the tricuspid valve partially occluding the lumen. Sperling *et al.*[27] found a thrombus attached to the atrial end of the catheter in each of 12 autopsied cases who had ventriculo-atrial shunts for hydrocephalus. Nugent *et al.*[17] in a study with angiocardiography and at postmortem examination found that 35 per cent of their cases had thrombotic disease of the veins entering the right atrium and/or of the right atrium itself. Calcification of venous or atrial thrombi may occur.[9]

Recently we have seen 2 cases of superior vena caval obstruction in individuals with ventriculo-atrial shunts. In each instance it was possible to suggest the diagnosis on the basis of a dilated azygous vein on the frontal chest roentgenogram. Venography in both instances proved that the obstruction in the superior vena cava was above the point of entry of the azygous vein (Fig. 5, *A–D*). Hooper[12] reported hydrocephalus in an infant which developed secondary to obstruction of the superior vena cava, suggesting that this type of obstruction in cases of ventriculo-atrial shunts may actually aggravate the primary condition.



FIG. 4. Male with non-communicating hypertensive hydrocephalus.

Frontal skull roentgenogram made at the time of surgery 3 months after insertion of a Holter system; 500 cc. of liquid had just been removed from bilateral subdural hematomata. The thick dura on one side is identified by silver clips. Note the collapsed brain and bilateral air-filled subdural cavities.

Although it is known that esophageal varices (downhill varices) may occur with superior vena caval obstruction,[19] barium study of the esophagus failed to disclose varices in our 2 recent cases.

Pulmonary embolization may occur after venous or atrial thrombosis has developed. Friedman *et al.*[10] found pulmonary vascular lesions at postmortem examination in 57 per cent of 65 cases who had ventriculovascular shunting procedures. Emery and Hilton[8] noted multiple pulmonary emboli in 14 of 15 similar cases. In the majority of

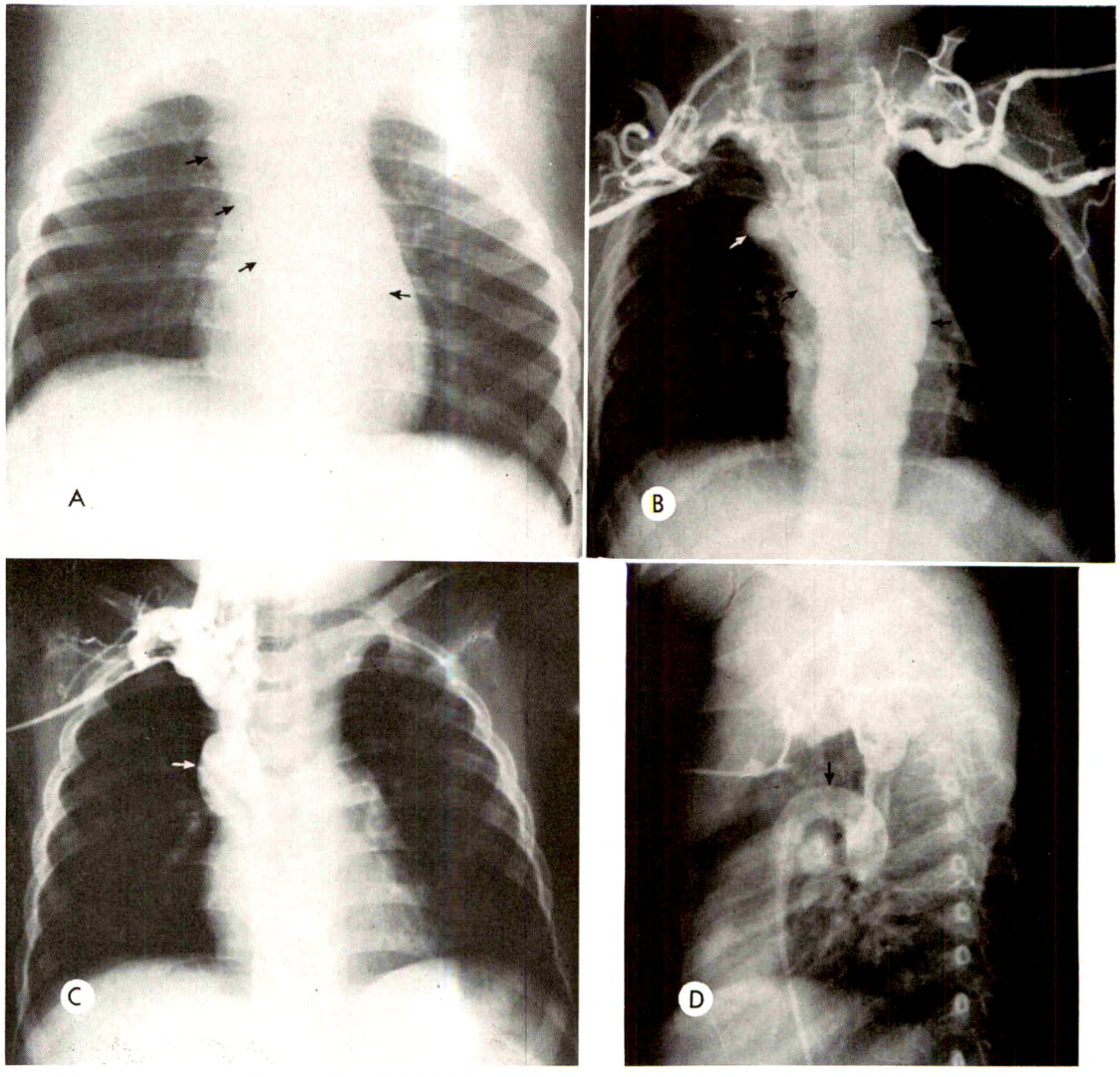



FIG. 5. Three year old female with hypertensive hydrocephalus.

(*A*) Frontal chest roentgenogram made $2\frac{1}{2}$ years after insertion of a Holter system discloses dilated azygous (arrows) and hemi-azygous (arrow) veins. (*B*) Frontal chest roentgenogram made after injection of radiopaque contrast medium into the antecubital veins of each arm. There is obstruction of the superior vena cava proximal to the point of entry of the azygous vein. The innominate and right subclavian veins fail to fill and may also be obstructed. The azygous (arrows) and hemi-azygous (arrow) veins are opacified. The tip of the distal catheter of the Holter system is in the right innominate vein.

(*C* and *D*) Four year old male who has had multiple ventriculo-venous shunt procedures for non-communicating hypertensive hydrocephalus. Frontal and lateral chest roentgenograms made after injection of radiopaque contrast medium into the right subclavian vein. The superior vena cava is obstructed proximal to point of entry of a dilated azygous vein (arrow). In addition to the injection catheter in the right subclavian vein, there is a catheter in the azygous vein which reached this point by passing through the inferior vena cava, right atrium and distal superior vena cava.

their 14 cases, however, the emboli were within small arteries which showed varying degrees of organization and recanalization. They also observed several pulmonary changes which seemed to be independent and unexplained by thrombosis and embolization. These changes were periarteritis, intimal degeneration, and one instance of hemosiderosis.

Although massive pulmonary embolism

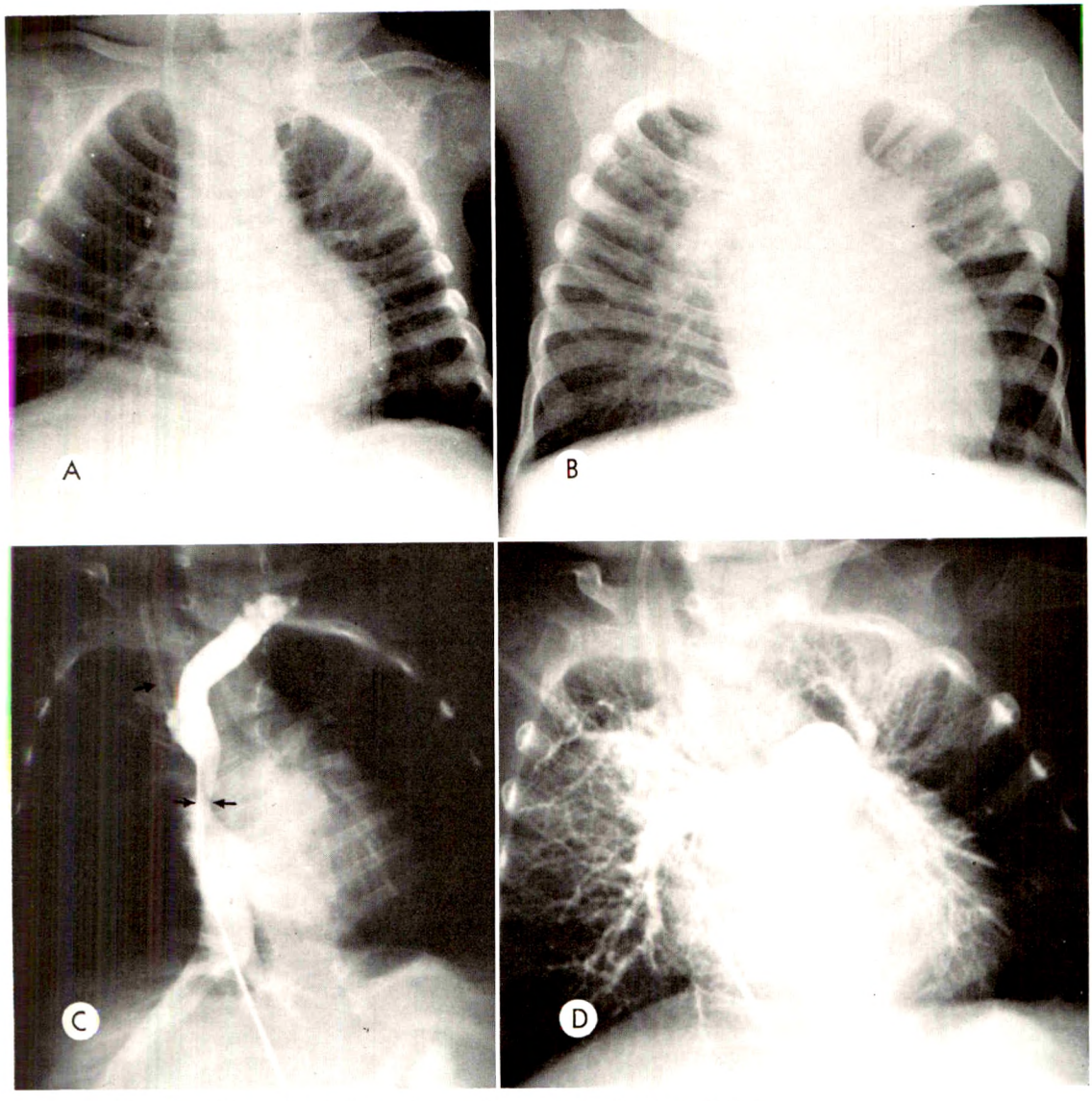



FIG. 6. Female with myelomeningocele and hypertensive hydrocephalus.

(*A*) Frontal chest roentgenogram made 5 months after insertion of a Holter system shows a heart of normal size and normal pulmonary arteries. (*B*) Frontal chest roentgenogram made $2\frac{1}{2}$ years later discloses cardiac enlargement with enlargement of the main pulmonary artery as well as the proximal right pulmonary artery. The proximal left pulmonary artery is obscured. There is rapid tapering of the branch pulmonary arteries. A right upper lobe infiltrate is identifiable. (*C*) Frontal chest roentgenogram made after injection of radiopaque contrast medium into the left innominate vein. There is narrowing of the lumen of the superior vena cava (arrows) just proximal to its entry into the right atrium. In addition, the right innominate vein is occluded. The distal tip of the Holter tubing can be seen in this vein (arrow). (*D*) Frontal chest roentgenogram made after injection of radiopaque contrast medium into the main pulmonary artery shows rapid tapering and tortuosity of the branch pulmonary arteries. The vascularity of the left lower lung field is diminished. Mean systolic pulmonary artery pressure was 55 mg. Hg.

resulting in acute cor pulmonale may occur occasionally, the real concern is the development of chronic cor pulmonale from continual seeding of the pulmonary arteries. The incidence of this latter complication is unknown. Sperling *et al.*[27] have recommended that every case with a ventriculoatrial shunt should have semi-annual eval-

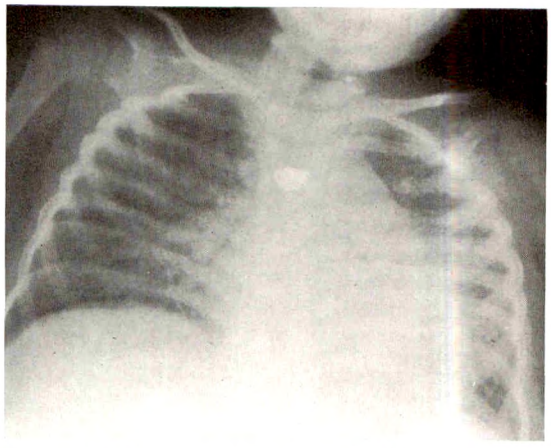


FIG. 7. Three and one-half year old female with myelomeningocele and non-communicating hypertensive hydrocephalus. Holter system inserted at 2 weeks of age.

Frontal chest roentgenogram made 10 days after revision of the Holter system discloses bilateral miliary infiltration which proved to be multiple pulmonary abscesses when studied post mortem.

uation for the detection of this complication and removal of the shunt if cor pulmonale is discovered. The chest roentgen findings of cardiac enlargement, dilatation of the main pulmonary artery, and central pulmonary arteries with rapid decrease in size of the branch pulmonary arteries should suggest this diagnosis (Fig. 6, *A–D*). Corroborative physical and electrocardiographic evidence are accentuation of the pulmonary 2nd sound, right ventricular hypertrophy, right axis deviation and P pulmonale.

The bronchial arterial collateral circulation to the lungs is capable in most instances of preventing infarction after embolic occlusions of small pulmonary arteries. This accounts for the absence or paucity of roentgen findings in the lungs of cases with ventriculo-atrial shunts in spite of the high incidence of pulmonary embolization discovered at postmortem examination. The onset of pulmonary hypertension and cor pulmonale in these patients is gradual and insidious. Periodic chest roentgenograms are capable of serving only as a gross indicator of the condition of the pulmonary arterial bed.

Even though the valve systems are of low tissue reactivity, they still act as foreign bodies inviting surface or internal bacterial colonization. The bacteria may be virulent such as *Staphylococcus aureus*, or saprophytic such as *Bacillus subtilis* and *Staphylococcus albus* whose pathogenicity has been enhanced by the presence of an intravascular foreign body.[26] The virulent bacteria may cause septicemia with embolization of the lungs producing lung abscesses and mycotic aneurysms (Fig. 7). Organisms of lesser virulence may produce a chronic bacteremia with persistent fever, progressive anemia and splenomegaly.[4] Overton and Snodgrass[20] found a 14 per cent incidence of postoperative infections in their series of 49 cases. Usually the presence of chronic bacteremia or septicemia necessitates removal of the shunt.

Crome and Erdohazi[5] noted bacterial endocarditis of the right atrium and/or tricuspid valve in 4 of 35 autopsied cases with ventriculo-atrial shunts. These vegetations in addition to serving as sources of septic emboli may cause obstruction of the tricuspid valve. We have not seen a case in which the diagnosis of tricuspid valve obstruction could be suggested from plain chest roentgenograms on the basis of right atrial or caval enlargement.

The distal catheter may break or become disconnected from the valve mechanism and act as an embolus to the right atrium or right ventricle. Several cases of successful removal of catheter emboli have been reported.[11,15] Since only the distal tip of the catheter is radiopaque in the Pudenz system, angiocardiography is necessary to establish the length and position of the catheter embolus. Breakage of the distal catheter proximal to its entry into the jugular vein may result in a fluid collection in the neck or erosion of the skin and external drainage.

Perforation of the right atrium or right ventricle followed by pericardial effusion and cardiac tamponade is an occasional complication of a ventriculo-atrial shunt.[5,10,13,29] This complication developed

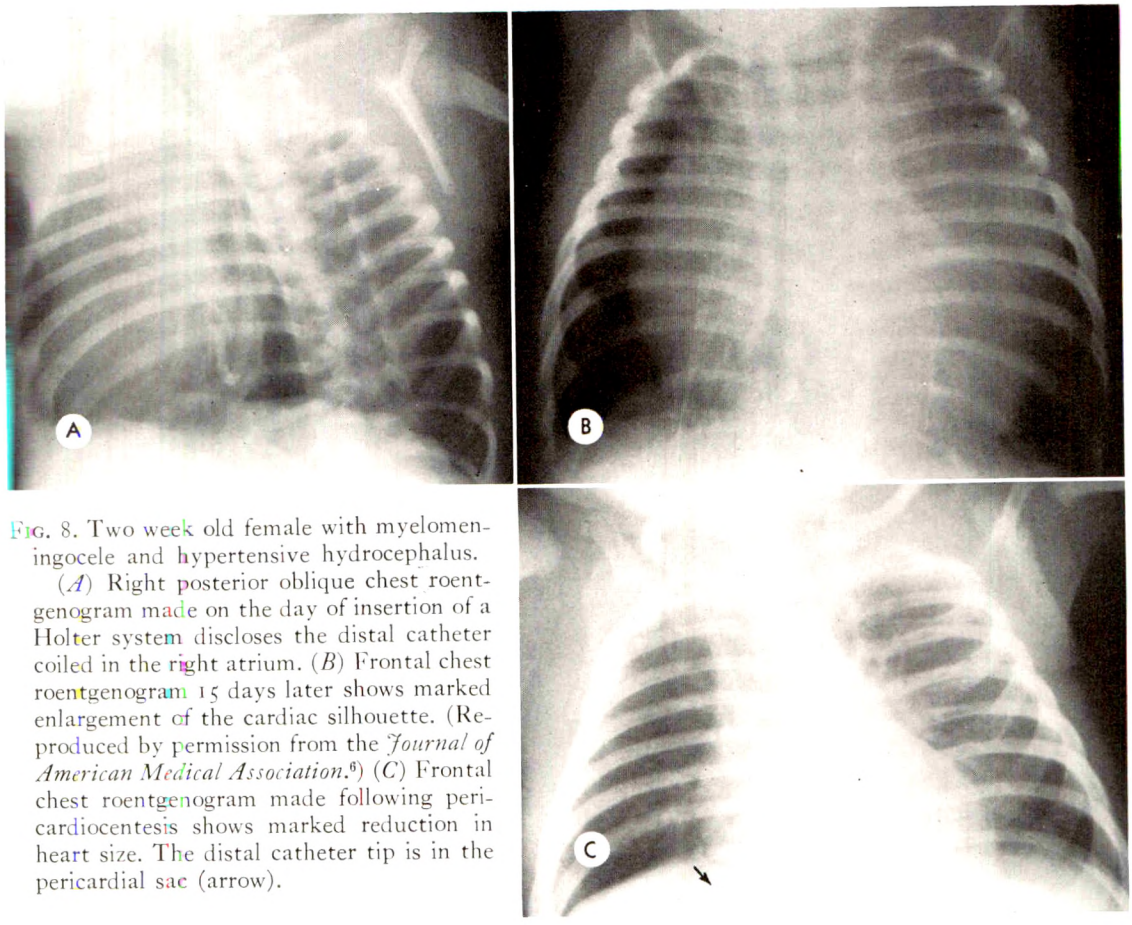



FIG. 8. Two week old female with myelomeningocele and hypertensive hydrocephalus. (*A*) Right posterior oblique chest roentgenogram made on the day of insertion of a Holter system discloses the distal catheter coiled in the right atrium. (*B*) Frontal chest roentgenogram 15 days later shows marked enlargement of the cardiac silhouette. (Reproduced by permission from the *Journal of American Medical Association*.[6]) (*C*) Frontal chest roentgenogram made following pericardiocentesis shows marked reduction in heart size. The distal catheter tip is in the pericardial sac (arrow).

in one case in this series and was successfully treated by thoracotomy and closure of the perforation (Fig. 8, *A–C*).[6] In this instance, before perforation, the distal end of the catheter was coiled and its tendency to straighten may have eroded the right atrial wall.

A functioning ventriculo-atrial shunt is a potential carrier of malignant cells from a cerebral ventricle to the blood stream. Makeever and King[16] have recently reported a case with disseminated medulloblastoma believed to have occurred through a ventriculo-venous shunt inserted for palliation of an obstructing medulloblastoma.

## DISCUSSION

Growth of an infant with a ventriculo-atrial shunt will be accompanied by cephalad migration of the tip of the distal catheter from the mid atrium to the superior vena cava where obstruction of the shunt from thrombosis is more likely to develop. Without angiography, it is difficult to be certain of the exact position of the catheter but a fair appraisal can usually be made from a frontal chest roentgenogram centered in the mid thoracic region. Although it has been suggested that the lower end of the superior vena cava corresponds to the level of the 4th thoracic vertebra,[21] in our experience it has usually been somewhat lower. We have found it more reliable to relate the catheter position to the heart and great vessels themselves calling upon anatomic concepts gained from experience with angiocardiography.

Some authors believe that when the catheter tip reaches the superior vena cava, a revision operation should be performed before thrombosis and occlusion occur.[2]

Where such a practice is popular, venous angiography by arm vein injection would be a safe and easy way of confirming the catheter position. Nugent *et al.*[17] have recently suggested that angiocardiography be performed in cases with a ventriculo-atrial shunt when there is evidence of sepsis, prior to shunt revision, and when the patient is doing poorly.

Jackson[13] has reported a 47 per cent incidence of shunt revision operations with the experience of others varying from 20–67 per cent. Manipulation of the catheter in attempting to replace the tip in the mid atrium could result in discharge of emboli to the lungs. Because of this possibility, a transthoracic or inferior vena caval approach to the right atrium has been suggested for revision operations.[21]

The head size of an infant or child with hydrocephalus after insertion of a satisfactorily functioning ventriculo-atrial shunt should decrease or remain stationary until body size catches up with head size. Evidence is conflicting regarding the period of time necessary for ventriculo-atrial shunting to be carried out. The work of Laurence[14] suggests that when spontaneous arrest of hydrocephalus occurs, it does so within the first 3 years of the disease. This would seem to be the critical period for shunting. Overton and Snodgrass,[20] on the other hand, suggest that spontaneous arrest does not occur once a ventriculo-atrial shunt has been established.

In spite of the high incidence of complications and the frequent necessity for revision of ventriculo-atrial shunts, there is evidence to suggest that both survival and mental development are better with this method than with any other shunt procedure thus far utilized.[13,25] In addition early insertion of a ventriculo-atrial shunt will result in a reduced incidence of spasticity and extremely large heads.[23]

## SUMMARY

Ventriculo-atrial shunts are currently being widely used in the treatment of hypertensive hydrocephalus. This report describes the role of roentgenology in the placement of these shunts as well as in the follow-up of individuals after cerebral decompression. Several case illustrations are presented along with a review of the pertinent literature.

Sequelae and complications of cerebral ventricular decompression include overlap of cranial bones, premature closure of cranial sutures, thickening of the calvaria, subdural hematomata and acute pulmonary edema.

Complications following insertion of a catheter in the right atrium include thrombosis, pulmonary embolism, pulmonary hypertension and cor pulmonale, cardiac perforation, and bacterial colonization.

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# PNEUMOGRAPHIC DEMONSTRATION OF SUB-EPENDYMAL HETEROTOPIC CORTICAL GRAY MATTER IN CHILDREN*

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HETEROTOPIC cortical gray matter is occasionally pathologically demonstrable within the depths of the central white matter of the "normal" brain. Usually, this ectopic cortex consists merely of small nests of cells of microscopic proportions. Larger aggregates, varying from a millimeter to 2 or 3 cm. in size may occur, often coexisting with a constellation of other structural abnormalities of the brain. When these larger aggregates lie immediately beneath the ependyma, they deform the ventricular surface and present a characteristic appearance at cerebral pneumography. Though this pathologic entity has been well known for many years, review of the radiologic literature fails to reveal a description of its pneumographic identification. The importance lies in recognition. In the absence of other readily demonstrable pneumographic abnormalities, it may be regarded as an index of profound structural disorganization of the cortex. In the presence of other roentgen abnormalities, it stands as further testimony to a marked degree of maldevelopment of the brain. Importantly from a clinical standpoint, patients with gross subependymal heterotopias show an unfailing association with impaired psychomotor development.

The pneumographic appearance of heterotopic cerebral cortex should not be confused with that of tuberous sclerosis, or other subependymal pathologic processes. The purpose of the author is to report a case of this entity demonstrated pneumoencephalographically, as well as pathologically, and to discuss its implication, including the differential diagnosis. Four other cases, with the diagnosis based on clinical and pneumographic criteria, are also presented.

## REPORT OF A CASE

CASE 1. L.M. (Unit #614801), the product of the first pregnancy of a 22 year old mother, was born after 4 days of difficult labor. Head size was described as "large" at birth. At the age of 2 months, a clinical diagnosis of hydrocephalus was made by the family physician. Development was retarded. Air studies performed at the age of $5\frac{1}{2}$ months revealed agenesis of the corpus callosum, with generalized ventricular enlargement and apparent hypodevelopment of the brain stem and cerebellum. A mass in the atrium of the left lateral ventricle was interpreted as a papilloma of the choroid plexus (Fig. 1). While undergoing a craniotomy 5 days after the air study, the patient expired suddenly.

Autopsy revealed congenital malformations of the brain. These included: heterotopic cortical tissue in the walls of the lateral ventricles and in the island of Reil (right); agenesis of the corpus callosum; multiple intraleptomeningeal glial masses with incomplete obstruction of the outlet of the fourth ventricle; incomplete fusion of the neural tube at the level of the aqueduct of Sylvius; and internal hydrocephalus. The lateral ventricles were moderately dilated. The lateral surface of the left lateral ventricle just above the area of the caudate nucleus was nodular. The head of the right caudate nucleus was slightly uneven on its superior and medial surfaces. The lateral surface of the right lateral ventricle was quite nodular and irregular over an area extending from about the middle of the caudate nucleus to the atrium. The nodules formed one continuous irregular elevation and the largest heterotopic masses had coalesced, particularly in the atrial areas, and

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projected into the lateral ventricles. No abnormalities of the choroid plexuses were seen.

Microscopically, these subependymal nodules were composed of readily identifiable clusters of heterotopic cortical gray matter. The third ventricle, aqueduct of Sylvius and fourth ventricle exhibited no evidence of cortical heterotopia (Fig. 2, *A* and *B*).

*Comment.* This child had multiple congenital abnormalities of the brain in conjunction with marked subependymal cortical heterotopia. A large mass of gray matter, measuring approximately 2×3 cm., projecting in the atrial area of the left lateral ventricle, was misinterpreted at cerebral pneumography as representing a papilloma of the choroid plexus. Other nodular ventricular irregularities in the air study were left unexplained, until necropsy proved them to be subependymal heterotopia as well.

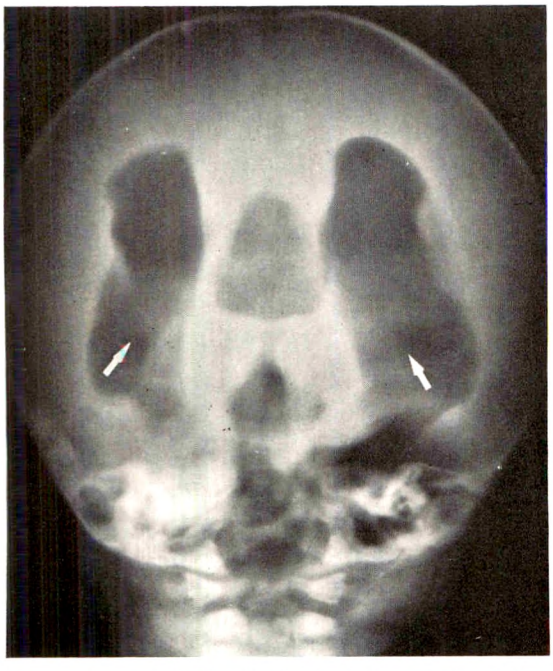


Fig. 1. Case 1. (Unit #614801). A 5½ month old boy admitted because of rapidly increasing head size since birth. Examination showed an infant with a wandering gaze, who rolled his head from side to side in his crib and was unable to hold it up. The ventriculogram made through the anterior fontanelle demonstrates nodular irregularities along the lateral ventricular walls. The atrial shadows resemble choroid plexus tumors (arrows). Generalized cerebral hypoplasia with agenesis of the corpus callosum is also demonstrated. At necropsy, all of the mural abnormalities were found to be caused by the presence of subependymal heterotopic gray matter




Fig. 2. (*A*) (Unit #290400). Coronal sections of the brain of an infant with multiple congenital anomalies who died 10 days after birth. Large subependymal cortical heterotopias project into the ventricles, giving the walls a bossed appearance. These nodular deformities are similar to those found at autopsy in the case shown in Figure 1. (*B*) (Unit #142768). Coronal section of the brain of a retarded infant with a seizure disorder and a coloboma of the right eye, who died at the age of 6 months. In addition to the mammilated subependymal projections in the roof and lateral aspect of the right lateral ventricle, there is a medially attached pedunculated heterotopia in the lumen. This is strikingly similar to the atrial heterotopias shown in Figure 1.

## Developmental Pathology and Associated Malformations of the Brain

In the embryologic development of the cerebrum, the neural tube is composed of three layers:

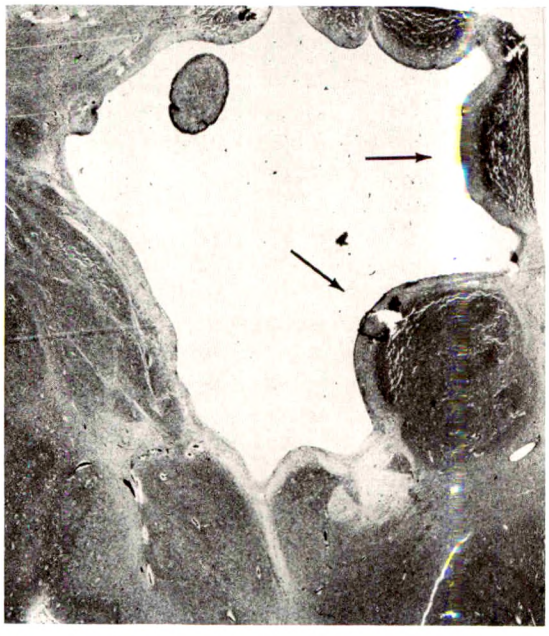


FIG. 3. (Unit #1706212). Low-power photomicrograph of lateral ventricle and surrounding tissue obtained from the brain of a 17 month old retarded child who had uncontrollable seizures and died of aspiration pneumonia. Hillocks of darkly staining heterotopic cortical gray matter covered by ependyma project into the lumen (arrows).

(a) The ependymal layer, which lines the lumen and becomes the ependyma of the adult nervous system.

(b) The mantle layer, the middle, bulky portion of the tube, which contains the germinal cells and the spongioblasts. Proliferation of the germinal cells provides neuroblasts which develop into the nerve cells of the central gray substance. Spongioblasts develop into the supporting elements, the neuroglia.

(c) The marginal layer, an acellular layer composed of cell processes from the inner two layers, which becomes the outer white substance when the fibers grow out from the mantle layer and accumulate at the periphery.

Beginning in the twelfth fetal week, neuroblasts destined to be part of the neopallial (nonolfactory) cortex migrate peripherally from the ependymal and mantle zones. They collect in the deeper part of the marginal zone, forming the gray matter of the cerebral cortex.[8,20]

Heterotopia of cortical gray matter is caused by migrational arrest of the affected neuroblasts as they move peripherally to the outer marginal (cortical) layer. Partial arrest leads to clumps of gray matter within the centrum semi-ovale. Total absence of migration results in aggregates of heterotopic cortical gray matter immediately beneath the ependyma. The tissue is nonfunctional, usually of an immature cell type. It is dysplastic (rather than neoplastic) tissue, and does not undergo malignant transformation (Fig. 3).

The presence of small microscopic areas of heterotopic cortex within the central white matter of the "normal" brain is an occasional finding on histologic study. It may be unaccompanied by any other cellular or structural abnormality. In patients with serious developmental abnormalities of the brain, however, it is a much more common finding. Norman[16] cites examples of heterotopia associated with agyria, pachygyria, microgyria, arhinencephaly, and microcephaly. Kernohan,[11] Kirschbaum,[12] and others[2,3] describe this as an accompaniment to abnormal brain development, such as micrencephaly, agenesis of the corpus callosum, congenital stenosis of the aqueduct of Sylvius, and the Arnold-Chiari malformation.

In experimental studies of prenatal roentgen irradiation of the rat, Cowen and Geller[3] have regularly seen sizable heterotopias in association with major architectural abnormalities of the brain. The association was particularly close in animals exhibiting micrencephaly, those having serious disorganization of cortical design, and those exhibiting total absence of the corpus callosum.

It is of interest that an early encephalographic study reported a patient with agenesis of the corpus callosum who, at autopsy, was found to have heterotopia of the occipital cortex, as well as complete agenesis of the corpus callosum, septum pellucidum and hippocampal commissure, microgyria, and anomalous development of the cerebellum.[11] It is perhaps worthy of note that while descriptions of sizable sub-

ependymal heterotopias of the lateral ventricles in patients with agenesis of the corpus callosum are scattered throughout the pathologic literature,[2,3,9,12] radiologic descriptions of this entity make no mention of it.

The postmortem findings of 10 patients with the diagnosis of subependymal heterotopia of cortical gray matter (examined by autopsy at the Columbia-Presbyterian Medical Center between 1940 and 1965) are summarized in Table 1. These cases are otherwise unselected and, in substance, illustrate what has been stated in reference to the association of cortical heterotopia with other congenital abnormalities. These include: (1) distortion of normal cortical architecture; possible microcephaly (Fig. 4*A*); (2) maldevelopment of the corpus callosum (Fig. 4*B*); (3) cerebellar anomalies (Fig. 4*A*); (4) stenosis of the aqueduct of Sylvius; and (5) high association of other bodily anomalies.

Importantly, from a clinical standpoint, it was found that of those patients who survived sufficiently long to be assessed for the attainment of developmental milestones, all showed moderate to severe psychomotor retardation.

In substance, then, it may be said that the presence of pneumographically conspicuous heterotopic subependymal cortical tissue is an index of profound developmental abnormality of the brain; the implication is direct, since sizable subependymal heterotopias do not otherwise occur. They are merely one manifestation of a severe (but nonlethal) insult sustained by the developing central nervous system of the fetus. The prognosis of all such patients is poor, insofar as normal psychomotor development is concerned, regardless of whether or not other structural anomalies are roentgenographically defined.

## OTHER MATERIAL

In light of the above findings, a review was undertaken of the pneumoencephalograms and ventriculograms of 60 patients who were coded in our files as having serious congenital anomalies of the brain. Nodular lateral ventricular irregularities were found in 4 instances; these cases fulfilled the criteria for the pneumographic identification of subependymal cortical heterotopias (to be defined) and are now presented.

## CASE SUMMARIES

Case II. K.L. (Unit #1693427), male, born at 32 weeks' gestation. Retarded development; sat at 1 year, walked at 20 months, talked at $2\frac{1}{2}$ years. A pneumoencephalogram at the age of 4 years showed aqueductal insufficiency and dilated lateral ventricles with nodular walls (Fig. 5). The patient had a ventriculo-atrial shunt; at the age of 4 years, 3 months, there was onset of grand mal seizures. Psychometric testing at the age of 4 years, 11 months, yielded a mental age of 3 years, 11 months and an I.Q. of 78.

Case III. J.H. (Unit #1050236), female with onset of seizures and increasing head size soon after birth. A pneumoencephalogram at the age of 18 months demonstrated agenesis of the corpus callosum and a large nodule projecting from the roof of the left lateral ventricle. Both atria were markedly dilated and nodular. The child (now 12 years old) has been followed and exhibits marked mental retardation with convulsions and paraparesis. A repeat pneumoencephalogram, made 11 years after the first, demonstrated no significant change (Fig. 6, *A* and *B*).

Case IV. P.R. (Unit #1493729), male, born at full term after difficult delivery, with onset of seizures at 4 months of age, and left hemiparesis at 20 months following a prolonged convulsive episode. At the age of 5 years, his I.Q. was 58. A pneumoencephalogram at the age of 22 months showed agenesis of the corpus callosum and multiple nodules in the posterior bodies of the lateral ventricles (Fig. 7).

Case V. J.C. (Unit #1660672), male infant, born with a "large head" after a prolonged labor because of cephalopelvic disproportion. Pregnancy had been complicated by a "viral illness" during the first trimester. At the age of 6 months, the child was admitted with the diagnosis of psychomotor retardation, failure to thrive, and an enlarged heart. A pneumoencephalogram at that time demonstrated agenesis of the corpus callosum, probable cortical hypoplasia, nodular lateral ventricles, hypoplastic cerebellum and a huge cisterna magna (Fig. 8). At the age of 26 months, he continued to show moderate retardation.

## TABLE I

AUTOPSY FINDINGS IN 10 PATIENTS WITH SUBEPENDYMAL HETEROTOPIC CORTICAL GRAY MATTER
COLUMBIA-PRESBYTERIAN MEDICAL CENTER, 1940–1965

| Case | Heterotopia | Corpus Callosum | Cerebral Anomalies | III Ventricle Aqueduct of Sylvius IV Ventricle | Hydrocephalus | Cerebellar Abnormalities | Other Congenital Abnormalities |
|---|---|---|---|---|---|---|---|
| C.S.<br>#790889<br>A#7529<br>8 yr. F | Subependymal of anterior horn of left lateral ventricle | No autopsy comment; presumably normal | Encephalomalacia secondary to infarcts related to cardiac surgery | Normal | No | None | Tetralogy of Fallot; malrotation of small and large intestine; omphalocele |
| B.G.S.<br>#122-94-68<br>A#8236<br>3 wk. F | Scattered small masses throughout white matter; lateral ventricle walls "sprinkled" with subependymal masses | Absent anterior commissure; corpus callosum thinned throughout to 1 to 2 mm. | Intraleptomeningeal glial masses | III slit-like, malformed; aqueduct absent; IV tapers to blind end | Obstructive | No gross abnormality | Imperforate anus; fused kidney; cleft palate; multiple skeletal, cardiac and gastrointestinal malformations |
| T.J.<br>#915559<br>A#7192<br>14 mo. F | Multiple nodules of lateral wall, rt. posterior horn, atrium, anterior roof of lt. occipital horn; largest nodule 7.5 ×17.5 mm. | No autopsy comment; presumably normal | Microgyria, with loss of normal cortical pattern | Normal | Obstructive | Asymmetric hemispheres with incomplete separation | Arnold-Chiari; spina bifida; hemivertebrae; meningomyelocele of thoracolumbar area; patent foramen ovale |
| S.S.<br>#458795<br>A#12450<br>21 mo. F | Multiple nodules of both posterior horns; lateral ventricles varying from 2 to 20 mm. in size | Absent septum pellucidum; corpus callosum attenuated to paper thinness | Microgyria | III, IV normal; stenosis of mid-portion of aqueduct | Obstructive | Hypoplasia of right cerebellar hemisphere | None |
| R.M.<br>#143-67-90<br>A#9130<br>6 mo. F | 6 nodules of rt. lateral ventricle, 3 nodules of lt. lateral ventricle, varying from 2 to 8 mm. in diameter, projecting 2 mm. on surface | Thinned to semitransparent membrane; septum pellucidum thinned and fenestrated | Microgyria of rt. greater than lt.; sulci disorganized; intraleptomeningeal masses | III normal; aqueduct narrow at level of intercollicular plate; IV elongated, flattened | Obstructive | Markedly deformed and hypoplastic | Deformed corpora quadrigemina; Arnold-Chiari; hydromyelia; spina bifida; meningomyelocele of lower dorsal lumbar area |
| C.M.<br>#142-76-80<br>A#9071<br>6 mo. F | Multiple nodules of lateral walls and roofs of both lateral ventricles from 2 to 6 mm. scattered nodules in white matter | Absent splenium; large cavum septi pellucidi anteriorly | Microgyria with disorganization of sulci; enlargement of frontal horn of lt. lateral ventricle; intraleptomeningeal glial masses | Normal | No | Absence of flocculus of right hemisphere | Patent foramen ovale; mobile cecum; accessory adrenals; small cerebral peduncles; coloboma of right eye |
| L.M.<br>#614801<br>A#13465<br>6 mo. M | Multiple nodules of both lateral ventricles, 4 to 8 mm. in size; scattered nodules in right island of Reil | Complete agenesis | Inequality of some lobes of frontal cortex | III normal; incomplete fusion of neural tube at aqueduct; partial obstruction of IV at outlet by glial masses | Obstructive | Normal | None |
| M.S.<br>#170-62-12<br>A#10127<br>17 mo. F | Multiple nodules of both lateral ventricles, and in central white matter; interhemispheric fissures between gyri recti | Complete agenesis of corpus callosum and cingulate gyri | Microgyria, focal of lt. superior and frontal gyri | Cysts of choroid plexus of III | No | Asymmetric lobes | Cysts of choroid plexus and roof of left temporal horn |
| S.M.S.<br>#290400<br>A#8455<br>6½ da. F | Small rounded nodules forming clusters, especially about bodies of lateral ventricles and atria, bilaterally; few nodules also in frontal horns | Septum pellucidum thinned and fenestrated | Microgyria and extreme cortical disorganization | III normal; aqueduct and IV small, but patent | Mild; obstructive | Herniated, but otherwise normal | Arnold-Chiari; multiple congenital anomalies of genitourinary, gastrointestinal and musculo-skeletal systems; teratoma of dorsolumbar spine |
| B.T.B.<br>#162-50-85<br>A#9768<br>3 da. | Frontal horn of lt. lateral ventricle studded with 2 mm. nodules, as was entire rt. lateral ventricular wall | Complete agenesis | Microcephaly; pachygyria; leptomeningeal glial masses | Normal | None | Normal | Parasitic twin of 2 infants joined at abdomen ("Siamese"); multiple anomalies; other twin had normal brain at autopsy |

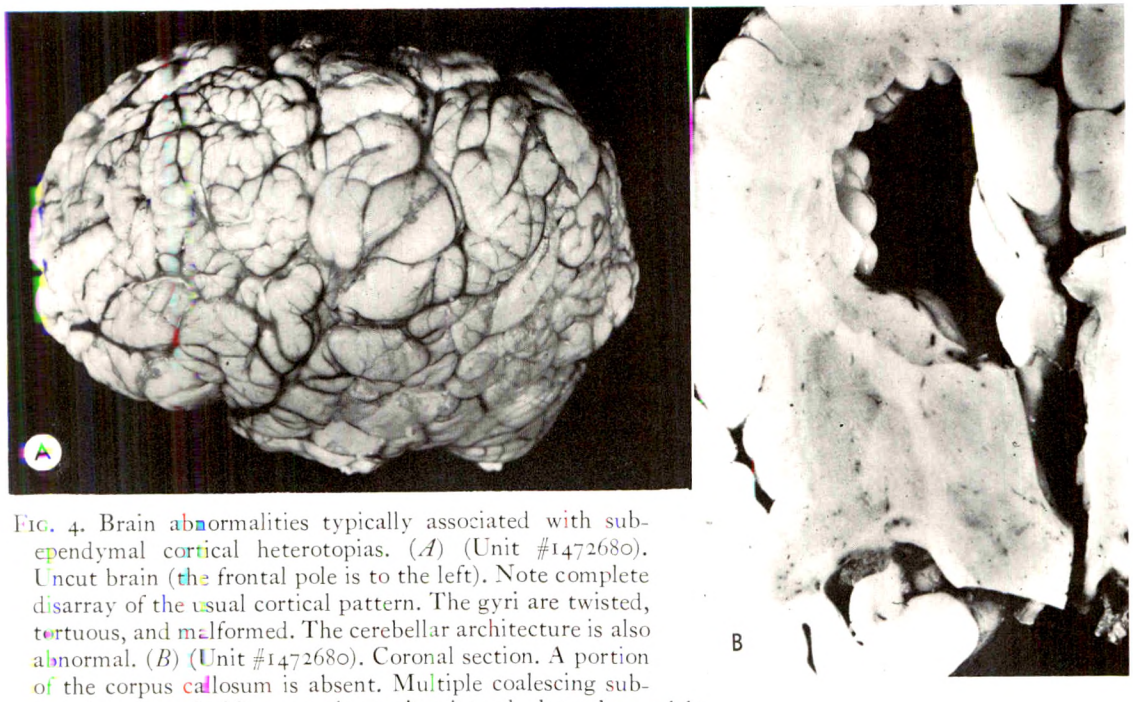



Fig. 4. Brain abnormalities typically associated with subependymal cortical heterotopias. (*A*) (Unit #1472680). Uncut brain (the frontal pole is to the left). Note complete disarray of the usual cortical pattern. The gyri are twisted, tortuous, and malformed. The cerebellar architecture is also abnormal. (*B*) (Unit #1472680). Coronal section. A portion of the corpus callosum is absent. Multiple coalescing subependymal cortical heterotopias project into the lateral ventricle.

*Comment.* All 4 patients were children with psychomotor retardation; none had evidence of other disease states with which the roentgen identification of subependymal cortical heterotopia might possibly be confused (tuberous sclerosis, choroid plexus papilloma, etc.). Despite many pathologic reports of subependymal cortical heterotopia found in congenitally malformed brains of adults, none was discovered in a review of our own pneumographic material in that age-group. This supports the contention that roentgenographically definable heterotopias are found in severe developmental abnormalities which usually necessitate study early in life.

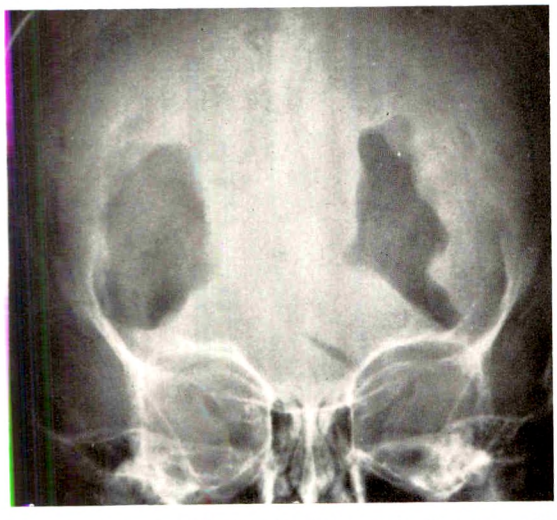


Fig. 5. Case 11. K.L. (Unit #1693427). A 4 year old retarded boy admitted for evaluation of gradually increasing head size. The pneumoencephalogram shows aqueduct stenosis, supra-aqueductal ventricular dilatation, and multiple nodulations of the atrial areas bilaterally. The nodules are broad-based, involve the lateral walls chiefly, and contain no calcium. The third ventricle was not affected.

## DISCUSSION

### ROENTGEN FINDINGS

Roentgenographically, subependymal heterotopic cortical gray matter will appear as varying sized nodular irregularities within the walls of the air-filled lateral ventricles. Involvement is confined exclusively to the lateral ventricles, and to no other portions of the ventricular system. The cerebral cortex is developmentally a forebrain phenomenon and the presence of cortical heterotopia in the walls of the third ventricle, aqueduct of Sylvius, or fourth

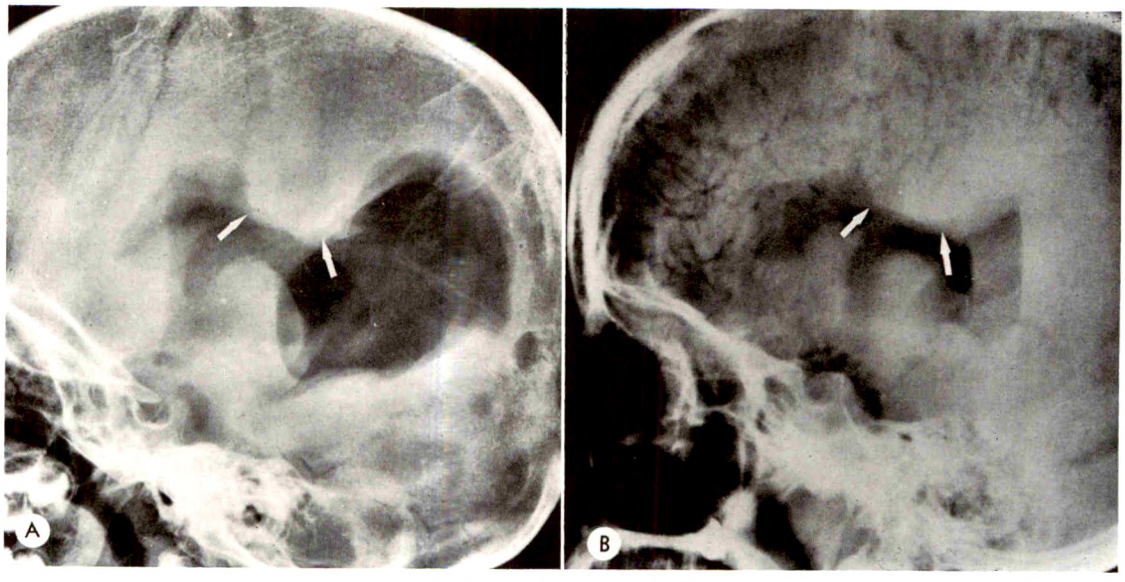



FIG. 6. Case III. (Unit #1050236). An 18 month old retarded girl admitted for evaluation of seizures and a large head. The pneumoencephalogram shows a single, large nodule projecting down from the roof of the left lateral ventricle (arrows). Both atrial regions are markedly dilated and irregular; agenesis of the corpus callosum is also present. (*B*) Follow-up pneumoencephalogram made 11 years after the first. The child continued to exhibit marked mental retardation and had uncontrollable seizures and spastic paraparesis. The nodule in the roof of the lateral ventricle has not changed appreciably in size (arrows). There is no intracranial calcification.

ventricle should be regarded as an ontogenic impossibility.

Heterotopias are more commonly identified in the frontal horns, bodies, and posterior horns of the lateral ventricles than in the temporal horns. The nodules are solid masses and may vary in size from 1–2 mm. (the smallest size likely to be recognizable at cerebral pneumography) to about 2 cm. or 3 cm. in diameter. Large aggregates may appear as variegated hillocks, others as "cobble-stone" clusters, completely replacing the normal smooth-walled appearance of the affected ventricular surfaces, giving them a mammilated or bossed appearance. Other aggregates may project far into the ventricular lumen, forming a broad-based pedunculated mass. In serial examination the nodules will never show increase in number or size. Calcifications within cortical heterotopias have not been reported.

Heterotopias are likely to be demonstrated in association with other anatomic abnormalities, such as agenesis of the cor-

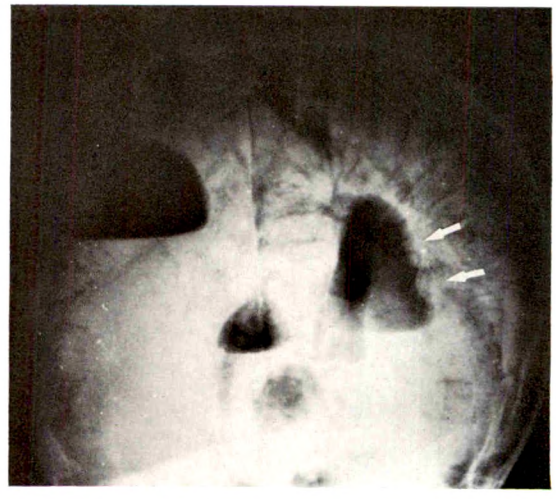


FIG. 7. Case IV. P.R. (Unit #1493729). A retarded 22 month old boy admitted for evaluation of a convulsive disorder since the age of 4 months and hemiparesis since the age of 20 months. The pneumoencephalogram demonstrates nodular irregularities in the atrium and posterior temporal horn on the left (arrows). The ventricles are dilated and agenesis of the corpus callosum is present. This is a common constellation of brain abnormalities seen with heterotopic cortical gray matter.

pus callosum (complete or incomplete), microcephaly, other cortical dysmorphoses (agyria, microgyria, pachygyria), congenital aqueduct stenosis, and cerebellar dysgenesis. Ventricular enlargement, especially marked in the atrial areas and posterior horns, may be prominent.

DIFFERENTIAL DIAGNOSIS

A. *Tuberous Sclerosis.* The key-stone of roentgen diagnosis of tuberous sclerosis lies in recognition of scattered nodular calcifications throughout the brain. In addition, subependymal nodules of this condition frequently calcify. Nodules of tuberous sclerosis are commonly seen in the walls of the third ventricle as well as in the lateral ventricles (Fig. 9). While ventricular dilatation (nonobstructive hydrocephalus) is commonly seen in tuberous sclerosis, other structural anomalies of the varieties frequently seen with heterotopias are rare. In addition, there is usually readily documented clinical evidence of this disease.[7]

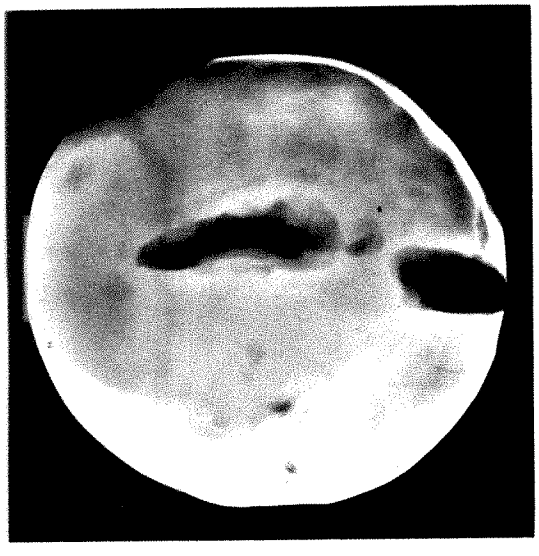


Fig. 8. Case v. J.C. (Unit #1660672). A 6 month old boy admitted with the diagnosis of psychomotor retardation, large head, failure to thrive, and an enlarged heart. The pneumoencephalogram shows characteristic mammilations of subependymal cortical heterotopias, projecting down from the roofs of the dilated lateral ventricles. The posteriorly placed air shadow is within a huge cisterna magna; the cerebellum is hypoplastic. Cortical hypoplasia and agenesis of the corpus callosum are also present.

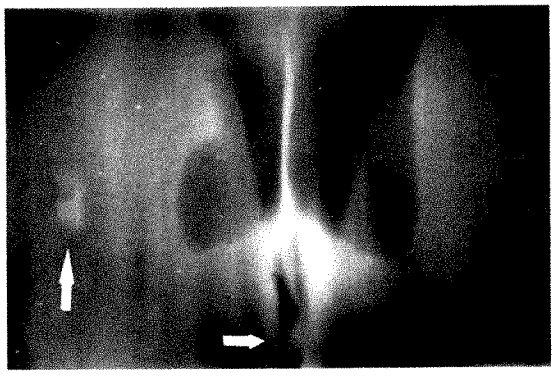


Fig. 9. Pneumoencephalogram with tomographic section (brow-up projection) of a young girl with tuberous sclerosis. Noncalcified nodular densities may be seen in the right frontal horn laterally. The third ventricle exhibits a nodule on the left (horizontal arrow). Calcification is demonstrated within the brain substance on the right (vertical arrow). In contrast to tuberous sclerosis, the nodules of heterotopic cortical gray matter do not calcify and do not involve the third ventricle.

B. *Papilloma of the Choroid Plexus.* In the pediatric age group, papillomas of the choroid plexus occur almost exclusively in the lateral ventricles. Their attachment is medial (in the choroid plexus), usually in the atrial region, involving the glomus. Uncommonly, light adhesions may form additional attachments to the ependyma elsewhere.[15] Oversecretion of cerebrospinal fluid causes generalized ventricular dilatation. This entity may constitute a difficult differential diagnostic problem in those cases of pedunculated heterotopias occurring in the atrial areas. Other sometimes helpful roentgen signs in the diagnosis of choroid plexus papilloma (bulky mass with possible partial lateral ventricular obstruction and distortion, midline shift) and clinical measurements (enlargement of the head, papilledema, markedly elevated cerebrospinal fluid pressure, elevated cerebrospinal fluid protein, with possible xanthochromia) may help to distinguish the entities.

The tumor is rarely malignant; when malignant metaplasia occurs,[17] ependymal seeding within the entire ventricular system may be observed.

A difficult diagnostic problem may also occur with a normal but conspicuous glomus body occurring in the presence of obstructive hydrocephalus. In light of these possibilities, it is hazardous to make the diagnosis of heterotopia when the ventricular irregularity is confined to the medial aspect of the atrial region alone.

C. *Ependymal Seeding from Other Malignant Tumors of the Central Nervous System.* Ependymal seeding is, on occasion, seen with ependymoma, medulloblastoma and glioblastoma of the cerebrum. In these cases, if the primary lesion is not obvious from both the clinical course and roentgen findings of the parent tumor, it may be noted that hydrocephalus, if present, is of the rapidly progressive obstructive type. On serial air studies the nodules will increase in size and number. Occasionally, these seedings calcify.

D. *Granular Ependymitis.* Inflammation may produce tiny nodules on the ventricular walls. It commonly is the result of either an infectious process or a subependymal vascular accident with ventricular bleeding, and the patient's clinical course is that of an acute illness with a subsequent progressive obstructive hydrocephalus. These nodules are unlikely to reach more than 1 mm. in size and commonly involve the aqueduct of Sylvius.

E. *Ribbing of the Ventricular Walls in Hydrocephalus.* As described by Taveras and Wood,[19] irregularities of the walls of the dilated ventricles due to variation of elasticity in supportive structures (vessels, fiber tracts, nuclei, etc.) are non-nodular in appearance and usually are seen only with very marked obstructive hydrocephalus. The third ventricle usually shares in the ventricular irregularity.

F. *Irregularities in the Roofs of the Lateral Ventricles due to "Coarctation" of the Lateral Ventricles or Due to Callosal Radiations.* With coarctation of the lateral ventricles, the lateral projection of the brain may reveal at pneumography one or more small nodular projections from the roof of the lateral ventricle (usually anterior to the interventricular foramen). The affected ventricle is smaller than its mate in the frontal projection, and has a truncated appearance. As elucidated by Davidoff,[4] this condition is the result of the formation of ependymal adhesions in the superiolateral aspect of the roofs of the lateral ventricles during fetal development. It is regarded as a developmental anomaly rather than the result of an inflammatory process.

Irregularities of the roofs and upper lateral aspects of the lateral ventricles may also be caused by radiations of the various bundles of the corpus callosum, as originally described by Lindgren.[14] The appearance is characteristic, and often bilaterally symmetric. Usually, these deformities, as with coarctation, are confined to the lateral ventricles anterior to the foramen of Monro.

G. *Irregularities of the Floors of the Frontal Horns Due to the Head of the Caudate Nucleus.* A nodular appearing defect is occasionally seen in the floors of the frontal horns at cerebral pneumography, anterior to the interventricular foramen. This defect is oftentimes symmetric within both ventricles and marks the anteriormedial aspect of the caudate nucleus as it impinges on the floor of the frontal horns. Unless frontal tomograms are obtained, these defects may be seen in the lateral projections alone, because air in the frontal horns anterior to this level usually obscures their identity in the frontal projection.

## SUMMARY

Heterotopic cortical gray matter occurs as a consequence of migrational arrest of primitive nerve cells (neuroblasts) in the developing fetal cerebrum. In the mature brain, these heterotopias may be subependymal; they deform the lateral ventricular surfaces and are demonstrable at cerebral pneumography.

Ten cases with autopsy findings are reviewed. An additional 4 patients are presented in whom the diagnosis is based on pneumographic and clinical criteria. The pneumographic features of subependymal cortical heterotopias include the following:

1. Nodular lateral ventricular irregularities; these are solid masses and may be

single or multiple; their attachment is usually broad based. Commonly, a large area is involved, giving the ventricular wall a mammilated appearance. The third ventricle, aqueduct of Sylvius and fourth ventricle are never involved.

2. The nodules do not calcify.

3. The nodules do not change in number or size on serial studies.

4. The nodules commonly occur in the presence of other brain abnormalities, such as agenesis of the corpus callosum, microcephaly, congenital aqueduct stenosis and cerebellar dysgenesis.

The differential diagnosis includes tuberous sclerosis, choroid plexus papilloma, ependymal seeding from malignant tumors, as well as anatomic variants.

Pneumographically demonstrable subependymal cortical heterotopias may be regarded as an index of significant insult sustained by the developing brain. Their presence correlates uniformly with retarded psychomotor development. The diagnosis in infancy or childhood is a warning that difficulties may be anticipated in motor and intellectual achievement.

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# THE NEURORADIOLOGY OF MELANOMAS OF THE CENTRAL NERVOUS SYSTEM*

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THE primary site of origin of cerebral metastases cannot be determined by means of carotid angiography according to Zachrisson.[23] Other authors dealing with this problem have arrived at the same conclusion.[21] Taveras and Wood,[19] however, in 1964, mentioned that metastatic deposits from the kidneys and thyroid may "tend to have a slightly more homogeneous distribution of abnormal vascularity on angiography than tumors originating in the lungs or in the breast." The roentgen manifestations of malignant melanoma were discussed by Hilbish,[10] in 1957. He described a case of intracerebral melanoma in the left temporal lobe diagnosed on pneumoencephalography. Angiography was not carried out, however.

We recently investigated a case of a metastatic cerebral melanoma in which the neuroradiologic features simulated those of a meningioma. In reviewing the case histories and neuroradiologic findings of 28 proven cases of melanomas of the central nervous system, certain clinical and radiologic features became apparent to us which, when present, suggested the diagnosis. A review of this material is the subject of our paper.

## MATERIAL

Twenty-eight cases of melanomas of the central nervous system compiled from the Radiologic Services of the Bronx Municipal Hospital Center, Montefiore Hospital and Medical Center, and St. Vincent's Hospital and Medical Center in New York were included in this study. The roentgenologic and clinical features are shown in Tables I–IV.

## DISCUSSION

About 18 per cent of cerebral neoplasms are metastatic according to Baker;[3] however, only 5–10 per cent of these originate from skin melanoma.[4] According to Levit,[13] malignant melanoma has an annual incidence of about 1.8 per 100,000 and nearly one-half of these metastasize to the brain.[2,9] The roentgenologic features in 24 of our cases in which angiography was carried out were those of avascular space occupying lesions in 14 cases, and vascular lesions in 7 cases (Table I). In 2 of the vascular lesions the appearances simulated a meningioma, in that there was an external carotid supply and a homogeneous blush was seen from the arterial through the venous phases of the angiogram (Fig. 1, *A–C*; 2, *A* and *B*). While supply from the external carotid circulation is usually considered characteristic of meningiomas, it can be seen in both primary intracranial tumors such as glioblastoma multiforme and metastases from any source. The location of the tumor appears to be the deciding factor as to whether it will derive its blood supply from the external or internal circulation. According to Courville and Schillinger,[5] who surveyed 18 cases of metastatic melanoblastomas of the brain and reviewed the literature, melanoblastomas have a predisposition to localize in the cortical gray matter or at the border of the gray and white substance. Similarly, Weller[22] in a case review of melanotic metastases described numerous nod-

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TABLE I

SUMMARY OF ROENTGENOLOGIC FINDINGS IN OUR 28 CASES

| Procedure | Findings |
|---|---|
| Angiography—22 cases: 9 Bilateral carotid angiograms<br>15 Unilateral carotid angiograms<br>3 Vertebral angiograms<br>(Including 10 cases with air studies in which displacements were seen in 7—4 were supratentorial lesions and 3 were infratentorial lesions) | Vascular tumors—7 cases (2 with external carotid supply and blush simulating meningioma; 5 with nonspecific features of a malignant lesion such as arteriovenous shunting, abnormal vessels, and tumor stain)<br>Avascular tumors—14* cases (shown as vessel displacements only)<br>Normal—3† cases |
| Pneumoencephalography without angiography—1 case | Flattening of the occipital horn—1 case |
| Myelography—2 cases | Extradural lesion—1 case<br>Intramedullary and extramedullary, intradural—1 case |
| Skull roentgenography only—1 case | Multiple osteolytic lesions in the calvarium—1 case |

* Includes 2 vertebral angiograms.
† Includes 1 vertebral angiogram.

ules lying cortically and subcortically. With growth of these metastases and their rupture through the cortex, cortical softening would allow contact to occur between the metastases and the leptomeninges. This mechanism might promote a blood supply from the meningeal vessels and could afford a ready explanation for our cases simulating meningiomas. The pattern of vascularity in the other vascular cases is well demonstrated in Figure 3, *A–E*, where multiple lesions are seen to be present in both cerebral hemispheres in a 42 year old female.

TABLE II

SEX AND AGE DISTRIBUTION IN THE 28 CASES

15 Male patients
13 Female patients

| Age Groups (yr.) | |
|---|---|
| 21–30 | 1 case |
| 31–40 | 3 cases |
| 41–50 | 15 cases |
| 51–60 | 4 cases |
| 61–70 | 4 cases |
| 71– | 1 case |

The proximity of vascular melanotic metastases to the surface of the brain would also account for the large percentage of our cases having subarachnoid hemorrhages (Table III). Madonick and Savitsky[14] reviewed the subject of subarachnoid hemorrhages in cerebral melanomas in 1951, and found 26 cases of bloody or xanthochromic spinal fluid in a total of 56 cases of metastatic melanomas. In a control series of 56 cerebral metastatic tumors from origins

TABLE III

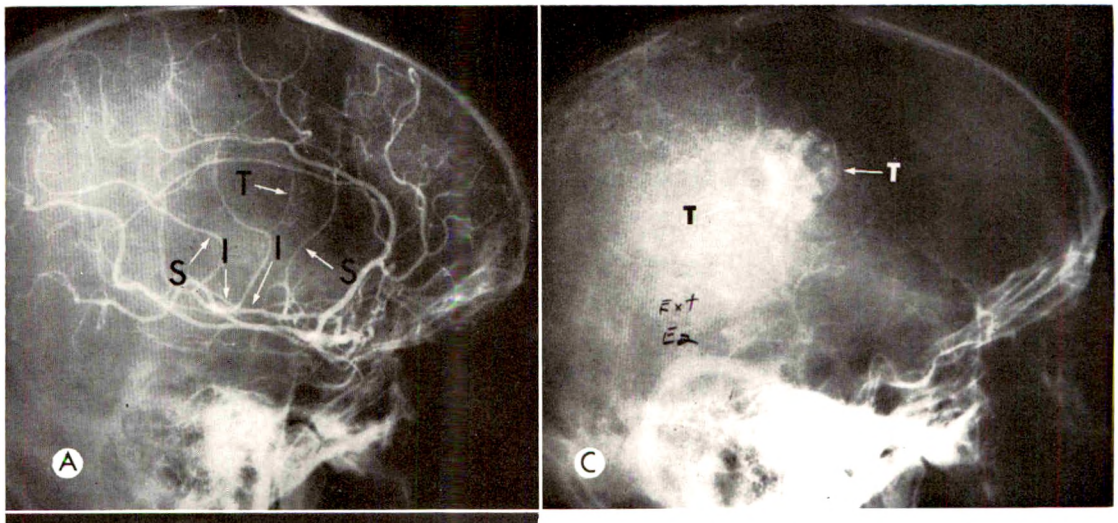
INCIDENCE OF SUBARACHNOID HEMORRHAGES

| | |
|---|---|
| No. of cases in which lumbar puncture was performed | 21 |
| Subarachnoid hemorrhages | 14 |

TABLE IV

SITES OF THE PRIMARY LESIONS

| | |
|---|---|
| Skin | 19 cases |
| Vagina | 1 case |
| Testis | 1 case |
| Eye | 1 case |
| No known primary | 6 cases |



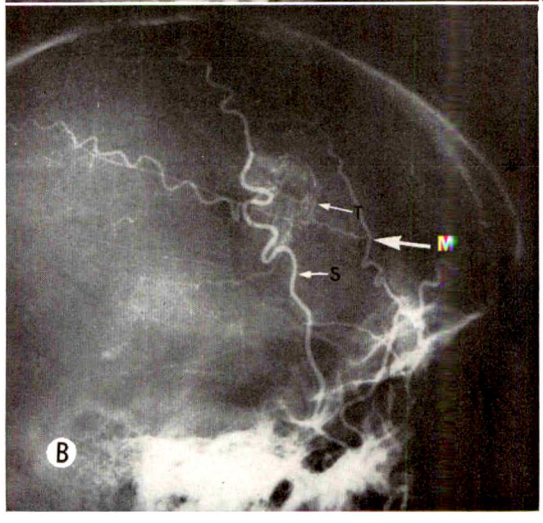




FIG. 1. Case 1. A 49 year old patient with melanomas on the trunk developed a sudden weakness of the left side of his body. Right carotid angiography demonstrated changes simulating a meningioma. An intracerebral melanoma was found at surgery. (*A*) Right lateral percutaneous internal carotid angiogram demonstrates depression of the insular branches, I, and stretching of the suprainsular branches of the middle cerebral artery, S. A faint tumor stain, T, is also seen supplied by one of the suprainsular branches. (*B*) Selective external carotid angiogram demonstrates superficial temporal artery, S, with moderately intense tumor stain, T. The middle meningeal artery, M, also supplies the tumor. (*C*) In the early venous phase a more intense tumor stain suggestive of a meningioma is now seen, T.

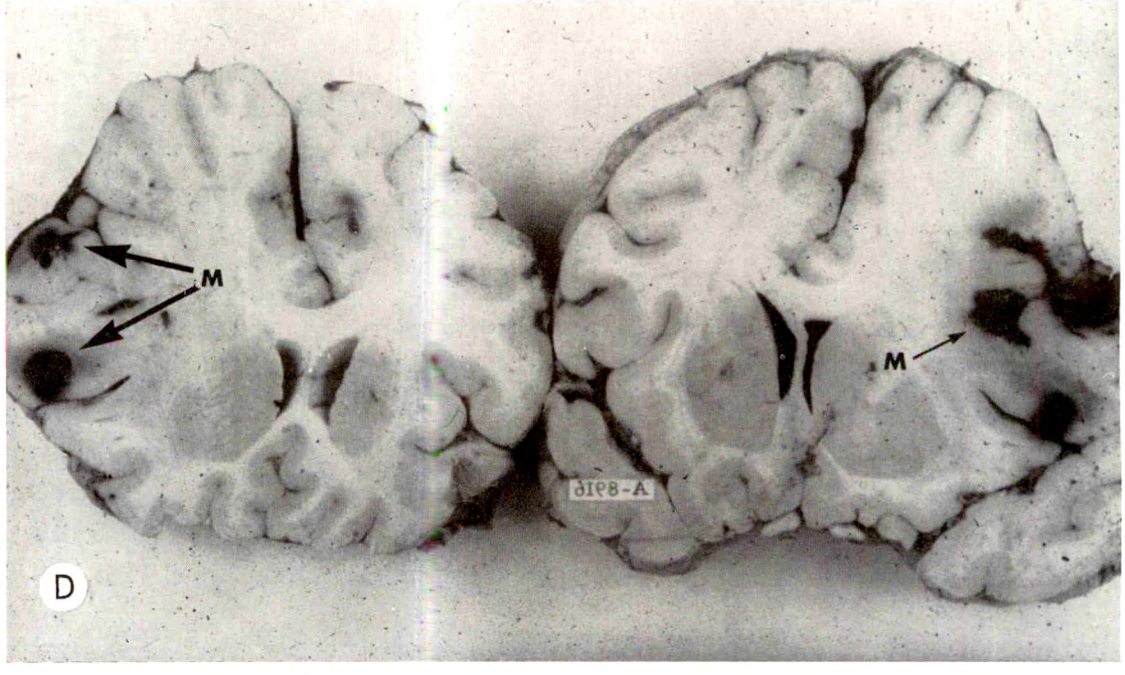



FIG. 1. Case 1. (*D*) Brain sections in the coronal plane demonstrate the melanotic lesion, M, adjacent to the surface of the brain.

other than melanomas only 2 cases of bloody or xanthochromic fluid were found.

The tendency for metastatic melanomas of the central nervous system to be associated with spontaneous hemorrhage is demonstrated in Figure 4, *A–F*. Here, over a period of 1 month, a marked increase in size of an avascular lesion was demonstrated. The increase in size was due to a hemorrhage into the lesion, as was proven by the surgical report of a large hematoma overlying a small melanoma implant.

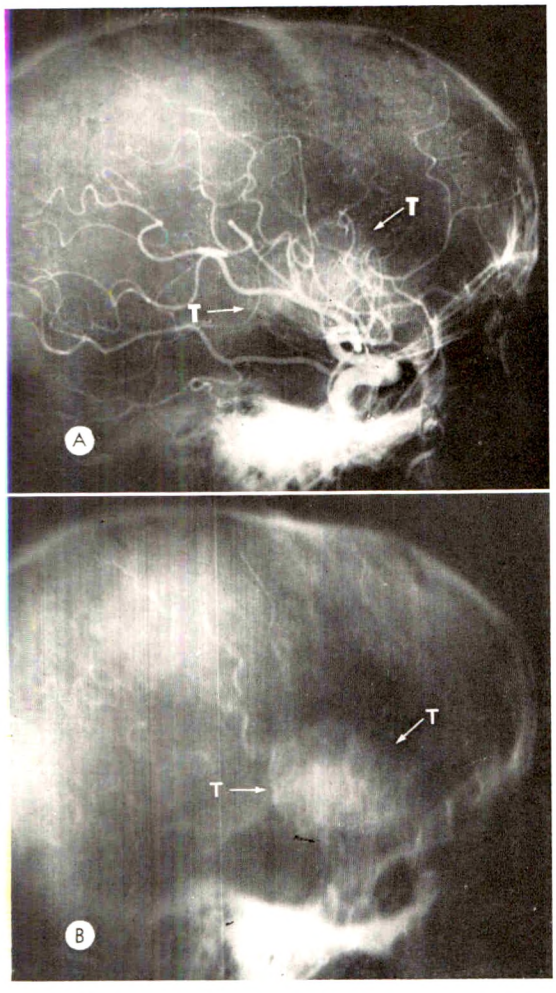



FIG. 2. Case 11. A 66 year old patient with a nevus in the region of the right eye and side of face developed personality changes 10 months before admission. She was admitted in a semistuporous state. On electroencephalography a slow wave focus was present in the right frontal region. Right carotid angiography demonstrated changes simulating a meningioma. An intracerebral melanoma was found at surgery. (*A*) Right lateral percutaneous common carotid angiogram demonstrates tumor stain, T, in the posteroinferior aspect of the frontal lobe. (*B*) In the early venous phase the tumor stain, T, is more intense, and is suggestive of a meningioma blush.

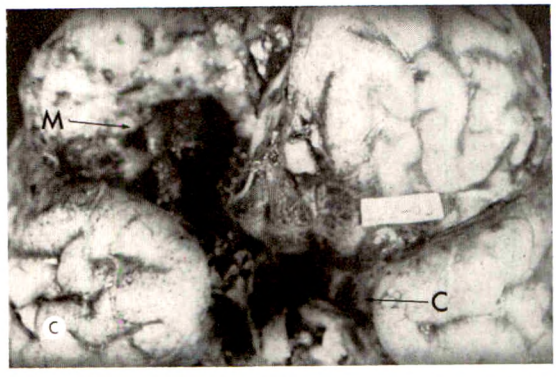



FIG. 2. Case 11. (*C*) Postmortem appearance of the base of the brain demonstrating the melanotic deposits involving the meninges in the chiasmatic cistern, C, with extension forward and burrowing into the base of the right frontal lobe, M. (Reproduced with permission of Dr. J. Gomez, Professor of Neurosurgery, University of Javeriana, Bogotá, Colombia.)

Metastatic melanotic deposits to the brain can manifest themselves as a diffuse involvement of the meninges in addition to discrete tumor masses in the brain. This is exemplified in the case of a 48 year old male with a melanotic skin lesion at the angle of the mandible. After he developed signs of a subarachnoid hemorrhage, angiography was carried out. Enlarged ventricles were demonstrated. At postmortem examination, in addition to metastatic lesions of the meninges and lobes of the brain, the ventricles were seen to be enlarged presumably on the basis of a communicating hydrocephalus (Fig. 5, *A–C*).

Brain scanning utilizing $Hg^{197}$ and $Hg^{203}$ was carried out in 3 cases, all of which were positive. In 2 of these, the lesion was well circumscribed (Fig. 6, *A* and *B*). In the other case the lesion was poorly circumscribed.

Bone metastases to the calvarium from metastatic melanoma have infrequently been described. According to Hilbish,[10] bone metastases in metastatic melanomas

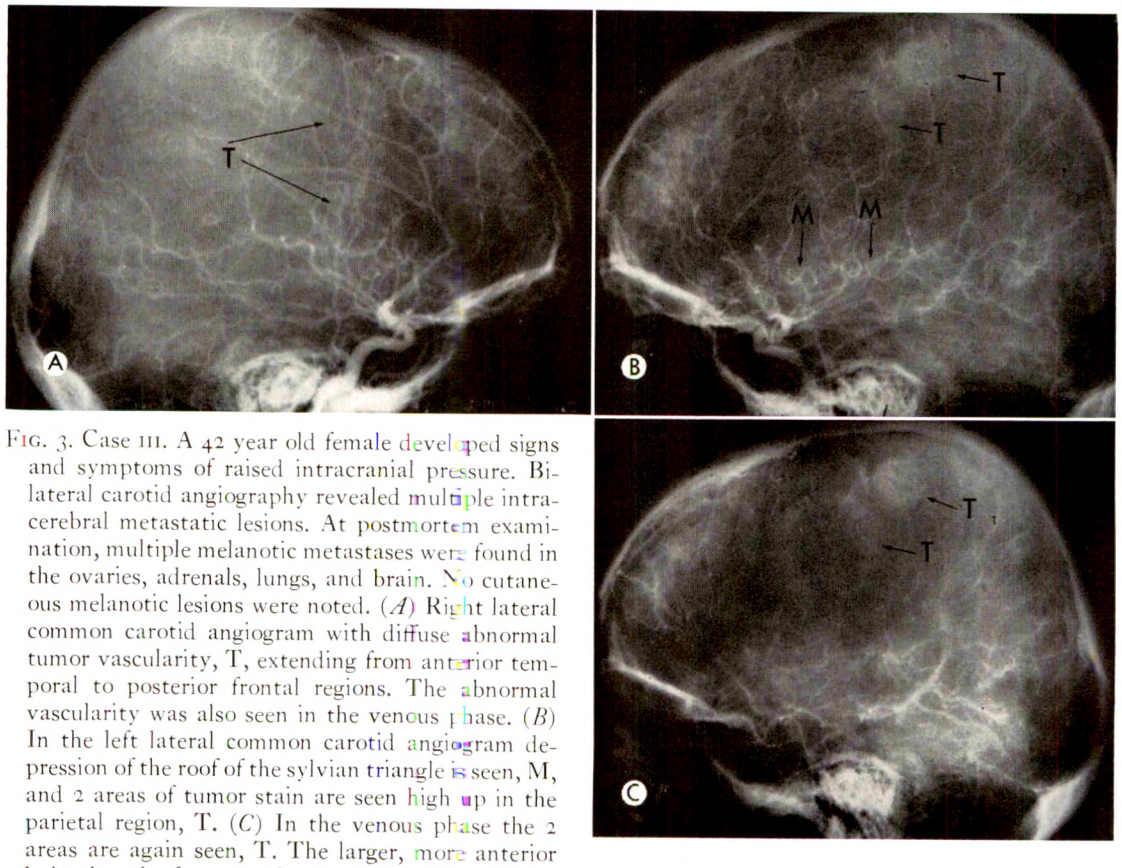



FIG. 3. Case III. A 42 year old female developed signs and symptoms of raised intracranial pressure. Bilateral carotid angiography revealed multiple intracerebral metastatic lesions. At postmortem examination, multiple melanotic metastases were found in the ovaries, adrenals, lungs, and brain. No cutaneous melanotic lesions were noted. (*A*) Right lateral common carotid angiogram with diffuse abnormal tumor vascularity, T, extending from anterior temporal to posterior frontal regions. The abnormal vascularity was also seen in the venous phase. (*B*) In the left lateral common carotid angiogram depression of the roof of the sylvian triangle is seen, M, and 2 areas of tumor stain are seen high up in the parietal region, T. (*C*) In the venous phase the 2 areas are again seen, T. The larger, more anterior lesion has the features of a ring stain with a relative avascular center.

only occur in 10 per cent of cases. In one of our cases, a 45 year old man with a melanoma in the left scapular region, a roentgenogram of the skull revealed multiple small well circumscribed lesions in the calvarium (Fig. 7).

In a series of 557 intraspinal tumors at the Mayo Clinic reviewed by Rasmussen and his co-workers[16] in 1940, 64 intramedullary tumors were found of which 3 were melanomas. In a series of 297 intraspinal tumors investigated at the Lahey Clinic and reviewed by Lang and Bridge[12] in 1959, 30 intramedullary tumors were found of which only 1 was a melanoma. The subject of spinal canal melanomas was investigated by Hirano and Carton,[11] in 1960. They reviewed 26 cases from the literature and found that 8 were intramedullary, 3 intra- and extramedullary, 10 extramedullary, but intradural, and 5 were purely extradural. They found that spinal cord metastases were far less common than cerebral metastases.

Two examples of spinal cord melanomas are included in our series. Both were investigated by myelography. In one, an epidural compressive lesion was present and in the other, a complete block to the passage of contrast material was noted at the level of D10. The lesion appeared to be extramedullary in the anteroposterior projection, but intramedullary in the lateral projection. At surgery the lesion was both intra- and extramedullary with erosion of the intervening dura. The patient had no peripheral cutaneous melanoma and at autopsy, no primary melanotic lesions were found in spite of a complete examination including microscopic study of all possible

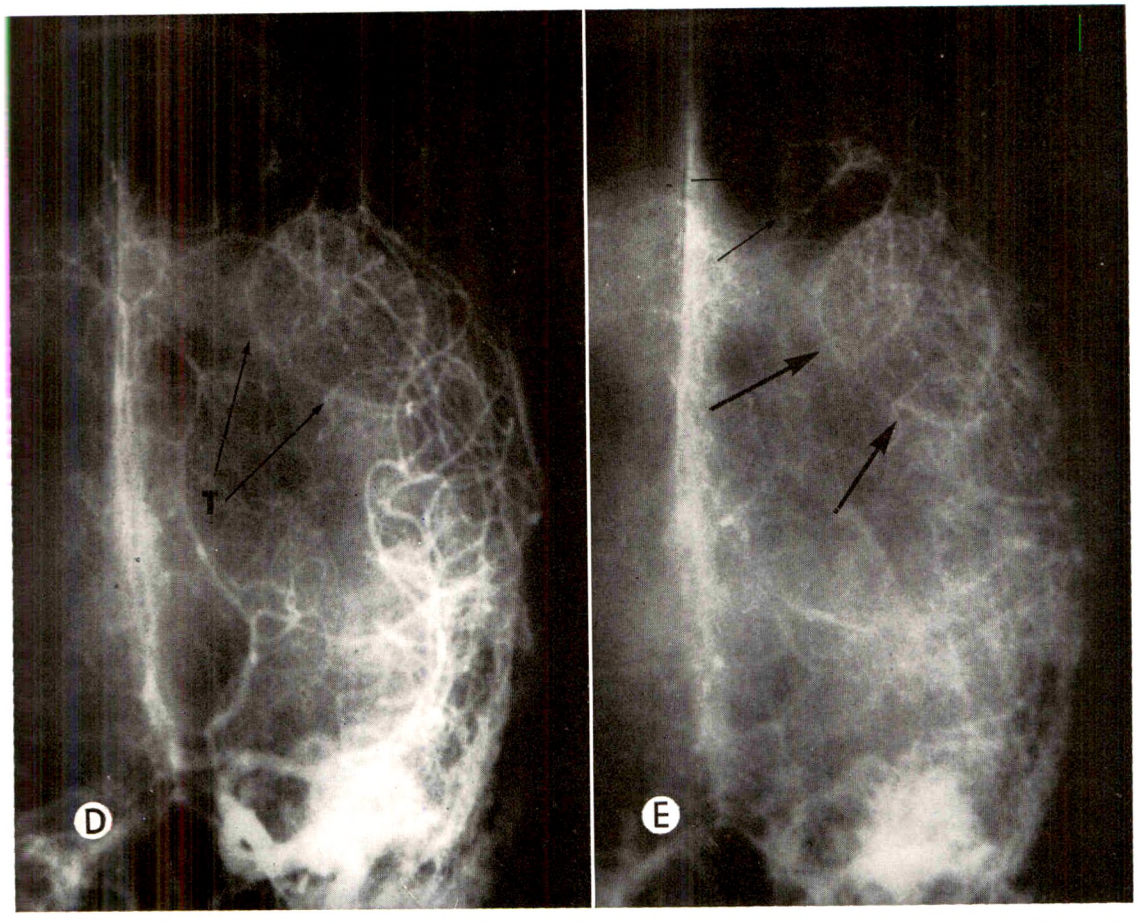



FIG. 3. Case III. (*D*) In the anteroposterior Towne projection the large tumor stain is again seen high up in the parietal region, T. Note the minimal deviation of the anterior cerebral artery due to the presence of bilateral lesions. (*E*) In the early venous phase the lesion is again demonstrated and the irregularity of its angioarchitecture is well seen (large arrows). The second smaller lesion (small arrows) is seen posteromedial to the large lesion.

sources. This case probably represents our only case of primary melanosis of the central nervous system. In our other 6 cases, in which no cutaneous lesions were found (Table IV), microscopic examination of the body for all possible sources was not carried out. Such an examination should include the rectoanal junction, the retina, the lower colon, the gallbladder, the lungs, and the adrenal medulla.[24] Pigment cells are normally present in the pia-arachnoid and have been found in 85 per cent of normal adults studied at necropsy.[17] Baader[1] found these cells most often in the sulcus between the pons and the medulla, over the cerebral peduncles and over the base of the brain. The cells have been described in a $5\frac{1}{2}$ month old fetus.[6] According to Fox *et al.*[7] melanoblasts probably arise from the neural crest although no experimental proof exists in man. When a profuse dusky melanosis of the leptomeninges is present without any focus elsewhere, the diagnosis of primary melanosis of the nervous system may be considered.[8] The published case material of primary melanosis is now extensive. Pappenheim and Bhattacharji[15] in 1962, using rigid criteria of acceptance, surveyed the literature and found 45 cases and added 1 of their own. The problem is con-

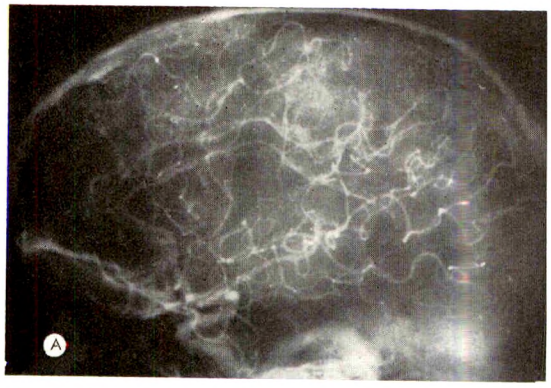



FIG. 4. Case IV. A 42 year old male, who had a nevus in his lumbar region for 10 years, developed numbness of his right hand and hypesthesia in his right upper extremity. Initial left carotid angiography was normal. He was discharged and 1 month later had a generalized seizure. Repeat studies then demonstrated a mass above the left sylvian fissure. At surgery a malignant melanoma was found in the left posterofrontal region. (*A*) Left lateral common carotid angiogram demonstrates no abnormality.

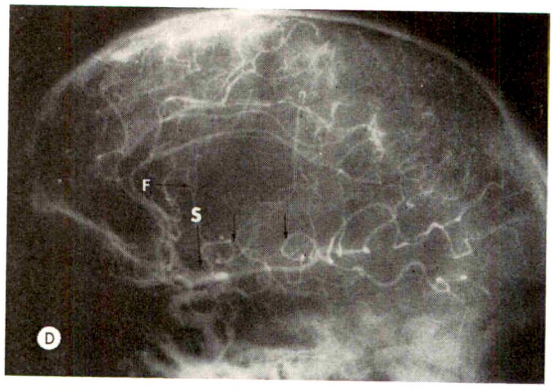



FIG. 4. Case IV. (*D*) Left lateral common carotid study 5 weeks later demonstrates compression of the supraclinoid carotid siphon, S, depression of the insular branches of the middle cerebral artery (small arrows) and stretching of the frontal arteries, F, around an avascular mass lesion above the sylvian fissure.

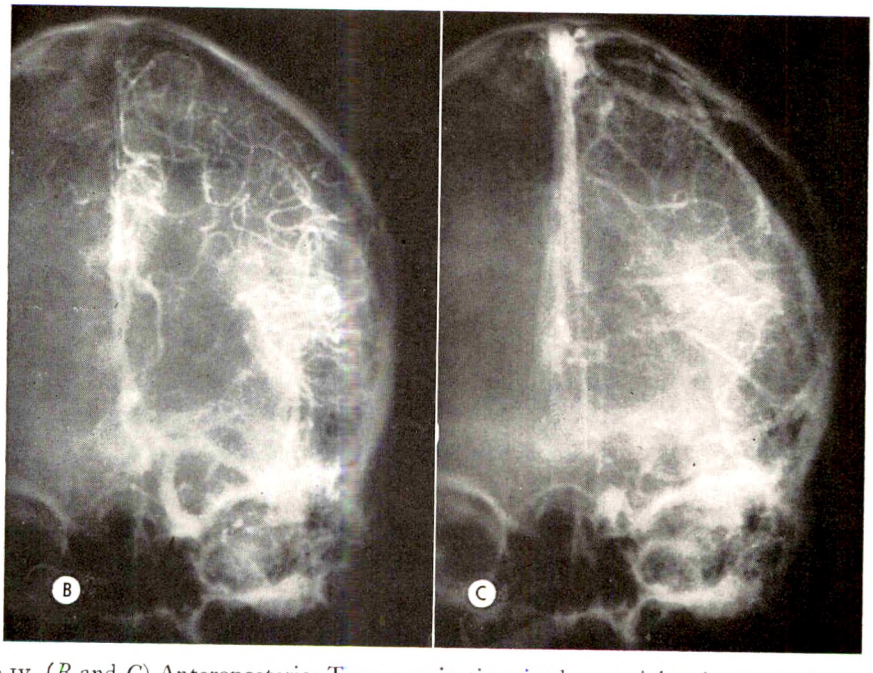



FIG. 4. Case IV. (*B* and *C*) Anteroposterior Towne projections in the arterial and venous phases also demonstrate no abnormality. Pneumoencephalography at this stage was also normal. The left carotid angiogram was repeated 5 weeks later.

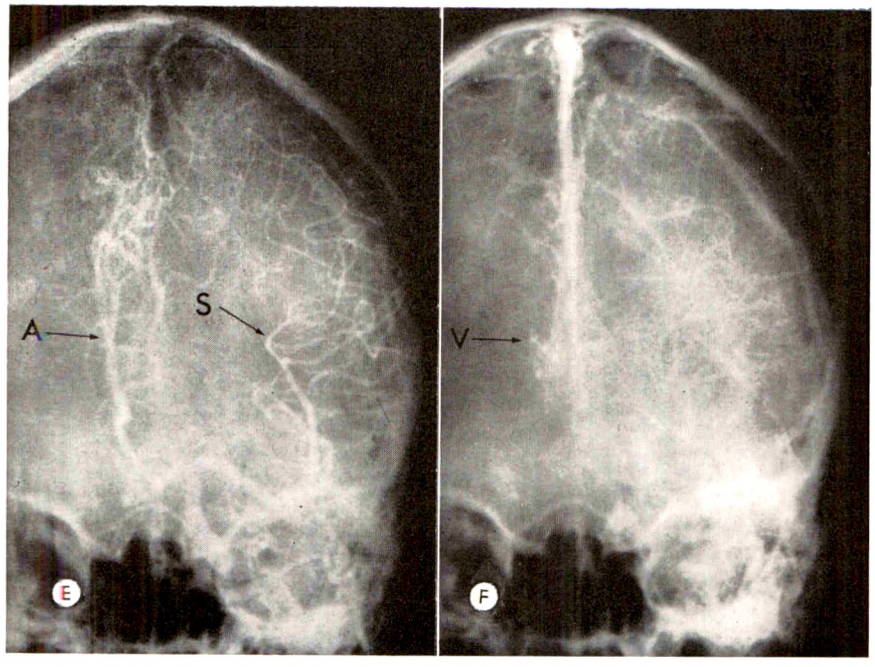



FIG. 4. Case IV. (*E* and *F*) In the anteroposterior arterial and venous phases the anterior cerebral artery, A, is seen to be displaced 13 mm. to the right, the sylvian point, S, is depressed and the internal cerebral vein, V, is displaced 10 mm. to the right.

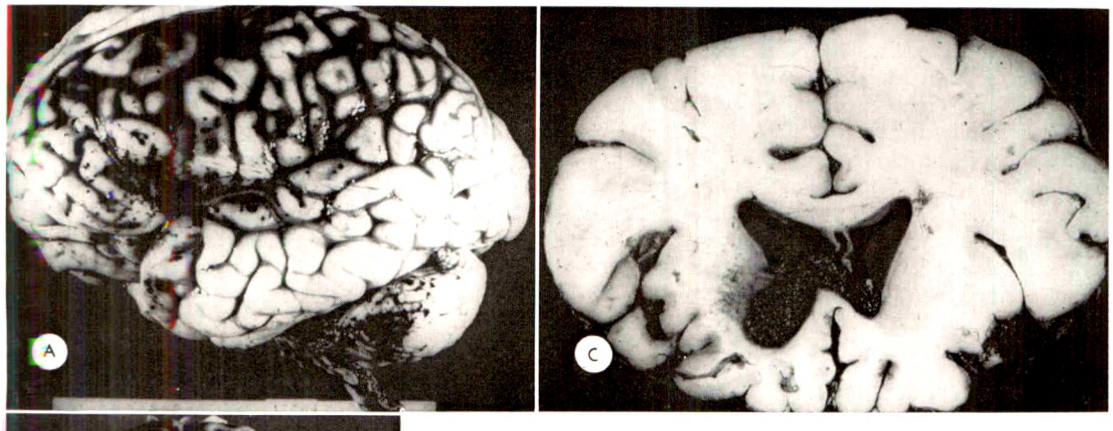






FIG. 5. Case V. A 48 year old male with a pigmented mole at the angle of his left mandible was admitted with a history of an acute onset of vomiting. A lumbar puncture revealed a subarachnoid hemorrhage. Bilateral carotid angiography disclosed large ventricles. Vertebral angiography was normal. He lapsed into a coma and died. (*A* and *B*) Lateral and basal views of the brain demonstrate diffuse melanotic involvement of meninges over cerebellum, over basal cisterns and over gyri and into sulci of the frontal and parietal lobes. (*C*) Coronal cross section demonstrates mild enlargement of ventricles and tumor mass in floor of both ventricles with extension into basal ganglia.

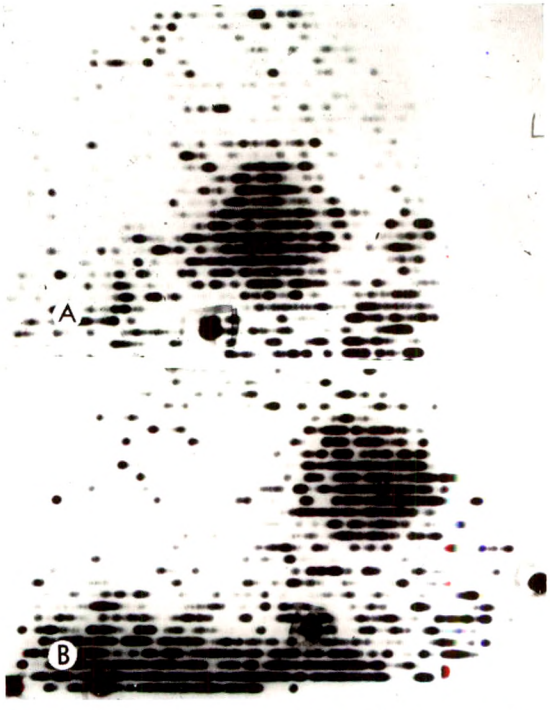



FIG. 6. Case VI. A 52 year old female with a malignant melanoma on her leg was noticed to become increasingly lethargic. On electroencephalography a left posterior temporal focus was found. Bilateral carotid angiography revealed an avascular space occupying lesion in the left posterior parietal region. A brain scanning was performed. Surgical exploration and postmortem examination were not carried out. (*A*) Posterior brain scan utilizing $Hg^{197}$ demonstrates large area of increased uptake immediately to left of midline. (*B*) Left lateral scan (occiput to the right) demonstrates the lesion in the posterior parietal region.

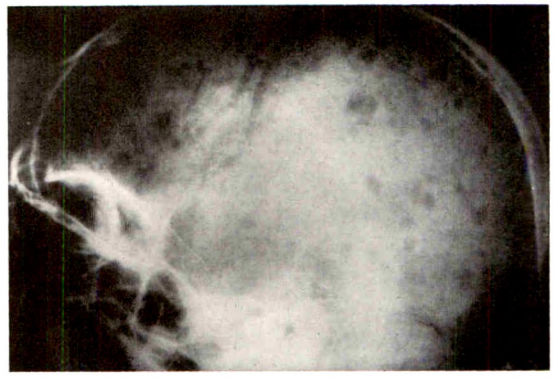


FIG. 7. Case VII. A 45 year old patient with a malignant melanoma behind his left shoulder was admitted confused and globally aphasic. A lumbar puncture revealed a subarachnoid hemorrhage. A roentgenogram of the skull was obtained. He died and postmortem examination revealed multiple metastatic malignant melanomata in his lungs, spleen, kidneys, brain, vertebrae and other viscera. Lateral skull roentgenogram demonstrates multiple small lytic areas of destruction with moderately well defined margins in the calvarium.

fused, however, by case reports of the spontaneous regression of a primary malignant melanoma with maintenance of regional metastases.[18,20]

## SUMMARY

The radiologic and clinical features of 28 cases of melanomas of the central nervous system are described.

The combination of the following features should suggest the diagnosis of an intracerebral melanoma: (1) Rapid increase in size of a known intracerebral lesion; (2) a space occupying intracerebral lesion associated with subarachnoid hemorrhage; and (3) a case with known peripheral melanoma in which a cerebral lesion is found with angioarchitecture suggestive of a meningioma.

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# ON PNEUMOENCEPHALOGRAPHIC MEASURING METHODS IN CHILDREN*

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SINCE the introduction of air encephalography the size of the lateral ventricles (and of the 3rd ventricle) has been stated on the basis of a personal estimate being described as normal, slightly, moderately, or severely dilated. In many respects this has not been satisfactory, so that in the course of time a number of different measuring methods have been recommended and used in order to eliminate partly the personal estimate which, however, is still included in the assessment. These measuring methods may be roughly divided into 3 main categories:†

1. Absolute measurements of the lateral ventricles (cella media distances and septum caudate distances) (Fig. 1).
2. Relative measurements calculating a ratio or index (Evans and Schiersmann).
3. Planimetric determinations.

The last mentioned method has been least used, as it is technically very cumbersome and not applicable in daily routine work.

The absolute measurements have come into extensive use, in particular the septum caudate distance (S-C), but all the published studies have been concerned with adult patients.[6,9,10,11]

Relative measurements, on the other hand, have been used mostly in children. There does not appear to be much difference between Evans' and Schiermann's methods.[7] For a number of years we have been using the Evans ratio which we have found reliable—and applicable at the same time as a prognostic sign—with the necessary reserve in the individual cases. Nevertheless, we occasionally come across an encephalogram in which it does not seem satisfactory to state a ratio around 0.30, as we have a feeling that in spite of this modest ratio the ventricles must be enlarged. In such cases, the S-C distance often seems more reliable. In the present study, therefore, we set out to compare the Evans ratio and the S-C distance in a series of children in order to ascertain whether either of these methods is preferable or whether they should be used to supplement each other.

## MATERIAL AND METHODS

The pneumoencephalograms (PEG) used for this purpose were derived mainly from material from the Department of Child Psychiatry in Rigshospitalet, Copenhagen.

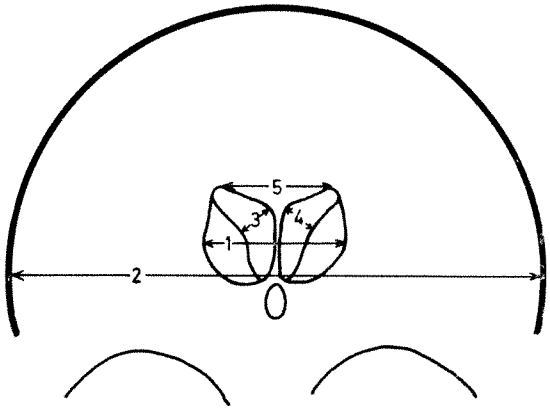



FIG. 1. Illustration of Evans' ratio, septum caudate (S-C) distance and cella media distance. 1. Greatest diameter of the anterior horns. 2. Greatest internal transverse diameter of the skull; 1:2 = Evans' ratio. 3. Septum caudate distance on the right side. 4. Septum caudate distance on the left side. 5. Cella media distance.

* From the Department of Neuroradiology (Heads: H. H. Jacobsen and J. Lester), Rigshospitalet (University Hospital), Copenhagen, Denmark.

† A fourth measuring method has been described by LÖNNUM in "The Clinical Significance of Central Cerebral Ventricular Enlargement," Oslo 1966; *i.e.*, the septum caudate distance divided by the width of the 3rd ventricle, which must not exceed 1.75. We received this valuable paper so late that we can only quote it in this way.

These pneumoencephalograms have been included in studies on the value of this examination in certain clinically abnormal psychiatric states.[1,12]

Comparative measurements were done on the PEGs of 100 children. Primarily the appearances had been considered normal in 47 and abnormal in 53 on the basis of a combination of numerical data and the subjective estimate of experienced observers (Group 1).

In age distribution, this material was dominated by the older children, so it was supplemented by the examination of 10 infants under 1 year of age (Group 2).

Lastly, we felt that it was of particular importance to include patients whose predominant abnormality was believed to affect the basal ganglia, because in such cases the ganglionic atrophy would be expected to change the shape of the lateral ventricles. The material was, therefore, further supplemented by 11 PEGs of 10 children whose clinical diagnosis was athetosis (Group 3).

Our routine method of assessing the PEGs is by determining Evans' ratio (greatest width of anterior horns in anteroposterior views/greatest internal width of the skull). The borderline between normal and abnormal is taken as 0.30. This ratio is calculated for the entire system and for each side separately. Furthermore, we measure on these PEGs the septum caudate distance on both sides. These measurements are illustrated in Figure 1.

It should be mentioned that on all PEGs we measure the width of the 3rd ventricle and estimate the configuration of the cortical air as well, but these estimates are not included in the present study.

## RESULTS

The measurements done on the PEGs from the material of the Department of Child Psychiatry (Group 1) are assessed on the basis of the total evaluation of the system as being normal or abnormal according to the named neuroradiologic criteria.

Figure 2 gives the relationship between Evans' ratio and the S-C distance in the 48 children of Groups 1 and 3 in whom the system was assessed as normal. The great majority had ratios below 0.30 and the majority had S-C distances below 12 mm.

Figure 3 sets out this relationship between the Evans ratio and the S-C distance for the 63 patients of Groups 1 and 3 in whom the system was assessed as abnormal. In these patients most ratios exceeded 0.30 and the S-C distances 12 mm.

Figure 4 presents the normal and abnormal PEGs in terms of Evans' ratio plotted against age at the time of examination. In the normal cases the ratios are, broadly speaking, distributed around a horizontal line corresponding to 0.29–0.30. Thus, Evans' ratio does not appear to increase with age. The abnormal values are on the whole above this line.

From Figure 5, showing the same features for the S-C distance, it is apparent that, unlike Evans' ratio, the values representing "normal" systems show a slight increase with increasing age.

It is hardly possible to obtain a corresponding material of "normal" PEGs from infants under 1 year of age (Group 2), as this procedure is usually not done except in the case of severe neurologic signs (cerebral palsy, oligophrenia, epilepsy, suspicion of tumor, hygroma, etc.). However, among the most recent examinations included in the present study, we have found that the ratio is as a rule very high and that where it exceeded 0.50 it was impossible to measure the S-C distance. On the whole, it must be admitted that in this age group we have not (neither from the literature nor from our own experience) been able to form an estimate of the limit between normal and abnormal ventricular size, with a reliability corresponding to that in groups of older children or adults.*

In the group diagnosed clinically as athetosis (Group 3) the results are the same as for the other abnormal PEGs, with no par-

* This is in keeping with Lönnum's paper (see Footnote on first page).

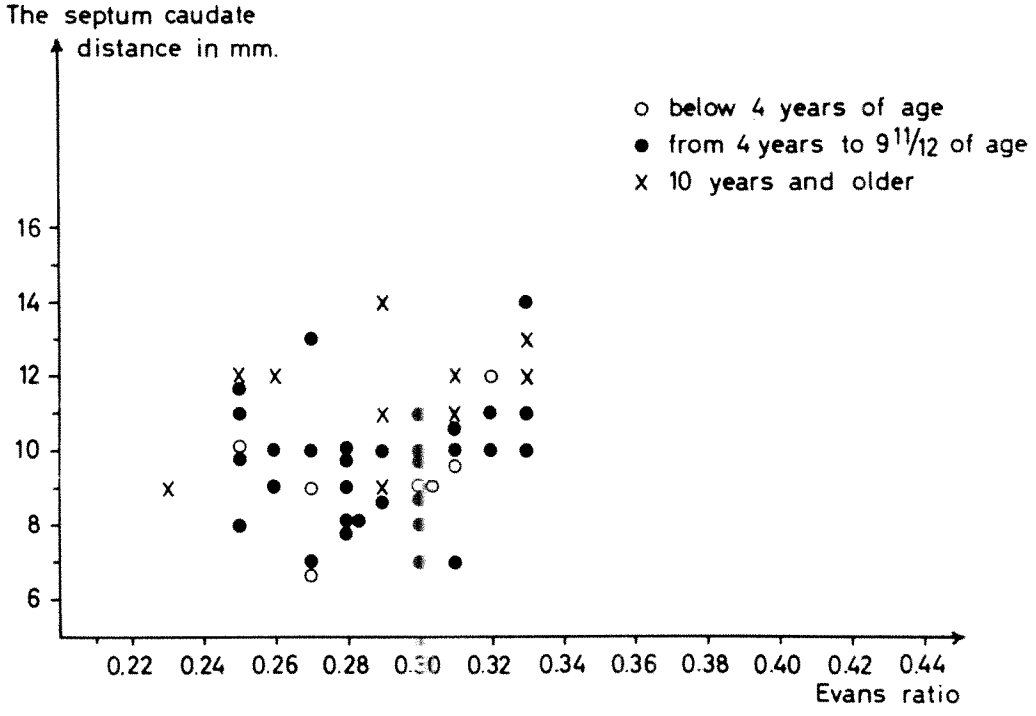



FIG. 2. Correlation between Evans' ratio and septum caudate distance in the 48 children whose pneumoencephalograms (PEG) were assessed as normal. The symbols indicate the 3 different age groups.

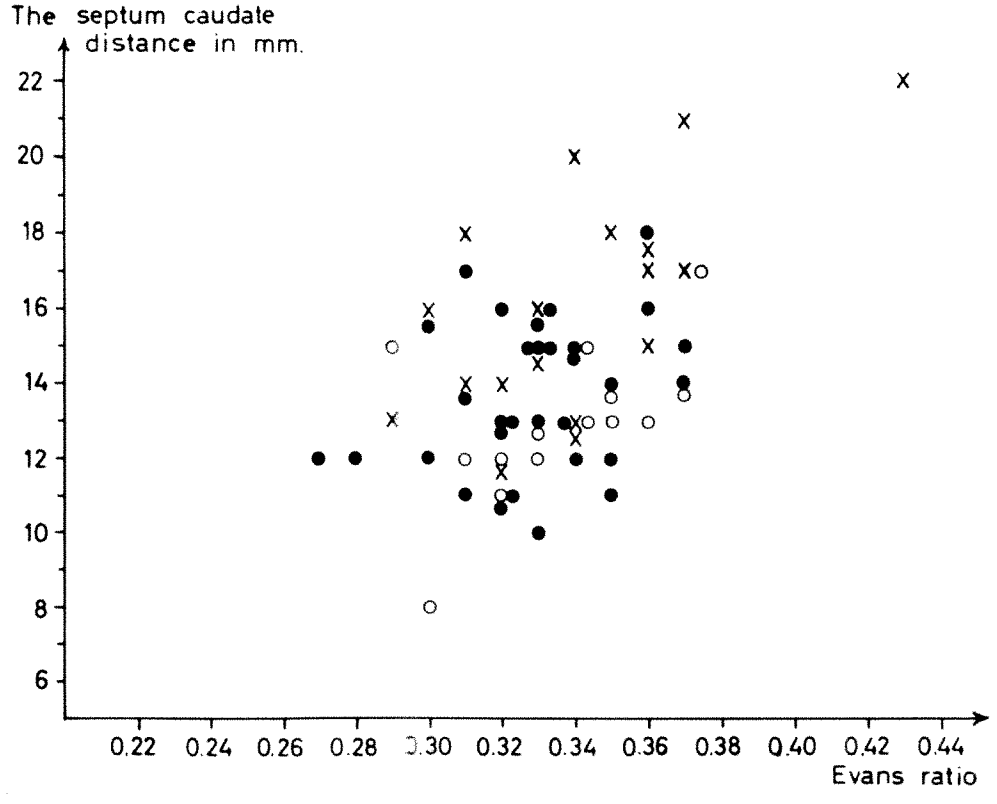



FIG. 3. Correlation between Evans' ratio and septum caudate distance in 63 children whose PEGs were assessed as abnormal. Symbols as in Figure 2.

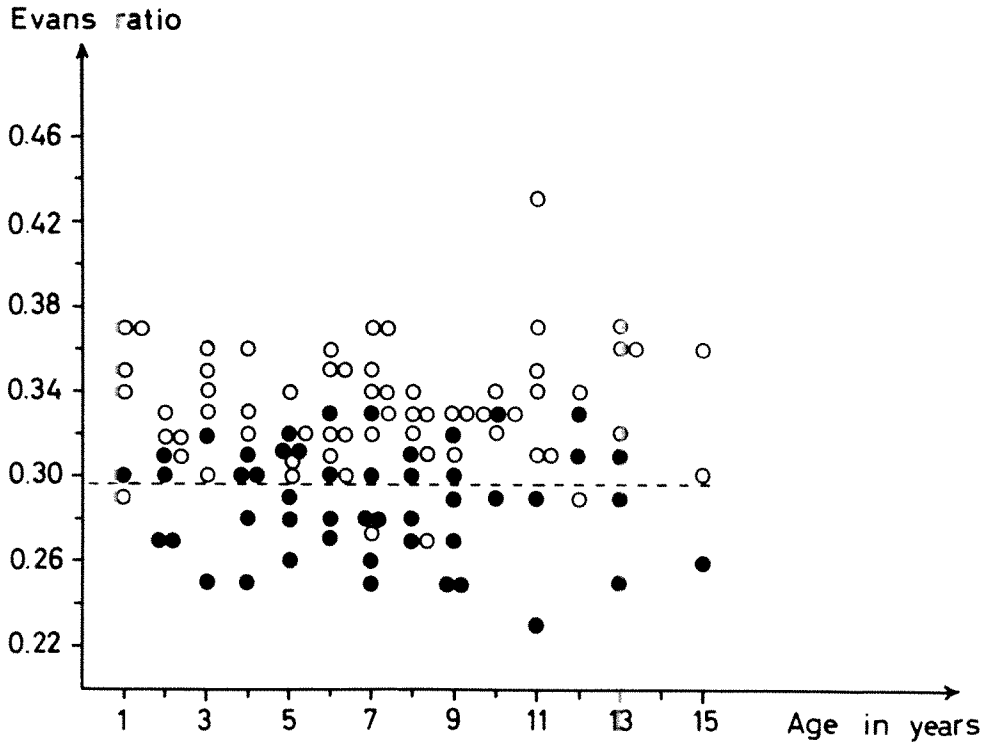



FIG. 4. Evans' ratio in relation to age. ● = PEG assessed as normal. ○ = PEG assessed as abnormal. The broken line indicates the calculated linear regression curve of the "normal" systems.

The equation for the regression curve was calculated to be as follows: $y = 0.297 + 0.0004\ (x-7)$.

The variance was found to be: $v^2 = 0.64$, which is not statistically significant with regard to the standard deviation.

The inclination of the curve was found to be 0.0004 with a standard deviation of $\pm 0.010$ (years$^{-1}$). This is not significant at the level of significance (P = 97.5%).

ticular divergence when comparing the Evans ratio and the S-C distance. Thus, this group showed no difference from Group 1.

It has previously been demonstrated that Evans' ratio shows very little dependence on the exposure technique. We have planned studying also the possible dependence of the S-C distance on the position of the head and the direction of the beam, as the reading of the roentgenograms gave the immediate impression that it might vary with variations in these factors.

## DISCUSSION

Measurements of PEGs have been done in many different ways and on many different materials. The absolute measurements, used predominantly on adult patients, have shown that the septum caudate distance, as already reported by Troland *et al.*,[11] may be of value, both in the measurement and in assessing the prognosis.[9,10] Measurement of the width of the cella media has also proved of value in adults.[4]

For many years Engeset[3] has been an ardent adherent of absolute measurement in adults, in respect to the 3rd ventricle as well as the septum caudate distance. In a study from 1964, Engeset and Skraastad[3] demonstrated that the latter is preferable and that with the Evans ratio the limit should be 0.31, while with the cella media distance it should be 41 mm.

In 44 adult males without neurologic symptoms the Evans ratio has been found to average 0.27 (minimum 0.21, maximum 0.36).[2]

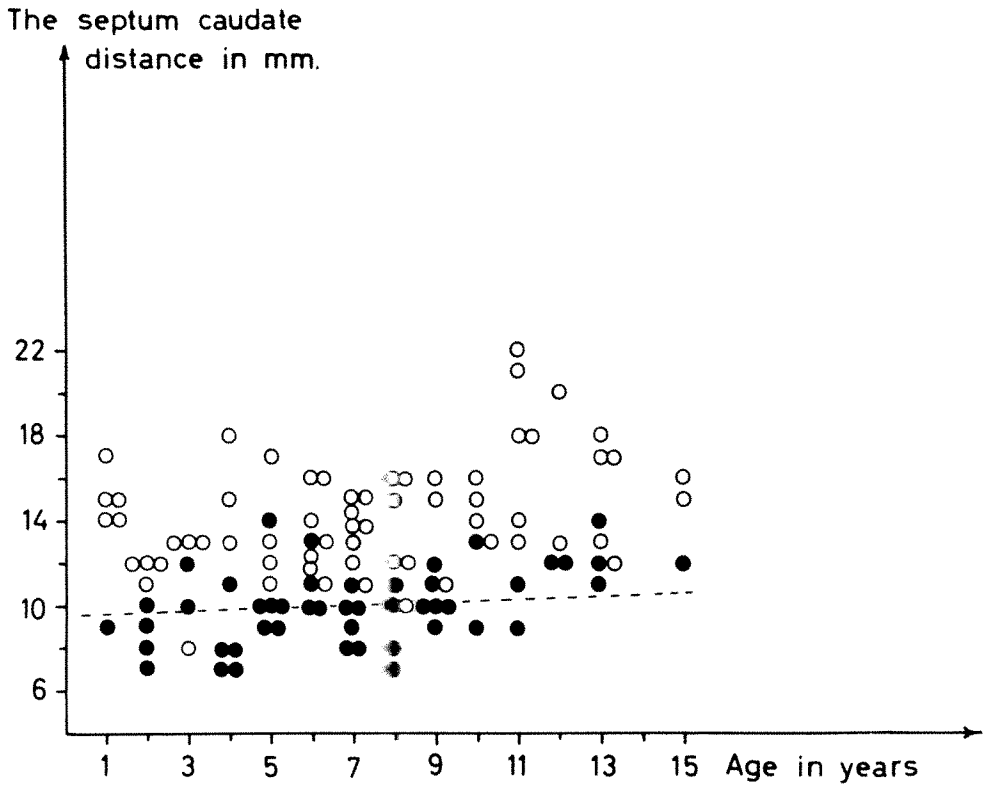



FIG. 5. Septum caudate distance in relation to age. ● = PEG assessed as normal. ○ = PEG assessed as abnormal. The broken line indicates the linear regression curve of the "normal" values.

The equation for the regression curve was calculated to be as follows: $y = 10.00 + 0.04\ (x - 7)$.

The variance was found to be: $v^2 = 5.73$, which is statistically significant with regard to the standard deviation.

The inclination of the curve was found to be 0.04 with a standard deviation of $\pm 0.18$ (mm./year). This is not significant at the level of significance (P = 97.5%).

During childhood[7] the Evans ratio has been found to be applicable and reproducible. It may also be used for prognostic guidance. Later[8] it has been demonstrated that the ratio is in conformity with the intellectual impairment in oligophrenics.

The present results show that the Evans ratio during childhood possesses the advantage of apparently being independent of age (Fig. 4). As far as the S-C distance is concerned, age plays a role, as might also be expected. Therefore, the normal age curve must be known before the findings can be evaluated (Fig. 5).

On the other hand, there is no doubt that there are PEGs in which the ratio is normal, but which give the immediate impression of being abnormal. In these cases the abnormality consists in the configuration of the lateral ventricle, rounded corners, and an abnormal outline of the caudate nucleus (Fig. 6).

The PEGs assessed as normal have a ratio below 0.35 and a S-C distance between 6 and 14 mm. But it is clearly evident that there are even transitions between "normal" and "abnormal," and that the same values in the borderline zone may be found at one time on a "normal" and at another time on an "abnormal" PEG.

Factors which should always be considered when using absolute measurement are the exposure conditions. These conditions should be strictly standardized. In other words, there must be a constant distance from the head to the focus, and regard

must be paid to the position of the head. In measuring the S-C distance we formed the definite impression of frequently varied values on exposures made in rapid sequence with little variation in head position. This has been given a preliminary test on a few patients whose heads were tilted to various degrees. It was revealed that very little variation is required to change the values. This finding is at variance with that of Troland *et al.*[11] who claim that a change of up to 10° is of no significance.

A reasonable solution is the use of both measurements, as we also measure the width of the 3rd ventricle. If this is considered unpractical, it should be pointed out that in our experience the value of the S-C distance in children is restricted to cases where the measurement of Evans' ratio does not appear in accordance with one's impression of dilatation or some other abnormality. If the S-C distance is to be of any particular use during childhood, it might be considered to introduce a kind of ratio for this value, *e.g.* by relating it to the diameter of the skull. However, it must be borne in mind that a slight uncertainty in measuring the small values which represent the numerator will mean a very great difference in the ultimate result.

The normal values of the S-C distance in relation to age we obtained, in fact, from the present study. This is in keeping with the autopsy findings of Löken[5] who reported a variation of 6–12 mm. from the age of 6 months up to 80 years.

It might be surmized that some individual clinical groups might give more reliable answers in terms of the S-C distance than of the Evans ratio. This applies primarily to diseases affecting the basal ganglia. To this end we examined children with athetosis. We found that the S-C distance was not increased relatively more than the Evans ratio in spite of lesions localized mainly to the globus pallidus.

SUMMARY

On pneumoencephalograms (PEG) of children, assessed as normal, the Evans ratio proved to be independent of age, while the septum caudate (S-C) distance showed a slight increase. In assessing the size of the lateral ventricles of children we, therefore, prefer using Evans' ratio.

Our knowledge of normal ventricular size in infants under 1 year of age has proved insufficient.

Whether the position of the head during the exposure influences the value of the septum caudate distance is discussed.



FIG. 6. Boy, aged 14 years. Evans' ratio is 0.30, but on the left the septum caudate distance is 15 mm., the upper lateral corner of the left lateral ventricle is rounded, and the left caudate nucleus is less prominent than the right. Therefore, the diagnosis was left-sided central atrophy.

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# IMPORTANCE OF THE PETRO-SAGITTAL (PETRON) ANGLES

## IN DIAGNOSTIC ROENTGENOGRAPHY*

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THE petro-sagittal (petron) angle is exceedingly important if we are to obtain comparable projections from side to side and reproducible projections on the same patient from time to time. Comparability and reproducibility are possible when the behavior of the placement of the petrous portion of the temporal bone (petron) is understood in the light of physical anthropology.

This work has been carried out during the past 4½ years.

### METHOD OF STUDY

A population was "constructed" from the card files of the Todd Museum at Western Reserve University in Cleveland. We attempted a rough approximation of the ethnic distribution of people to be found in the current spread of the population in the United States. The collection of skulls comprises some 4,500, so that it was possible to select 1,009 of them for study.

The skulls of the Todd Museum were collected between 1910 and 1940–1945. The people dying during that period were mostly immigrants, who moved into the greater Cleveland industrial complex in the last half of the nineteenth century and first quarter of the twentieth century. The ethnic groups then were easier to define than the greater admixture of today. Anthropologically this is of some predictable importance.

Conventional anthropometric measurements were made on all dried skulls in this study.[1,23] All the data and detailed results of this portion of the study are in preparation for publication in anthropologic journals (by Robert Gilbert, University of Kansas, Department of Physical Anthropology).

Immediately following the anthropometric measurements lead shots were placed upon the following points: glabella, nasion, subnasale, gnathion, gonion, mastoidale, krataphion, pterion, asterion, porion, and opisthinon.

The skulls were then roentgenographed in true basilar and true left lateral projections. All above points were observed and re-measured from the roentgenograms. In addition, the eurion-eurion and zygion-zygion measurements were obtained as were the craniocaudad angle, the petrous portion of the temporal bone (petron), made with a plane parallel with the cantho-meatal line. The petro-sagittal angles (petron angles), both anterior and posterior, were measured on each side.

A special 15 cm. ruler marked at 1 cm. intervals was placed along the left cantho-meatal line. All skulls were assigned their museum number projected onto the films.

The above voluminous data were recorded at the time of anthropometric measurement on specially designed IBM data sheets (designed for this purpose by Lockheed Space and Missile Co., of Sunnyvale, California).

Later the roentgenographic measurements were placed alongside the anthropometric measurements for exact comparison.

From all of the above data, one very clear point of interest emerged for the Radiologist and Otologist; namely, that the base of the skull and anterior petron angle measurements are of considerable

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The anthropometric portion and the statistical evaluations were supported by a grant from the Kaiser Foundation.
† Deceased.

importance. This particular study was confirmed in 100 consecutive live patients, using the Hirtz basilar projection.

## COMMENTS

Study of the many descriptions of alleged identical examinations[4–23] caused us to try to establish the basic reasons for the differences in the several texts to determine, if possible, a base line for comparison of deviations.

Basic anatomy and development,[3] and the behavior of sutures in their periods of closure, especially those closing unusually early (20 per cent), were carefully evaluated.[2]

Because of the known 20 per cent early closures usually occurring by the age of 6 years, we felt that some basic behavior patterns at the base of the skull past the age of 6 years would be useful. If no single pattern could be established then the basic deviations would be worthy of note. Examples of Hirtz's basilar views on which the mid-sagittal plane has been marked and on which lines have been drawn down the mid-portion of each petron are presented for orientation in accompanying Figure 1.

## FINDINGS

### AGE

The live patients were all in the third and fourth decades.

The age distribution of the dried skulls is shown in Figure 2 (USA, 1910–1940).

### ETHNIC GROUPINGS

The "constructed" population consisted of 18 per cent Negroid, and roughly equal parts of Southern, Northern, Eastern and Western European. A scattering of true Negro, Orientals, and American Indian was added (less than 3 per cent of total). The pertinent data are shown in Figure 3.

### ROENTGENOGRAPHIC STUDY

The lateral roentgenogram of the skull added nothing new; therefore, the findings of the base of the skull only are reported. A standard Hirtz projection was the most productive from a roentgenographic standpoint in the live patient.

### MEASUREMENT OF PETRON ANGLES (PETRO-SAGITTAL ANGLES)

The sagittal leg of the angle is established by drawing a line between the central incisors (if present) posteriorly through the dense raphe separating the right and left nasal cavities to the crista between the left and right posterior cerebellar fossa. The occipital spine should not be used as it frequently deviates perceptibly to one side. One should not expect this line to bisect the foramen magnum; usually it will not (Fig. 3). See examples of Figure 1. The lateral legs of the angles are established by drawing a line through the mid-portion of each petron (petrous portion of each temporal bone). There is a dense line seen on nearly all petrons which represents the superior linear thickening of the tegmen. This excellent roentgenographic landmark is formed partly by the depression for the semilunar ganglion anteroposteriorly and an excavation for the lesser petrosal sinus inferosuperiorly. It matters little whether one uses this mid-petron landmark, or the anterior margin of the petron, or the posterior margin of the petron, as long as one is consistent from side to side and throughout the departments. With only a little directed attention the technologists will become surprisingly adept at this measurement.

These three lines will form two anterior petron (petro-sagittal) and two posterior petron (petro-sagittal) angles, and a right and left pair.

Most of the procedure we are interested in is studying the approach to the petron (petrous portion of the temporal bone) either axially (along the long axis of the petron) or perpendicularly to it. Our findings are valid for all such projections.

The reference point for angle measurement herein presented is anterior (Fig. 1). We find in a large number of skulls that the *posterior petron angles* will be acute and the *anterior petron angles* will be obtuse. We

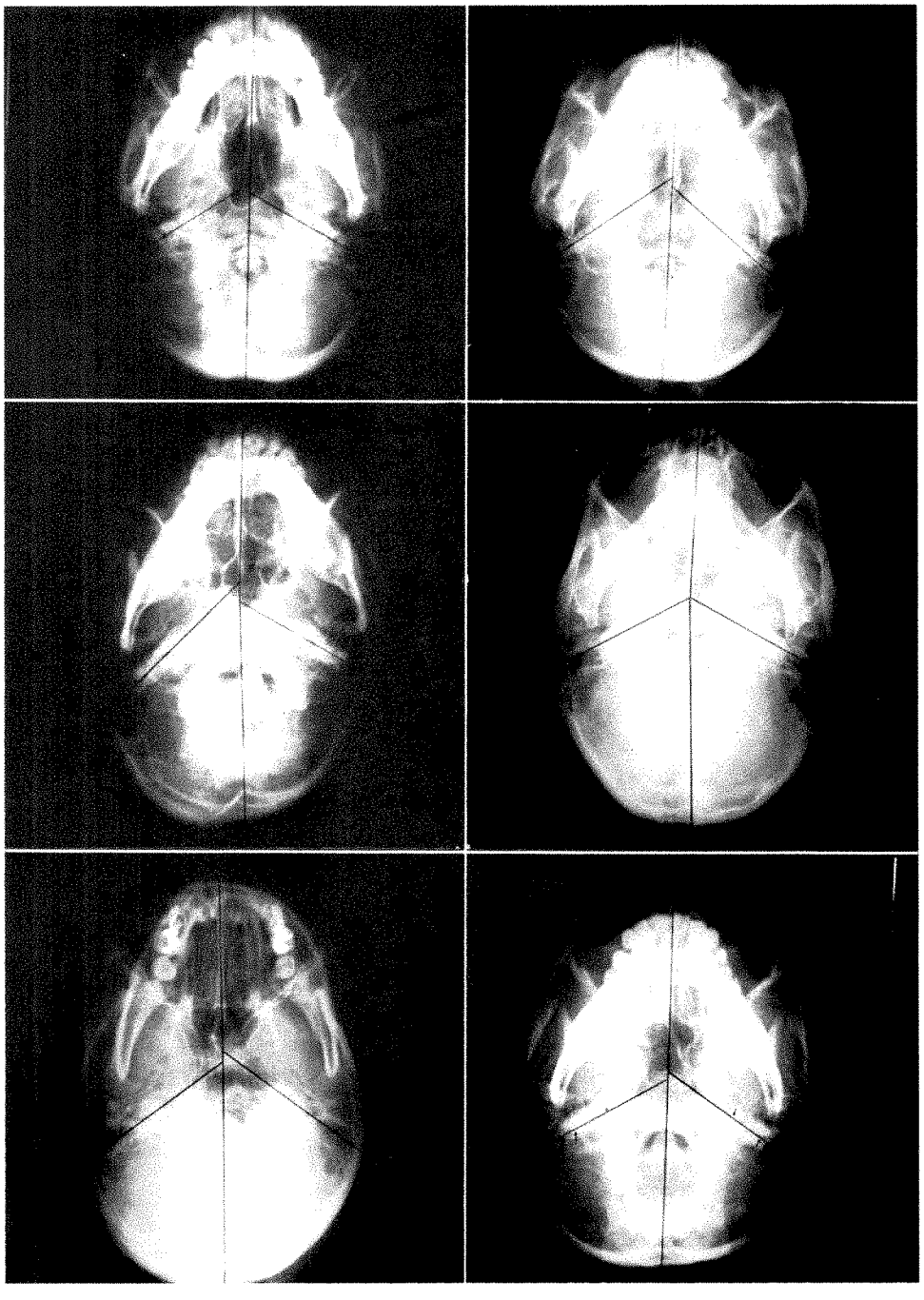


FIG. 1. Examples of basilar views, their variations and some of the asymmetry of moderate degree found in the live patients.

prefer to refer *only* to the anterior petron angles (anterior petro-sagittal angles) for technique measurements.

Most techniques approaching the petrons axially or perpendicularly must approach them from the side. Ninety degrees must be subtracted from the measured angle (see Fig. 1). Example: 135° less 90° equals 45°. This will be the angle as seen from the side.

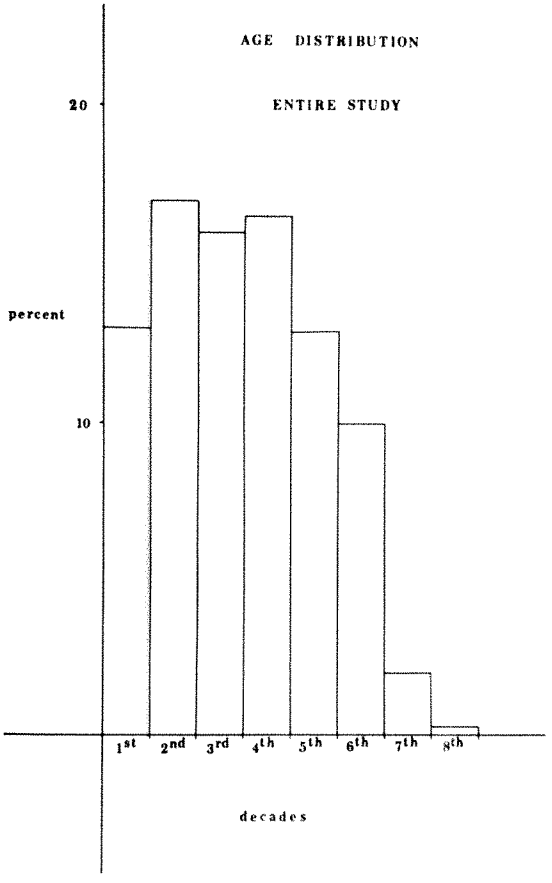



FIG. 2. Histogram of age distribution.

It is easier to work with a positive rather than with a negative angle--our reason for using the anterior petron angle.

An enormous spread of the anterior petron angles was found. The smallest anterior angle was 72°, the largest 145°—a spread of 73° (Fig. 3).

In a single skull the anterior petron angles varied as much as 48°. The posterior petron angles in another skull varied as much as 33°.

Two-thirds of all skulls studied showed deviations from one side to the other plus or minus two degrees. Only 5 of 1,009 were equal either anteriorly or posteriorly. Not a single skull was symmetric enough to give equal anterior and posterior angles.

Study of the whole skull, presented as ethnic distribution by angles and by percentage of each angle grouping of 3° compartments gives a most interesting picture.

The ethnic distribution per each 3° compartment of this group is shown in Figure 4. Note that the mean is 122.8°. The mean has little significance for study of any particular patient. Unfortunately, it has little correlation even from side to side. Careful study of this complicated chart reveals that the Northern, Western and Southern Europeans *tend* to have a lesser petron (anterior petro-sagittal) angle and a lesser total spread, and the Negroid skulls show the anterior petron angle *tending* to be wider, and the spread greater. Over-all, the variations of the Negroid skull are very great (Fig. 3)—the widest distribution of any ethnic group.

The mean of both sides throughout the study was 121.6° (Fig. 5).

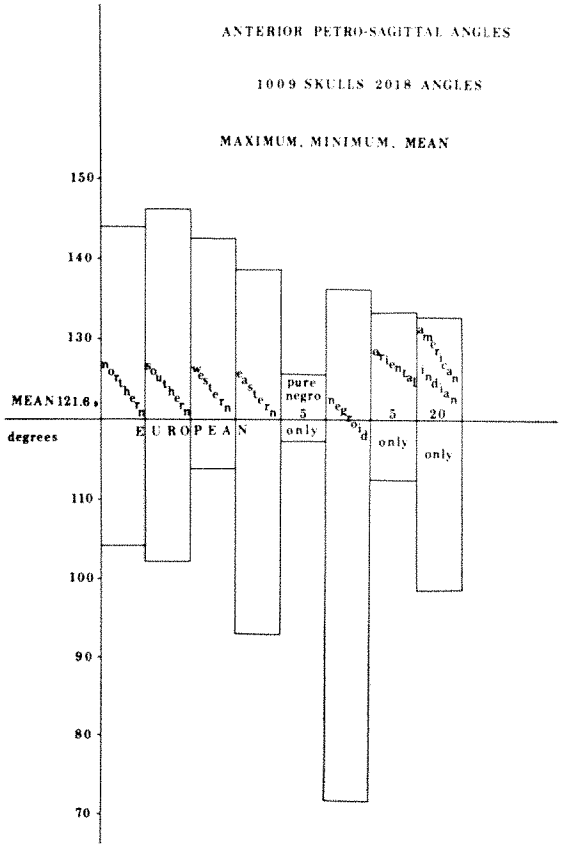



FIG. 3. Histogram of anterior petron angle spread in each ethnic group.

Left to right: Northern, Southern, Western and Eastern European; pure Negro, Negroid, Oriental and American Indian.

## DISCUSSION

Unfortunately, the deviation within any specific ethnic group is so variable that dependence upon this modality is highly unsatisfactory in predetermining technical exactness.

We found that variations within the same skull from right side to left side were sufficient to warrant prior survey of the base of the skull before making any approach to detailed study of the temporal bones.

Our anthropometric studies, even those applicable to the live patient, can predict with a very high degree of accuracy (99.5 correlation on $T^2$ and $R^2$ factors) the angles the petrons make with the mid-sagittal plane. The use of a Western Reserve head spanner and calipers about the eyes and ears becomes a technical matter; it is likewise time consuming. We, in the field of Radiology, and those in Otology and Otolaryngology as well as Medicine in general, are accustomed to convenient roentgenographic techniques. Measurements of the petron angles from a basilar projection of Hirtz are exact and easy to do and consume little time.

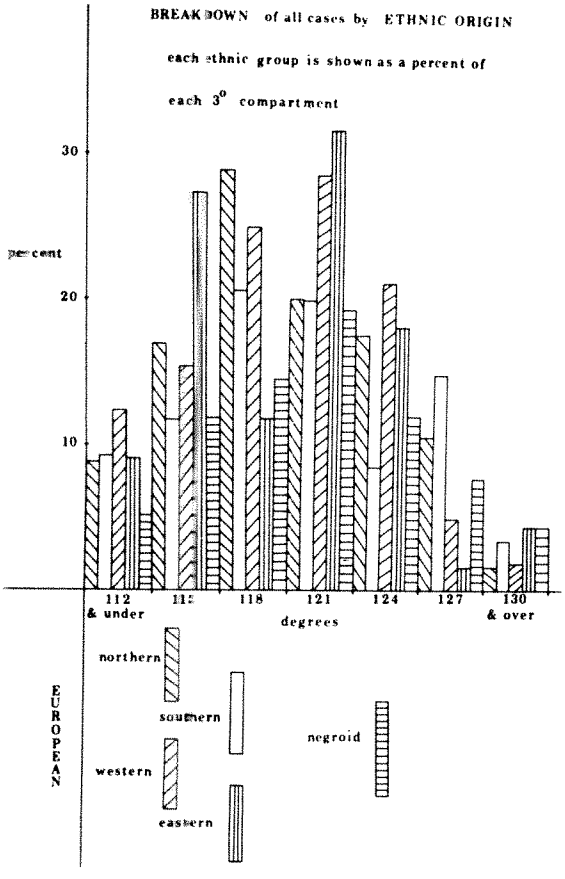



FIG. 4. Histogram showing percentage of each ethnic group per 3° compartments within the designated angle category. Values are anterior petron angles. Read from left to right in each compartment.

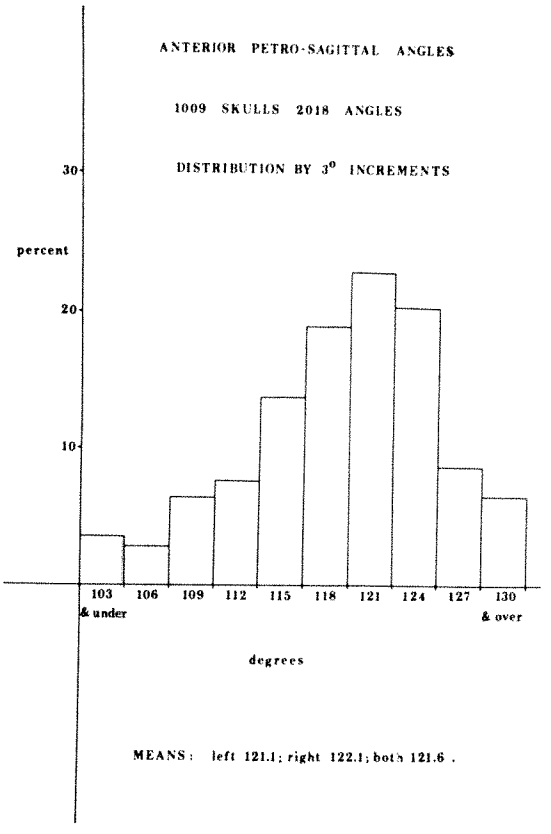



FIG. 5. Histogram showing statistical distribution of all ethnic groups by 3° increments of anterior petro-sagittal angles. Represented as a percentage of the whole group (1,009 skulls).

## CONSIDERATION OF TECHNIQUES

*Techniques using more than one angle.* We refer here to all projections approaching the petron (petrous portion of the temporal bone) either perpendicularly or axially. Examples: Owen, Mayer, Stenvers, and Low-Beer techniques.

*Mayer technique.* Usual description: tube

angle 45° caudad, mid-sagittal plane angle 45°.

*Owen technique.* Usual description: tube angle 30° caudad, mid-sagittal plane angle 30°.

*Stenvers technique.* Usual description: tube angle 12° cephalad, mid-sagittal plane angle 45°.

*Low-Beer technique.* Usual description: tube angle 10° cephalad, mid-sagittal plane angle 33°.

The tube angle should not be changed from that described; this is the primary constant angle. The mid-sagittal plane angle (facial angle) should be adjusted to the degree the *face* needs to be turned toward or away from the film, for each side of each patient. This should be considered the secondary or variable angle. Only a rare patient has exactly the same angle on each side, 1 in 200 or more in this series.

*Reference points on the head.* We have found that marking a point on the skull for specific entrance or exit of the roentgen ray beam varies from technologist to technologist; however, skillful adaptation may be achieved in a surprisingly short time.

## ALL HUMAN HEADS ARE REMARKABLY DISSIMILAR

With conventional left (only) anthropometric and bilateral anterior and posterior petron angles as baselines we were unable to find any 2 skulls alike. If we add an additional 30 measurements on the right side as well, we still feel that there are no 2 human skulls alike—30×30×2×2 or *3,600 possible combinations*. Furthermore, we suspect that there never have been nor will there ever be any 2 skulls exactly alike.

## SUMMARY

Human skulls differ so widely that it is reasonable to assume that no 2 are exactly alike.

The variations of the anterior petron angles (petro-sagittal) varied from 72° to 145°—a 73° spread in this series. In a single skull the anterior petron angles varied as much as 48° apart. In another skull the posterior petron angles varied as much as 33°. In not 1 case of 1,009 did we find the anterior angles equal at the same time the posterior angles were equal. In only 5 of 1,009 did we find *either* the anterior or the posterior petron angles equal.

With such a wide spread possible, and with such a known and expected asymmetry in nearly every skull, a simple approach to the solution is to find *where* the petrons (petrous portions of the temporal bones) will be in the patient under study. Direct measurement off a Hirtz basilar skull projection is recommended. Adjusting of the facial angle prior to roentgen study is necessary.

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The guidance of Dr. Bass and the work of Robert Gilbert of the Department of Physical Anthropology, University of Kansas, were invaluable throughout this study as were the services of James Pinkham, R.T., of our Santa Clara office.

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# ARTERIAL CALCIFICATION IN ADULT CRETINS*

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CRETINISM is a condition of marked hypothyroidism which usually develops before birth.[6] The finding of extensive arterial calcification in a 41 year old cretin led to the present study.

## MATERIAL

Only cretins who have survived for at least 18 years have been included in the case material. The clinical records and roentgenograms of 14 such patients were available for review. Table 1 summarizes the age and sex of these patients. The age at the time of initial treatment for hypothyroidism and the presence or absence of arterial calcification are also shown. The search for arterial calcification was limited to the aorta and iliac arteries.

TABLE I

| Patient | Age | Sex | Age When First Treated | Arterial Calcification |
|---|---|---|---|---|
| T.S. | 18 | M | 4 years | Absent |
| N.H. | 19 | F | 3 months | Absent |
| P.P. | 20 | F | 3 years | Absent |
| S.W. | 21 | F | 9 years | Absent |
| R.E. | 22 | M | 4 months | Absent |
| E.T. | 23 | F | 4 months | Absent |
| Y.W. | 27 | F | 4 years | Absent |
| J.P. | 29 | F | 28 years | Absent |
| L.E. | 33 | F | 24 years | Absent |
| C.W. | 38 | F | 38 years* | Absent |
| L.S. | 43 | M | 9 years | Absent |
| A.N. | 45 | M | 41 years | Very Marked |
| F.G. | 50 | F | 23 years | Minimal |
| H.F. | 57 | F | 45 years | Very Marked |

* Received thyroid medication for 2 years as a child. Treatment not resumed until the age of 38 years.

All patients were mentally deficient. With the exception of those patients who had the benefit of thyroid replacement therapy at an early age, there were bone changes from cretinism.

## DISCUSSION

Untreated adult cretins are now rare, as most cretins are treated for at least a short interval at such an early age that their characteristic features are lost.[2] Adults with cretinism or myxedema commonly have pronounced arteriosclerosis.[3,4,6] Calcification is often a conspicuous part of atherosclerosis and senile arteriosclerosis.[1]

After an extensive review of the medical literature, we have found only 2 authors who commented on the presence of marked arterial calcification demonstrated on roentgenograms of 2 adult cretins. The patient described by Jackson[4] was 51 years old and

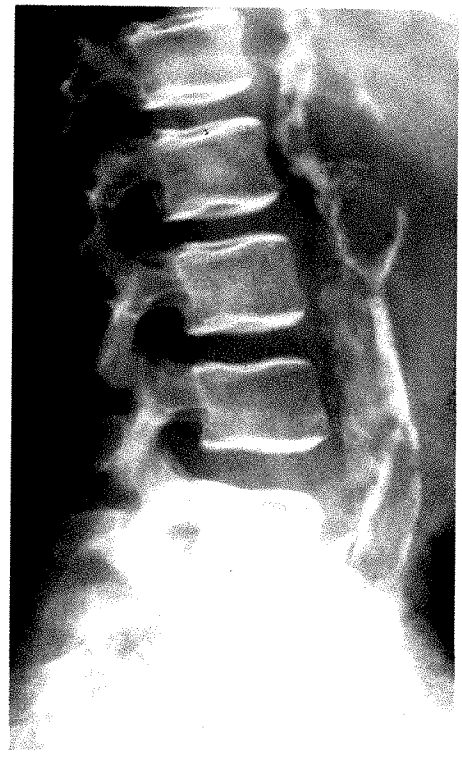


FIG. 1. Lateral roentgenogram of the lumbar spine of a 51 year old cretin shows extensive calcification in the aorta and iliac arteries. There are changes consistent with cretinism elsewhere in the skeleton.

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had received "grossly inadequate" treatment. The second one is illustrated in an article by Middlemass[5] and is patient A.N. in our series. This cretin was first treated with thyroid replacement therapy when he was 41 years old. Figure 1 is a lateral roentgenogram of the lumbar spine of our only other cretin (H.F.) with marked arterial calcification. This patient was not treated for hypothyroidism before the age of 45 years. All of the remaining patients in our current study received treatment prior to the age of 24 years. The minimal aortic calcification in patient F.G. is not considered unusual and cannot be definitely related to cretinism.

The present study and review of the literature strongly suggest a causal relationship between untreated cretinism in adults and extensive arterial calcification on the basis of arteriosclerosis. It seems probable that the extent of arteriosclerosis in cretinism may be limited by treatment for hypothyroidism even after osseous and mental changes have become irreversible.

SUMMARY

A roentgenographic search for arterial calcification was carried out on 14 adult cretins. There is strong suggestion of a causal relationship between untreated cretinism in adults and extensive arterial calcification on the basis of arteriosclerosis.

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# CONGENITAL SPINAL EXTRADURAL CYST IN TWO SIBLINGS*

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CONGENITAL spinal extradural cyst is a rare clinicoroentgenologic entity. Among 250 spinal tumors, Elsberg, Dyke and Brewer[12] found only 4 such cases. Taveras and Wood[45] stated that not a single case had been seen at the Neurological Institute in the last 10 years.

The author had the rare opportunity to examine a sister and a brother with this disease, in an interval of about 5 years. The myelographic findings were unusual and both patients were proven to have extradural cysts.

The purpose of the author is to first, report the 2 sibling cases, as congenital extradural cyst in the same family has hitherto not been recorded in the literature; and second, to discuss the specific myelographic feature, which is, in his opinion, pathognomonic for the lesion.

## REPORT OF CASES

CASE 1. The patient, a $10\frac{1}{2}$ year Negro girl, was admitted to The New York Hospital-Cornell Medical Center with the chief complaint of staggering and falling down frequently in the past 5 months. Her first hospitalization, about 11 months earlier, was for evaluation of "abdominal pain." She was sent home after 2 days with no diagnosis. The mild, vague "abdominal pain" continued, occurring only early in the morning. Two months before admission she complained of "not knowing where my legs are." One month later it was noticed that she staggered when her eyes were closed or covered, as when slipping off a dress. This progressed until the time of admission, when she staggered with eyes open. Two weeks prior to admission she began to complain of numbness and tingling of the lower extremities.

Physical examination showed an incomplete pin and touch level at the upper thoracic region, more on the right side than the left. Vibration and position sense were diminished below this level. The abdominal reflexes were absent bilaterally. There was bilateral hyperreflexia in knees and ankles and bilateral extensor plantar responses.

*Roentgenography.* Roentgenographic examination of the thoracic spine (Fig. 1*A*) showed flattening of the vertebral pedicles from T5 to T10. The atrophic pedicles were reduced to an almost shell-like appearance at T7, 8, 9 levels. On plotting the interpedicular distances (IPD) of the thoracic vertebrae on the normal graph for the age of $10\frac{1}{2}$[19] (Fig. 1*C*) it became obvious that the patient's curve did not run parallel to the normal average curve or the normal tolerance range. As noted on the graph, the IPDs from T11 toward the upper thoracic region normally decline. In this patient, there is, instead, a gradual increase in the IPDs from T11 toward the middle thoracic region and the IPDs in the thoracic region are maintained at a curve higher than in the lumbar region. Although the IPDs of this patient still fall within the upper limit of the normal range, the abnormally high curve in the thoracic region in comparison with the lumbar curve indicates an expanding process in the thoracic region. This is further evidence that the general practice of measuring IPD alone against normal upper limits is inadequate in detecting pedicle abnormalities. One should not overlook the general tendency of the IPD curve when compared with the normal. *Any significant deviation of the curve direction should indicate an abnormal finding, even though the IPD measurement may still be within normal limits.*

*Myelography.* The myelographic findings (Fig. 2, *A* and *B;* 3, *A* and *B;* and 4, *A* and *B*) are most interesting and are, therefore, detailed below. The manometric opening pressure was 150 mm. $H_2O$. There was a very slow rise in manometric pressure with jugular compression, and a very slow fall, especially with head extended. The protein content was 53 mg. per cent. Owing to the partial manometric block,

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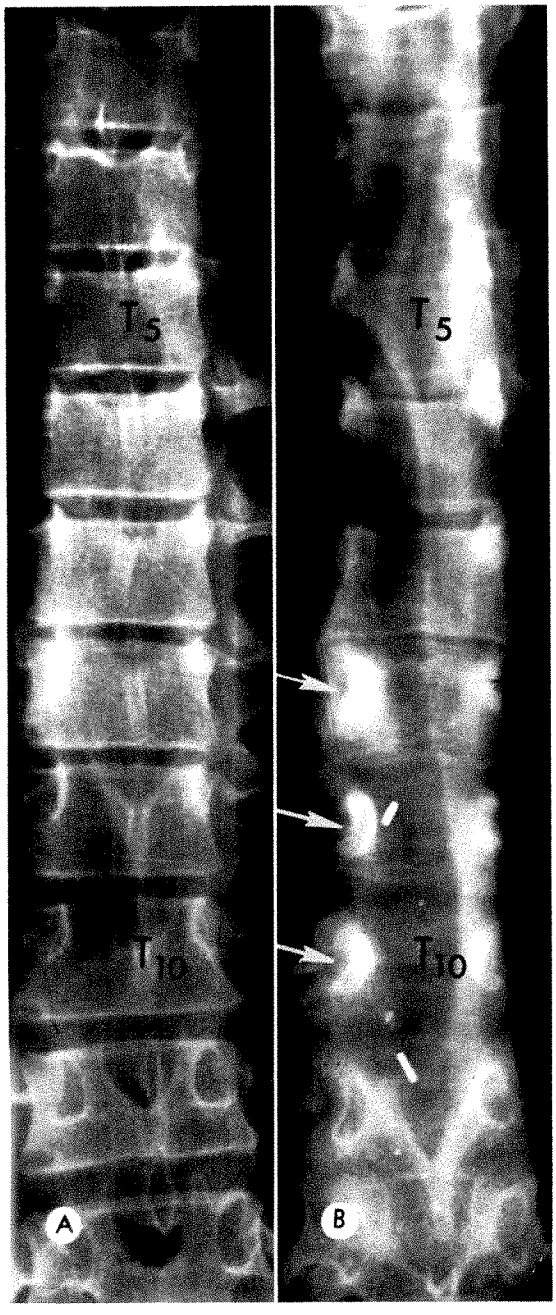



Fig. 1. Case 1. (*A*) Roentgenogram of the dorsal spine showing flattening and atrophy of the pedicles and an increase in the interpedicular distances (IPD) from T5 to T10. (*B*) Roentgenogram of the dorsal spine 1 year after removal of cyst. Normal pedicles have reappeared and the IPDs have returned to normal. Compare especially the pedicles of T8, T9 and T10 (arrows) with *A*.

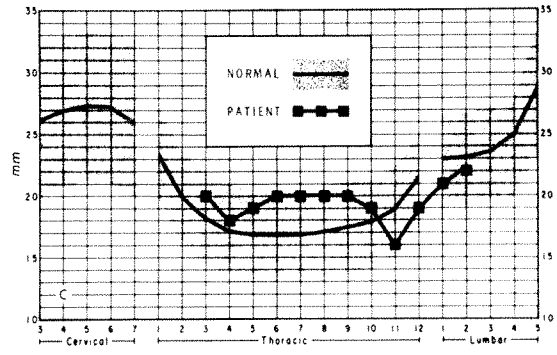



Fig. 1. (*C*) Graph showing patient's interpedicular distances (■–■–■) in *A* as compared with the normal curve (————) by Hinck *et al.*[19]

only 2 cc. of pantopaque was introduced into the lumbar subarachnoid space. The myelographic findings are best described in 3 phases, wherein 3 different levels of block to the contrast medium flow were encountered. The sketches accompanying the spot roentgenograms aid in the explanation of the formation of the 3 blocks.

(1) When the patient, in prone position, was tilted head downward, a block to the contrast medium flow was encountered at T9 (Fig. 2, *A* and *B*). The myelographic appearance of the block is that of an extradural lesion, since the contrast medium in the subarachnoid space below the block is gradually thinned out. Although the thinning of the pedicles extends down to the T10 level, the spot roentgenograms in the prone position showed that the contrast medium passed the T10 level and was blocked only when it reached T9. As shown on the sketch, this first block occurred because the contrast medium, still in the subarachnoid space, was compressed by the extradural cyst.

(2) The spinal needle was then removed and with the patient in the supine position, (Fig. 3, *A* and *B*) the table was tilted again head downward. The contrast medium then flowed past the T9 level and continued craniad to the T4–5 level, where a complete block was encountered with a cul-de-sac appearance at the head of the contrast column. The contrast medium clearly demonstrates the upper end of the extradural cyst. The sketch (Fig. 3*B*) shows the opening of the extradural

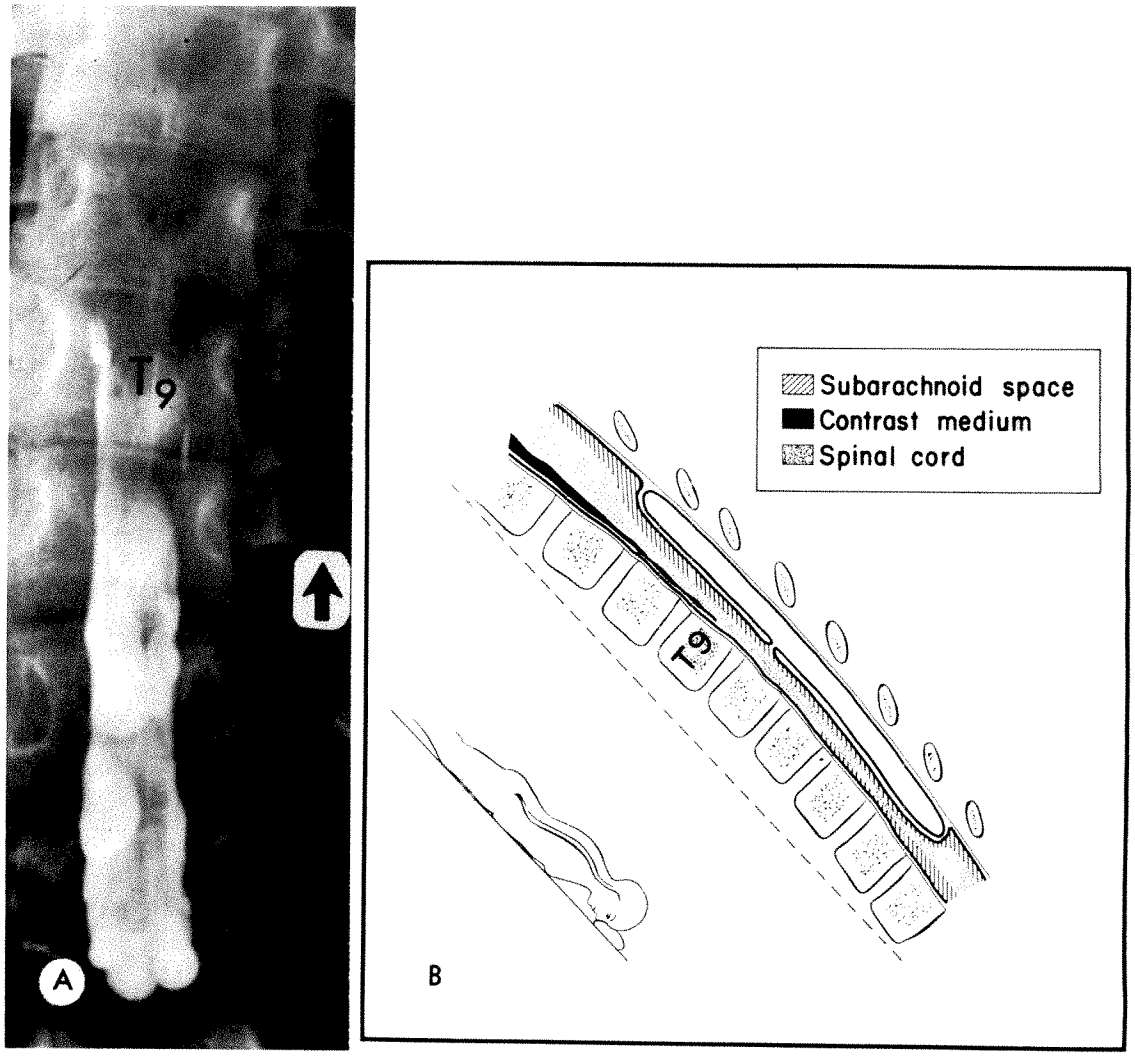



Fig. 2. Case 1. Prone position. (*A*) Ascending myelography showing block at T9. (*B*) Sketch showing contrast medium in the subarachnoid space, compressed by the cyst.

cyst to be on the dorsal aspect of the spinal canal; thus contrast medium flows easily into the cyst when the patient is placed in the supine position. The contrast medium which demonstrated the second block at the T4–5 level has entered the cyst.

(3) With the patient remaining in the supine position, (Fig. 4, *A* and *B*) the table was tilted feet down and the contrast medium flowed caudad. This time, no block was noticed at the T9 level, as was the case in the prone position, because the contrast medium is within the cyst. Instead, another complete block is encountered at the T11 level, which represents the lower end of the cyst.

In summary, 3 levels of block were encountered. The first block occurred in the prone position at T9. The contrast medium was then still outside the extradural cyst in the subarachnoid space which was compressed by the cyst. The second and the third blocks, both identified in supine position, occurred when the contrast medium entered the cyst through a dorsal opening and represented the upper (T4–5) and lower (T11) ends of the cyst.

*Operation.* At operation, the myelographic impression of a long extradural cyst was confirmed. A fluctuant cyst with a bluish-gray translucency, measuring about 10×2½ cm. across, underlay almost the entire length of the laminectomy from T3 to T11. This cyst was dissected away from the underlying intact dura

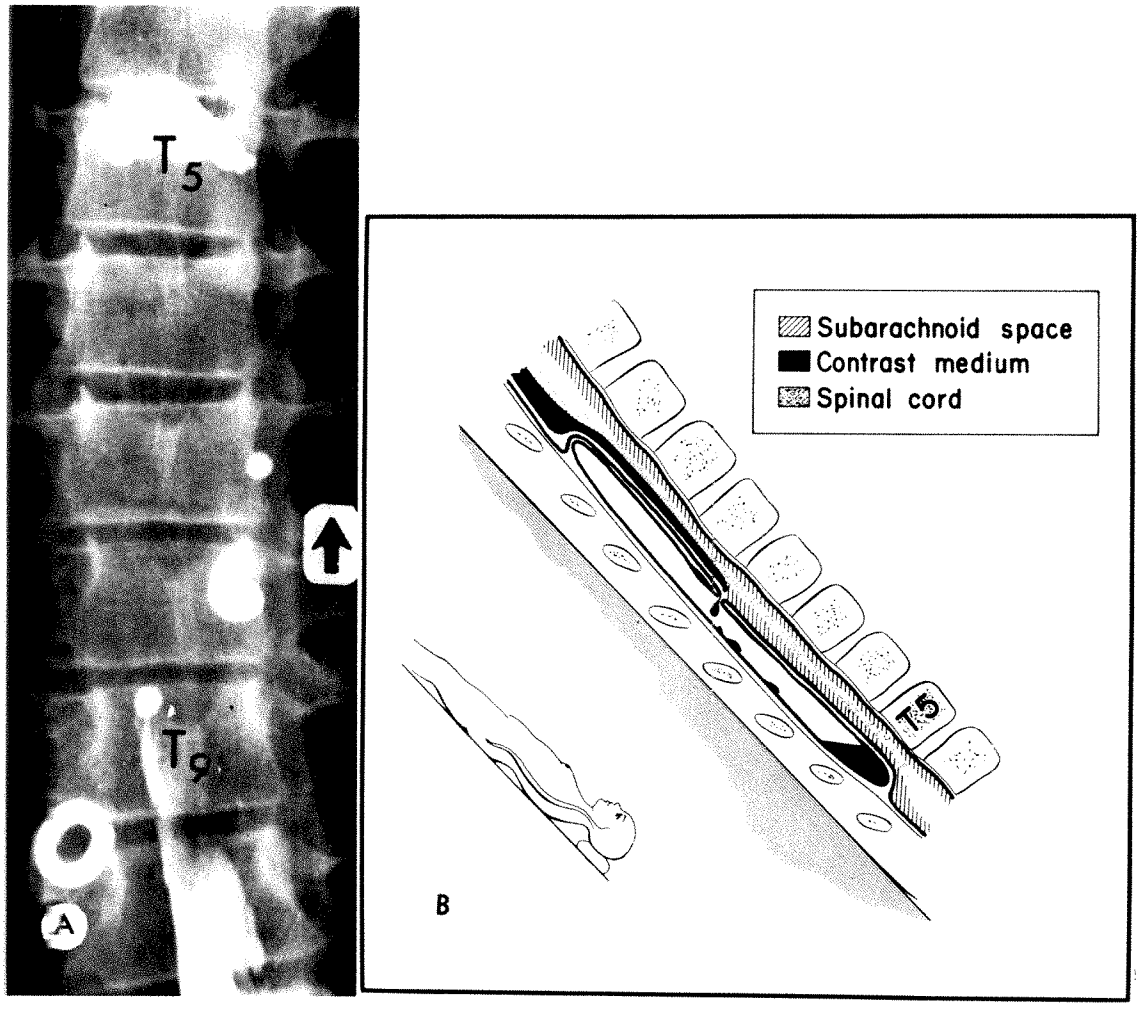



FIG. 3. Case 1. Supine position. (*A*) Ascending myelography. Contrast medium entering the cyst and collecting at its upper end at T5. (*B*) Sketch showing flow of the contrast medium from the subarachnoid space into the dorsally situated extradural cyst.

in a grossly enlarged spinal canal. A communication, about 1 cm in length, existed in the dorsum of the dura and slightly to the right at about the T8 level.

*Pathology.* Microscopically, the wall of the cyst was a membranous portion of dense, fibrous tissue resembling dura. The membranes were not infiltrated by inflammatory cells nor did these tissues contain hemosiderin.

CASE II. About 4 years and 10 months later, the 12 year old brother of Case 1 was admitted to The New York Hospital-Cornell Medical Center with the chief complaint of progressive weakness of both lower extremities for 2½ years and deterioration of gait for the past 2–3 months. For the past 4 years the patient had intermittently complained of dizzy spells, for which he had been seen about 12 times in the clinic. He noted dizziness only on standing for a long time in school and while kneeling for a long time in church. He also complained of occasional severe headache which could be relieved by walking. For at least 1 year he had been unable to keep up with his friends in sports owing to the weakness of the lower extremities.

Neurologic examinations had been repeatedly negative except for a lumbar puncture 3 years prior to admission which revealed a spinal fluid protein of 60 mg. per cent. There was never any sensory or reflex loss apparent until 2 months before admission, when bilateral Babinski reflexes were noted for the first time.

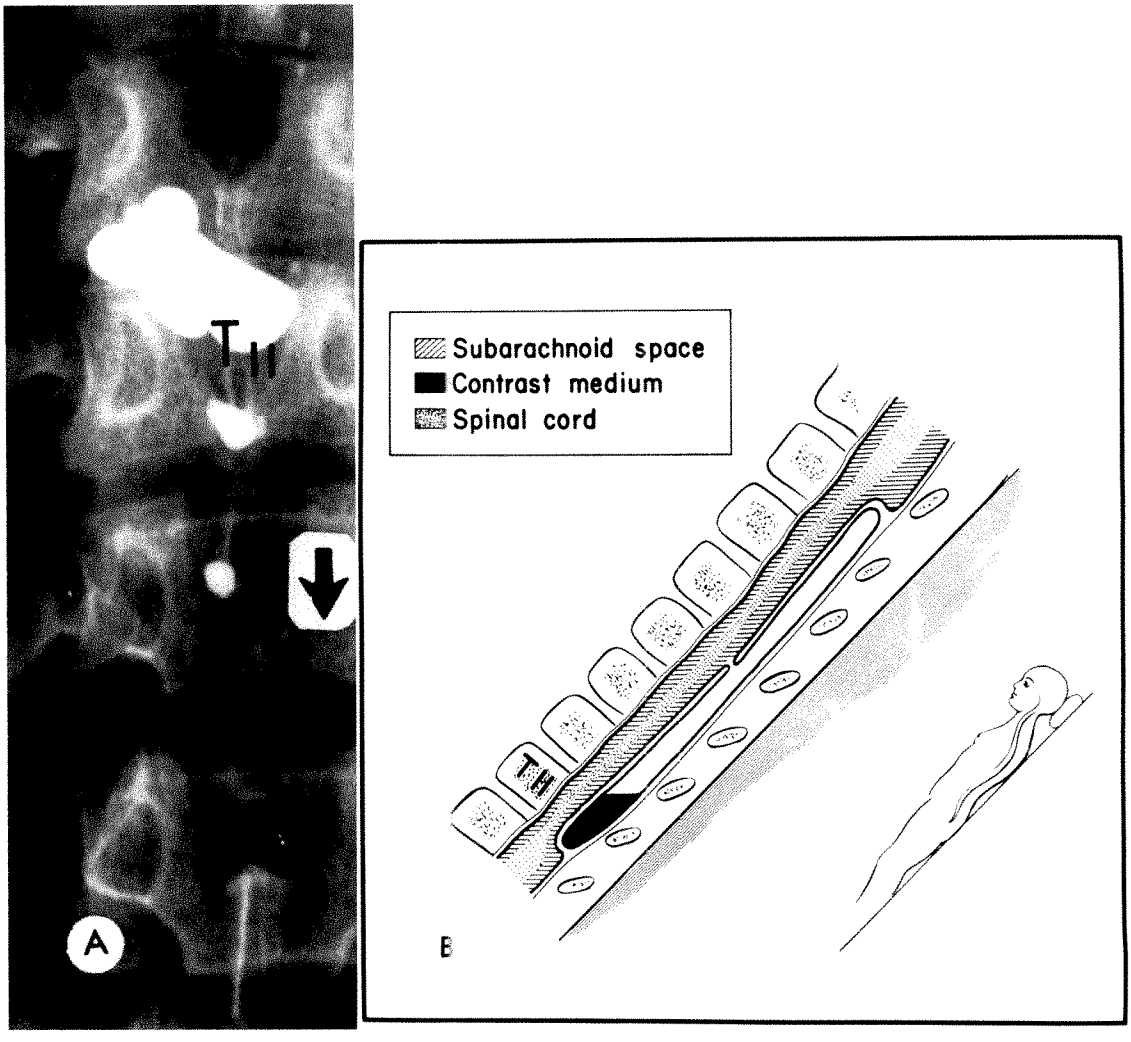



FIG. 4. Case 1. Supine position. (*A*) Descending myelography showing the contrast medium at the lower end of the cyst at T11. (*B*) Sketch showing the contrast medium remaining within the cyst in the supine position, thus outlining its lower end.

Physical examination revealed slight weakness of the quadriceps and hamstrings. The toes of both feet appeared weak. The patient walked with a spastic, shoveling gait, dragging the feet. All other muscles were strong. There was slight decrease in sensation to vibration, position and touch below the level of T4. There was a bilateral positive Babinski sign.

*Roentgenography.* A roentgenogram of the dorsal spine (Fig. 5) revealed atrophy and flattening of the pedicles and an increase in the interpedicular space from T3 to T9. There were 7 cervical and 5 lumbar vertebrae and 13 thoracic vertebrae. There was partial posterior fusion of the right 13th to the 12th rib

*Myelography.* The manometric opening pressure measured only 50 mm. $H_2O$. Three cubic centimeters of pantopaque were injected into the lumbar subarachnoid space. Myelographic findings were reminiscent of those in the sister, on whom myelography had been done 5 years previously.

(1) In the prone position, (Fig. 6*A*) on tilting the table head downward, a partial block was encountered at the T6–7 level. As in his sister's case, the T6–7 level lay above the lower level of vertebral pedicle changes of T9. A small amount of contrast medium, however, passed the T6 level, remained in the subarachnoid space and flowed to the cervical region.

(2) On placing the patient in the supine posi-

tion (Fig. 5*B*) and tilting the table head downwards, some contrast medium entered the extradural cyst and accumulated at the level of T2, thus outlining the upper cul-de-sac of the extradural cyst.

(3) In the supine position (Fig. 6*C*) with the table tilted feet downward, the contrast medium flowed caudad, remained inside the cyst and passed the T6–7 level, arresting at the T9–10 level, which then represented the lower end of the cyst.

Again, as in Case 1, the 3 levels of block were encountered and again the lower end of the cyst T9–10 was lower than the initial partial block level of T6–7 in the prone position, but corresponded with the level of pedicle changes. The similarity of the myelographic features in both cases was most remarkable.

*Family history.* The significance of the family history was not appreciated until this second case was encountered. The patient had an array of congenital anomalies which were also present throughout the family. The child had a double row of eyelashes, history of bronchial asthma and familial lymphedema of the right lower extremity (Milroy's disease) for approximately 2 years with progression. The older sister also had an extra row of eyelashes and an extradural cyst (Case 1). The occurrence of an extradural cyst in the second case, brother of Case 1, stimulated further interest in the remaining members of the family. Survey roentgenograms of the spine of the parents and 2 other younger sisters were therefore obtained. The dorsal spine of the youngest sister, (Fig. 7, *A* and *B*) aged $2\frac{1}{2}$ years, revealed widening of the interpedicular distances and flattening of the pedicles from T3 to T7. It is almost certain that she, too, has an extradural cyst. She is still asymptomatic and myelography has not been performed. She also has a double row of eyelashes and a history of asthma.

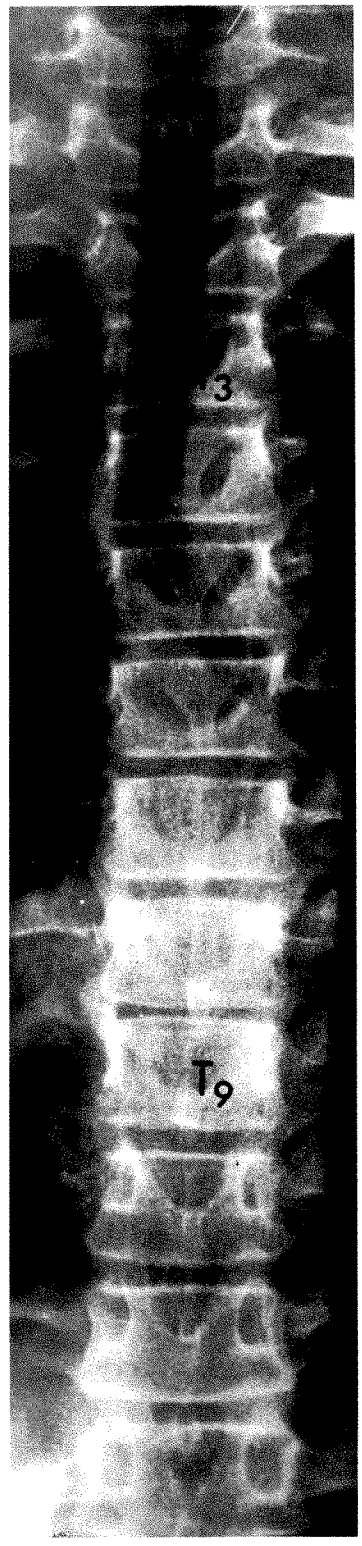



FIG. 5. Case II. Roentgenogram of the dorsal spine showing flattening of the pedicles and an increase in the interpedicular distances from T3 to T9.

## DISCUSSION

Since the clinical aspects of congenital spinal extradural cysts have been reviewed by Nugent *et al.*,[31] Wise and Foster,[45] and Smith and Chavez,[41] they will not be detailed here.

The exact number of cases of congenital extradural cysts recorded in the literature is not clear. Some authors even include traumatic cyst, postoperative pseudocyst, Lumbar or sacral meningeal cyst[11,26,27,42]

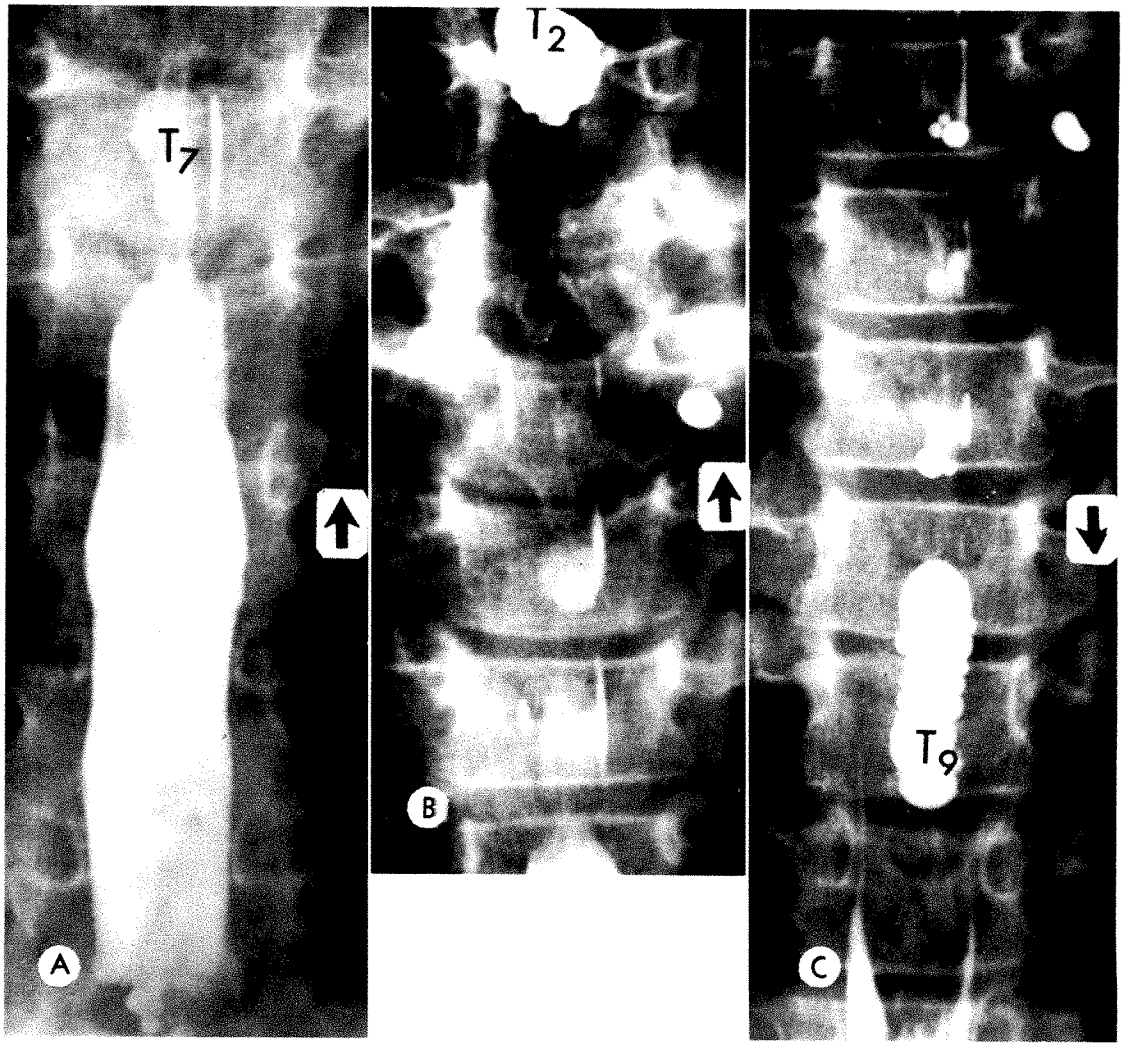



FIG. 6. Case II. Note the striking resemblance to Fig. 3*A*, and 4*A*, in Case 1. (*A*) Prone position. Ascending myelography showing extradural block at T6–7. (*B*) Supine position. Ascending myelography showing contrast medium now having entered the cyst and collected at its upper end at T2. (*C*) Supine position. Descending myelography showing that the contrast medium still within the cyst has collected at its caudal end at T9.

under spinal extradural cyst. In the author's opinion, the syndrome of congenital spinal extradural cyst is unique in its clinical and roentgenologic aspects, has been well-established in the literature, and should not be confused with other cysts. As Smith and Chavez[41] have already convincingly stated, the clinical findings in lumbar extradural cysts are extremely dissimilar from those in congenital extradural cyst in the dorsal level, and should be termed lumbar extradural cyst to avoid confusion. Extradural cysts of traumatic origin, as reported by Swanson and Fincher[44] and others,[20,28,29,38,39,43] have clinical and roentgenologic features also distinctly different from those of congenital extradural cysts.

Excluding other cysts mentioned above, 57 cases of genuine congenital extradural cyst (dorsal and dorsolumbar)[1–18,20–43] have been reported.

### SEX, AGE INCIDENCE AND SITE

The sex ratio between males and females is about 2:1. The overwhelming majority of

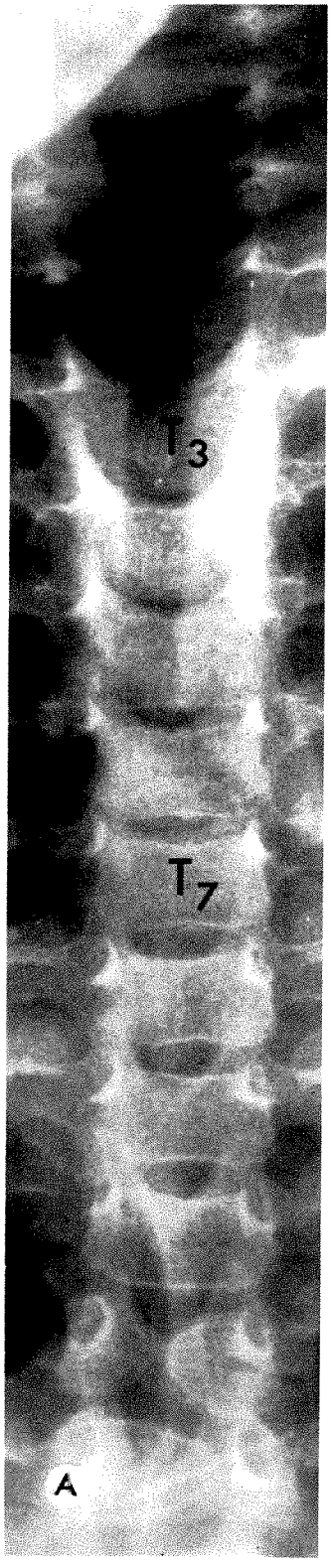



FIG. 7. (*A*) Roentgenogram of the dorsal spine of the youngest (2½ years) sibling showing hemivertebra of T11 and T12. Flattening and atrophy of the pedicles and increase in the interpedicular distances from T3 to T7.

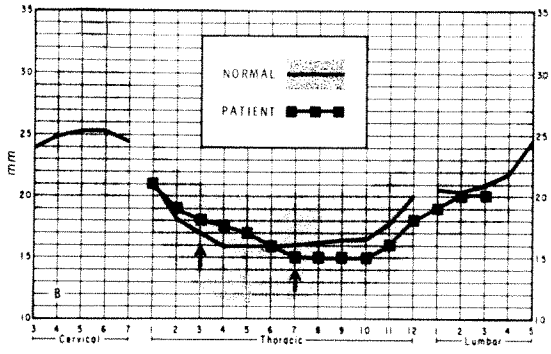



FIG. 7. (*B*) Graph showing abnormal ascending curve of patient's interpedicular distances from T3 to T7 as compared with the normal.

cases occur in adolescence, although cases in patients ranging from 6 to 64 years have been reported. The 2½ year old sister of Case I and Case II is the youngest patient ever recognized as having a congenital extradural cyst to be mentioned in the literature. The cyst occurs predominantly in the mid- and lower dorsal spine. Only a few cases have been reported to have extended down to the lumbar region from the thoracic level.

### PATHOGENESIS

Elsberg *et al.*[12] proposed the theory that these cysts were due to "a congenital diverticulum of the dura" or "a herniation of arachnoid through a congenital defect of the dura." Cloward and Bucy[4,5] stated that the cyst is probably an evagination of both dura mater and arachnoid membrane, the 2 layers fusing to one membrane under intracystic pressure. Because of the histologic similarity between the cyst walls and dura, most authors have accepted congenital diverticulum of the dura as the likely theory.

### CLINICAL FEATURES

Although earlier writers[24,30,36,37] including Dandy[8] have described intraspinal cysts which may well be the same as those here under discussion, it was not until

1934, that Elsberg, Dyke and Brewer[12] first established a distinct clinical and roentgenologic syndrome. Their description of the syndrome can hardly be improved upon and has been repeatedly quoted in the literature.

"The individual is in adolescence with the history and symptoms of progressive spastic paraplegia. Pain is absent or is not a prominent symptom. The objective disturbances of sensibility are slight, and their upper level is in the midthoracic region, usually the 6th or 7th thoracic dermatome. Manometric tests demonstrate a subarachnoid block with the characteristic spinal-fluid changes of cord compression. Measurements on antero-posterior x-ray films show that the interpeduncular space of three or more vertebrae somewhere between the 4th and 9th thoracic vertebrae are enlarged. Pedicles of the affected vertebrae, especially those of the 6th, 7th, and 8th, are narrowed and atrophic."

Lehman[25] added two more important features to the clinical syndrome, namely: (1) vertebral epiphysitis involving the vertebral bodies in the region of the lesion; and (2) marked fluctuation in the signs and symptoms of spinal cord compression. He suggested that the fluctuation corresponds to the variations in the amount of the fluid in the cyst owing to communication between the cyst and the subarachnoid space. Indeed, in a number of later papers, because of the fluctuation of the clinical course, the lesion was misdiagnosed as multiple sclerosis for some years.

Lehman[25] also observed a case in which kyphosis developed $3\frac{1}{2}$ years after operation and suggested that the disease process had probably already affected the vertebral bodies, since kyphosis does not commonly follow laminectomy in the absence of disease of verterbral bodies. In our Case 1, kyphosis developed about 1 year after laminectomy (Fig. 8, *A* and *B*). In the experience of Shenkin and co-workers,[40] the kyphosis is due to extensive laminectomy in young persons. Hamlin *et al.*[17] suggested the postoperative use of spinal brace to prevent kyphosis. Cloward and Bucy[5] believed that the bone changes in the vertebral bodies are those seen in kyphosis dorsalis juvenilis (Scheuermann's disease) and are due to impairment of venous drainage from constant compression of the veins by the cysts. Kyphosis, however, is not always associated with extradural cyst, as Cloward and Bucy originally assumed. It is present in about 40 per cent of cases.

In 1944, Good *et al.*[13] described the first cyst to fill with lipiodol.

### PLAIN ROENTGENOGRAPHY OF THE SPINE

The cyst usually extends more than 2 or 3 vertebrae. Almost all cases show widening of interpedicular distances and flattening and atrophy of the pedicles. The changes of the pedicles correspond to the extent of the cyst, thus involving most of the mid- and low thoracic spine and only occasionally extending down to the lumbar region.

It should be emphasized here again that in evaluating the interpedicular distances (IPD) of a patient, one should always compare the general direction of the IPD curve with that of the normal curve. Any significant deviation of the IPD curve from the normal is an indication of abnormality, even though the absolute measurement of the IPD may still be within the upper limits of normal range. This point is well illustrated on the plain roentgenogram of the dorsal spine and the accompanying graph in Case 1 (Fig. 1, *A* and *C*).

In the lateral views, exaggerated concavity of the posterior border of the vertebral bodies is often seen.

In our Case 1, the pedicles are seen to be fully reformed within 1 year, with normal interpedicular distances (Fig. 1*B*). In Kelly's case,[23] operated upon by Jefferson, the thinning of the pedicles disappeared 1 year after surgery, their width appearing to be normal again.

The lesion may be associated with vertebral epiphysitis with typical stepladder notching. Kyphosis may occur preoperatively owing to compression by the cyst of the venous drainage of the vertebral bodies,

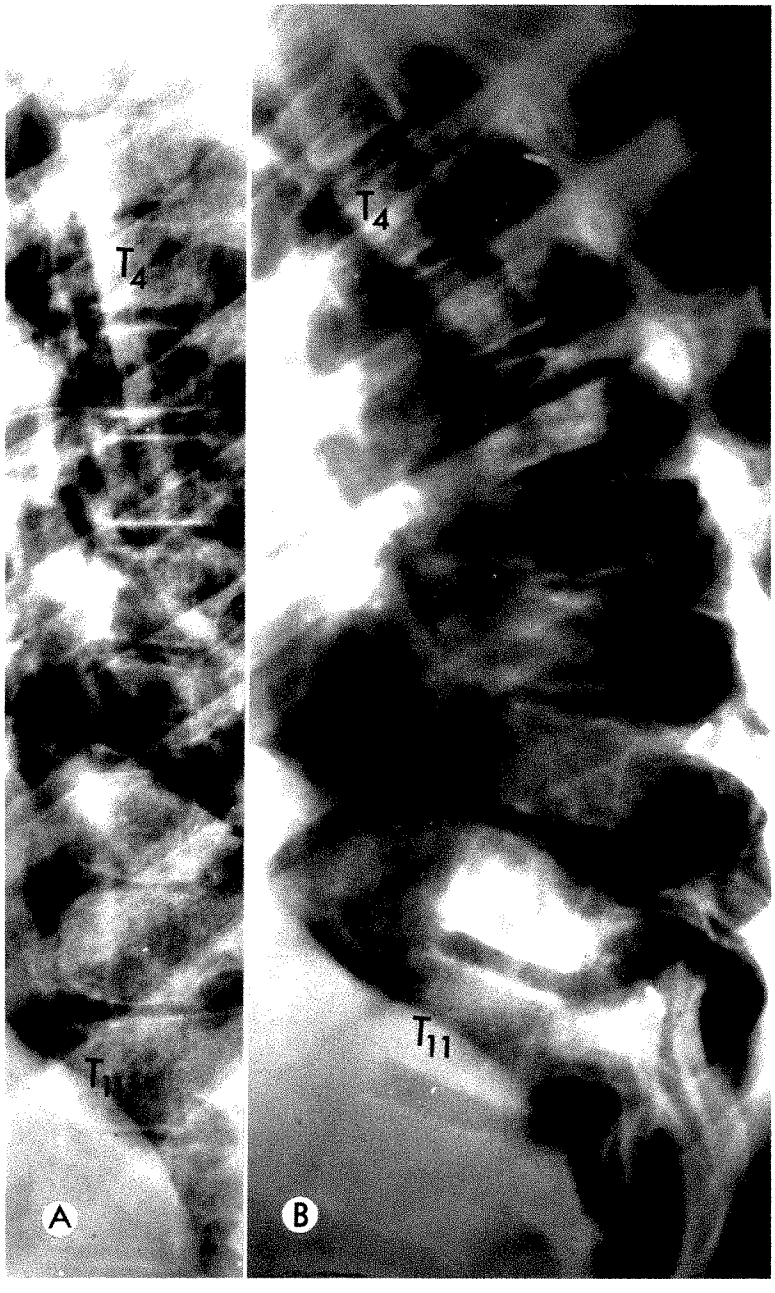



Fig. 8. Case 1. (*A*) Roentgenogram of the lateral dorsal spine before operation. (*B*) Marked kyphosis 1 year after laminectomy and removal of extradural cyst.

or postoperatively owing to extensive laminectomy in young adults. In our Case 1, kyphosis developed one year after laminectomy (Fig. 8*B*).

Cysts may also give rise to rounded paravertebral soft tissue shadows caused by their lateral extension.[9]

MYELOGRAPHY

The pedicle changes described above, although suggestive, are by no means absolutely diagnostic of extradural cyst. Similar pedicle changes have been observed in long-standing intramedullary glioma or syringomyelia. Myelography, when properly

performed, is the only examination which will permit a definite diagnosis.

As illustrated in our cases of 2 siblings, conventional prone examination with spot roentgenograms only reveals an extradural block of nonspecific nature. However, as clearly illustrated in the sketches, and because of the unique nature of this congenital cyst, namely that its opening is always on the dorsal aspect of the spinal cord, there was no difficulty in running the contrast medium into the cyst in both of our patients in the supine position, thus defining the upper and lower ends of the cyst and permitting an unequivocal diagnosis.

### ASSOCIATED ANOMALIES AND FAMILIAL OCCURRENCE

Only a few anomalies associated with the congenital cyst have been reported in the literature. In Balestrieri's case,[2] the extradural cyst coexisted with atresia of the foramina of Luschka and Magendie (the Dandy-Walker syndrome) and bone defect in the posterior arch of $C_4$. In Strang and Tovi's case,[42] there was a midline defect in the arch of the atlas. No associated Milroy's disease or double row of eyelashes, which occurred so widely in this family, has ever been recorded. Search of the literature reveals no evidence of a familial tendency, although survey spine roentgenograms of the family have never been mentioned. The closest approach to a familial incidence mentioned in the literature is Krauss' case,[24] operated upon by Dr. Roswell Park* only on condition that it be performed at the patient's home.

### SUMMARY

Two cases of congenital extradural cyst are reported. No similar cases occurring in the same family have ever been recorded in the literature. Myelographic examination in the conventional prone position revealed an extradural block of no specific nature. It was only in the *supine* position, that the contrast medium could flow into the cyst from the subarachnoid space, thus making a definitive diagnosis possible. Myelographic blocks at 3 different levels were almost identical in the 2 cases. The formation of the 3 myelographic blocks is explained.

The literature is briefly reviewed and the syndrome is described.

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* The patient, a 46 year old male, had 1 brother who had a history of some spinal trouble, nature not known. One sister died at the age of 34 years of so-called "paralysis."

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# NEUROPATHY FOLLOWING IRRADIATION*

## AN UNUSUAL LATE COMPLICATION OF RADIOTHERAPY

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PERIPHERAL neuropathy as a late consequence of radiation therapy is generally considered rare. Van Cleave[15] reviewed the effects of ionizing radiation on peripheral neural tissue, and his report draws entirely from the experimental literature. No adverse effects were noted in the dosage range or schedules followed in the usual clinical situation; however, electrophysiologic alterations were experimentally detectable in the megarad dose range.[2,5] On the other hand, adverse reactions of the central nervous system, particularly of the brain stem or spinal cord, are extensively documented by clinical experience.[3,7,14] This report describes a case in which radiotherapy was associated with delayed neuropathy of the brachial plexus.

### REPORT OF A CASE

B.T., U.M.H. #736273. This white female was initially admitted to the University of Minnesota Hospitals in September, 1943 at the age of 23 years. She complained of anorexia, fatigue, and weight loss of 17 pounds over the preceding 2½ months interval, and had noted recent progressive growth of a left supraclavicular mass which had been present for 1 year. The mass had originally been walnut-sized until 1 week before admission, but on examination at admission, was 6×9 cm., with several adjacent, satellite nodular masses. Chest roentgenograms showed widening of the mediastinum, and biopsy of the supraclavicular mass disclosed Hodgkin's disease. Radiation therapy was initiated using 250 kvp. roentgen rays, with a half value layer of 2.4 mm. Cu, and was directed to the mediastinum and to the left cervical region. Opposed anterior and posterior ports were used to treat the mediastinum with a midplane dose of 1,700 rads (1,750 r/air was delivered to each field). The left cervical region was treated by lateral, anterior and posterior oblique ports, with a tumor dose of 3,500 rads (1,500 r/air to lateral and anterior oblique fields, and 900 r/air to the posterior oblique field). Both sites received 14 treatments in 24 days. There was complete resolution of both the mediastinal mass and the lymph node masses in the neck. In the subsequent 12 years, the patient was completely free of disease; however, she developed essential hypertension and required, in 1953, hospitalization for hypertensive encephalopathy manifested by blurring of vision, dizziness and vomiting.

In 1955, a submental mass appeared about 3 months before admission and was 3 cm. in size when biopsied. Biopsy again showed Hodgkin's disease. Chest roentgenograms revealed a recurrent mediastinal mass, and hilar lymphadenopathy. The submental mass was treated with 250 kvp. roentgen rays, half value layer of 2.4 mm. Cu, delivering 2,100 r/air to a single anterior port for an estimated tumor does of 1,900 rads in 8 days (7 treatments). The mediastinal mass was treated with 1,200 r/air to anterior and posterior ports, and a mid-plane dose of 1,200 rads in 11 days (8 treatments). All lesions again regressed completely.

Over the next several years, the patient required active medical therapy for hypertension. Several episodes of headache, nausea and vomiting, blurred vision, leg edema, and markedly elevated blood pressures with blurred disks on fundoscopic examination were noted. Extensive diagnostic studies were performed. Renal angiograms in 1959 were negative. In 1960, a full neurologic evaluation disclosed an unsteady gait, bilateral Babinski signs, and weak facial musculature—findings consistent with diffuse encephalopathy due to the patient's hypertension.

By 1961, the hypertension was under control. Neurologic evaluation showed progressive re-

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pair of the diffuse findings; however, the patient complained of low back pain. Lumbar puncture, roentgenograms of the cervical-dorsal-lumbar spine and myelographic study of the entire spinal canal were negative.

In 1963, because of progressive weakness and pain of the left arm, she was readmitted for neurologic evaluation. Examination disclosed mild atrophy of left shoulder girdle, weakness of left deltoid, biceps, triceps and wrist dorsoflexor muscles; absence of deep tendon reflexes in the left arm, while present in all other extremities; and hypesthesia and hypalgesia of the lateral aspect of the left arm. Spinal fluid examination, and myelographic study again showed no abnormalities (Fig. 1). An electromyogram showed denervation of left deltoid and biceps muscles.

In 1966, the weakness of the left arm had progressed. Neurologic evaluation showed moderate paresis of the biceps, triceps, deltoid, and trapezius muscles on the left; absent deep tendon reflexes in the left arm; and sensory loss of thumb and index fingers of the left hand and medial left arm. There was no evidence of pyramidal tract involvement. Slight brawny edema of the left forearm and arm relative to the normal right extremity was apparent. Chronic radiation skin changes with marked skin atrophy and telangiectasia were present in the left neck region (Fig. 2). Electromyogram was compatible with a neuropathy of the C5, C6, C7 inervated muscles. A left axillary venogram (Fig. 3) was normal; a left upper extremity lymphangiogram (Fig. 4) disclosed atrophic lymph nodes only. Chest and cervical-dorsal-lumbar spine roentgenograms were within normal limits. In view of the minimal symptoms noted, the patient was again discharged for symptomatic management.

Since 1955 the patient has presented no evidence of active Hodgkin's disease. The neurologic findings of the left upper extremity, compatible with a partial brachial plexus palsy, have shown progression and have persisted despite reversal of diffuse findings attributed to hypertensive encephalopathy. The patient's blood pressure has also returned to upper limits of normal.

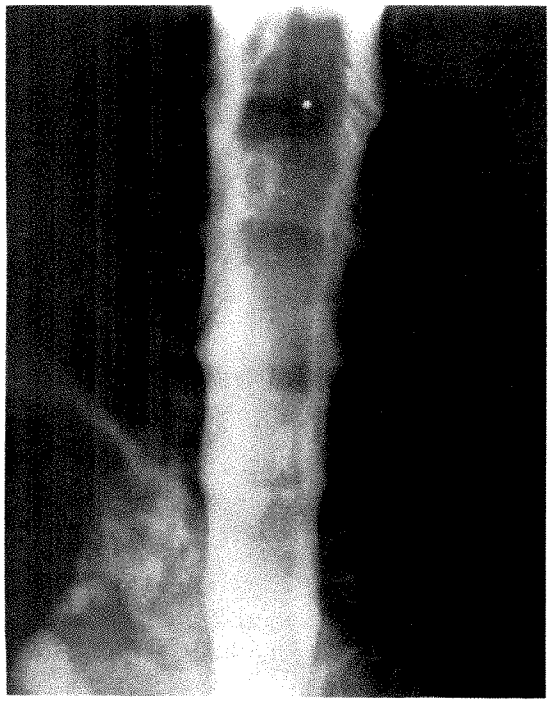


FIG. 1. Cervical-dorsal myelogram. There was no mass lesion or any obstruction to flow. The spinal cord did not show any radiographic evidence of abnormality.



FIG. 2. Appearance of left supraclavicular-cervical region. There were marked atrophic changes with evidence of incipient ulceration of the skin. The underlying subcutaneous tissues were solidly adherent to the skin in a fibrotic plaque.

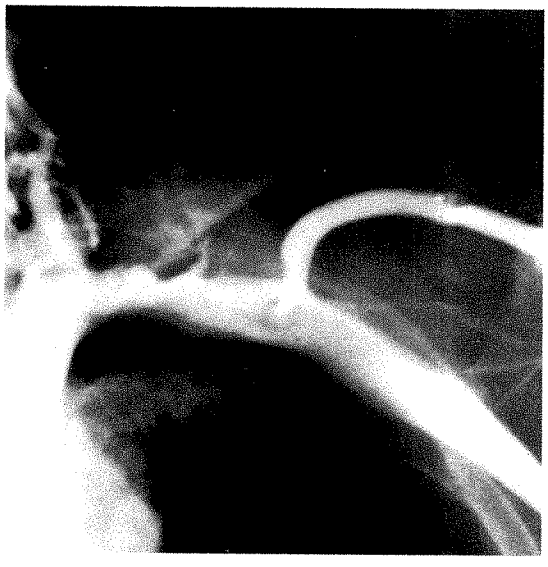


FIG. 3. Left axillary venogram. The study did not disclose any axillary masses.

## DISCUSSION

Tissue injury by ionizing radiation is the consequence of both direct cell injury, and of secondary factors contributed by injury to the supporting vasculo-connective tissues. For most tissues *in vivo*, one must distinguish between cell depletion, *e.g.* with tissues undergoing continuous renewal, and alterations of functional activity or physiology of a cell. The former mode of injury acts through alterations in cellular proliferation and renewal, and the latter through injury of membranes, organelles, enzymes or control systems, and transport or bioenergetic mechanisms. For mature and nonrenewing neural tissues such as peripheral nerves, little or no cell renewal, maturation, or proliferation occurs;[1,10] hence, they are relatively "radioresistant." Similarly, alterations of functional activity are only measurable with doses of ionizing radiation in the megarad range,[2,5] where extensive disruption of physiologic activity, as well as of cell membrane and transport mechanisms can be expected. Such doses of radiation are never used clinically. Hence, secondary factors are likely to be of greater importance than primary neural cell injury. In view of the fibrosis produced by orthovoltage roentgen rays, especially to skin and subcutaneous tissues,[6] the fact that the brachial plexus is anatomically located in a susceptible site, and in view of the extensive skin changes shown (Fig. 2), the pathogenesis of the clinical findings described is attributed to progressive perineural fibrosis. Perineural fibrosis and sclerosis of connective tissue around, and surrounding, the plexus with a secondary encasement of the emergent nerve sheaths has, in fact, been shown.[11]

The experience reported by Mumenthaler[11] was largely with patients afflicted with carcinoma of the breast, a situation where long survival and large proportions of patients surviving 5 years, 10 years, and longer periods, are frequently observed. Hodgkin's disease represents, similarly, a clinical affliction with an extended natural history, and the success of radiation therapy in the treatment of this condition is attested to by clinical experience,[4,12,13] and has led to the term "cure" by some.[4,8] In fact, this patient represents a more than 23-year radiotherapeutic success, and earlier reports from this department[12] indicate that such success was achieved in 77 per cent in early stages for periods up to 10 years. In view of the radical and aggressive radiotherapeutic attitude regarding Hodgkin's disease currently advocated,[9] late consequences of the type described, may be



FIG. 4. Left axillary lymphangiogram. The lymph nodes visualized were small and atrophic, without evidence of lymphomatous involvement.

encountered more frequently as permanent cures are achieved. Such late complications might possibly be avoided by the use of supervoltage radiation.

SUMMARY

A patient with Hodgkin's disease developing radiation neuropathy, involving the brachial plexus, as a late consequence of orthovoltage radiation therapy is described. Peripheral nerves are "radioresistant" to acute irradiation, but are susceptible to delayed (and secondary) alterations.

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# EVALUATION OF COMPLICATION RATES OF MEGLUMINE DIATRIZOATE AND MEGLUMINE IOTHALAMATE IN CEREBRAL ANGIOGRAPHY*

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THE purpose of this study was to technically evaluate a new contrast medium, meglumine diatrizoate, and to compare its safety with that of meglumine iothalamate, another non-sodium containing vascular contrast medium. It was the intent of the authors to randomly evaluate a large enough number of patients by double blind techniques to obtain a clinically significant comparison.

## CONTRAST MEDIA

Meglumine diatrizoate is not actually new, but has been used for some time as a constituent of hypaque-M 75 per cent and hypaque-M 90 per cent which contain 50 per cent (weight/volume) and 60 per cent, respectively, of meglumine diatrizoate in combination with sodium diatrizoate. Extensive laboratory toxicity studies have been performed which indicated the favorable toxicity of meglumine diatrizoate when administered to 4 different species of laboratory animals, *i.e.*, dogs, rabbits, rats and mice.[2] On the basis of these data meglumine diatrizoate was accepted for clinical trials.

Meglumine iothalamate (60 per cent) has been used extensively in cerebral angiography and in urologic studies. Most studies have indicated it to be one of the safest contrast materials for cerebral angiography.[1,3]

Both contrast media provide excellent visualization of the injected structures.

## MATERIALS AND METHODS

Two hundred and ninety-seven consecutive patients were included in the study. The contrast media were selected by a random method. None of the personnel involved in either the performance of the angiographic procedure, the interpretation of the roentgenograms or in the subsequent evaluation of the patients were aware of which contrast medium was being used. There were 203 percutaneous carotid angiograms (86 right, 117 left) and 94 percutaneous brachial angiograms (56 right, 38 left) (Table I). All of the injections were

TABLE I

CONTRAST VISUALIZATION STUDY SITE OF INJECTION, SEX, AGE

| | Conray | Meglumine Hypaque |
|---|---|---|
| *Site of Injection* | | |
| Brachial artery | | |
| Left | 17 | 21 |
| Right | 31 | 25 |
| | 48 | 46 |
| Carotid artery | | |
| Left | 54 | 63 |
| Right | 42 | 44 |
| | 96 | 107 |
| *Sex* | | |
| Male | 104 | 102 |
| Female | 40 | 51 |
| *Age* | | |
| 0–10 | 2 | 1 |
| 11–20 | 3 | 4 |
| 21–30 | 2 | 7 |
| 31–40 | 10 | 11 |
| 41–50 | 23 | 27 |
| 51–60 | 31 | 36 |
| 61–70 | 61 | 53 |
| 71 + | 12 | 14 |

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made by a mechanical injector, the Taveras Delux. The carotid injections were made at a pressure setting of 225 lb. per square inch through an 18 gauge, thin-walled Cournand needle or a modification of the Cournand needle. In very young children a No. 20 gauge thin-walled Cournand needle was used.

The percutaneous brachial punctures were made in the mid upper arm, using a 16 gauge thin-walled Cournand needle or a similar modification. The injections were made at a pressure setting of 625 lb. per square inch.

One hundred and forty-four studies were performed using meglumine iothalamate 60 per cent and 153 using meglumine diatrizoate 60 per cent. The same quantity of contrast medium was used in both groups. A scout roentgenogram employing 2 cc. of contrast medium was made in all cases. The subsequent study was performed with 8 to 10 cc. of contrast medium for carotid angiography and 40 to 80 cc. for retrograde brachial angiography. In all cases the bolus of contrast medium was delivered in 1 to 1½ seconds. All retrograde brachial injections were made using 40 cc. of contrast medium; however, 2 injections were made using 40 cc. of contrast medium for right brachial examinations (1 to study the neck vessels and another to study the intracranial circulation).

There were 206 male patients and 91 female patients. The age of the patients ranged from less than 1 year to 89 years, with the bulk of patients in the 5th, 6th and 7th decades (231 patients) (Table I). Many of the patients in this study were extremely ill, several were maintained on respirators at the time of angiography. One hundred and fifty-one patients had cerebrovascular disease; 33 had subdural, epidural or intracerebral hematomas or cerebral contusions; 28 had intracranial neoplasms, and 9 had aneurysms. There were 15 patients with miscellaneous conditions and 61 were angiographically normal (Table II). Technical complications ranging from tubing leakage to hematoma and subintimal blebs of various sizes were encountered in 27 patients receiving the contrast media studied (Table III).

TABLE II
CONTRAST VISUALIZATION STUDY MATERIAL

| Diagnosis | Conray | Meglumine Hypaque |
|---|---|---|
| Cardiovascular disease | 76 | 75 |
| Tumor | 19 | 9 |
| Trauma | 14 | 19 |
| Subarachnoid hemorrhage | 2 | 7 |
| Normal | 26 | 35 |
| Other | 7 | 8 |

It is believed that the random selection and number of patients studied negates the influence of patient's disease and its natural course as well as the effects of technical factors.

## RESULTS

Voluntary complaints after injection included moderate burning in the arm, face or neck, tingling, headache, unpleasant taste sensations and undifferentiated discomfort. Twenty-eight patients who received meglumine diatrizoate offered voluntary complaints and 20 of those with meglumine iothalamate had voluntary complaints (Table III). The patients were also asked specifically about pain or discomfort in the arm, neck or face. Thirty-seven patients who had meglumine diatrizoate injections admitted having moderate or severe painful sensations, whereas 30 patients who received meglumine iothalamate admitted such discomfort. Blood pressure changes of greater than 20 mm. Hg systolic were encountered in 8 patients receiving meglumine diatrizoate, but in only 1 patient who received meglumine iothalamate. Local spastic narrowing of the brachial artery was present nearly twice as often with meglumine iothalamate (22 patients) as with meglumine diatrizoate (12 patients) (Table III). Only 1 patient developed a new neurologic sign immediately after angiography, and only 1 patient showed any immediate

TABLE III

CONTRAST VISUALIZATION STUDY COMPLICATIONS

| | Conray | Meglumine Hypaque |
|---|---|---|
| *Technical Complications* | | |
| Subintimal bleb | 8 | 10 |
| Intramural injection | 1 | 0 |
| Tubing leakage | 0 | 1 |
| Hematoma | 0 | 2 |
| Extravasation | 2 | 2 |
| Local spasm | 0 | 1 |
| *Voluntary Complaints* | | |
| Burning in arm | 12 | 7 |
| Warmth in face | 2 | 3 |
| Burning in face and testicles | 1 | 1 |
| Taste in mouth | 2 | 2 |
| Headache | 1 | 1 |
| Pain in neck | 1 | 1 |
| Burning in face | 1 | 3 |
| General discomfort | 0 | 1 |
| Warmth | 0 | 1 |
| Burning in throat | 0 | 1 |
| Warmth in back and chest | 0 | 2 |
| Face flushing | 0 | 1 |
| Tingling all over | 0 | 1 |
| Discomfort in face and headache | 0 | 2 |
| Burning in chest wall | 0 | 1 |
| *Local Spasm* | 22 | 12 |
| *Burning in Arm* | | |
| Mild | 15 | 15 |
| Moderate | 15 | 16 |
| Severe | 5 | 3 |
| *Burning in Face* | | |
| Mild | 24 | 31 |
| Moderate | 9 | 18 |
| Severe | 1 | 0 |
| *Significant Blood Pressure Changes* | 1 | 8 |
| *New Neurologic Signs* | 0 | 1 |
| *Worsening of Present Neurologic Signs* | 0 | 1 |
| *Worsening of Neurologic Status* (1 week follow-up) | 2 | 3 |
| *Transient* | 1 | 3 |
| *Death* | 1 | 1 |
| *No Change* | 25 | 33 |
| *Improvement* | 10 | 6 |
| *Other* | 0 | 2 |
| | (a: claudication) | |
| | (b: hematoma-neck) | |

worsening of already present neurologic signs.

All patients who did not have an intervening neurosurgical procedure were evaluated 1 week after angiography. Many patients who were critically ill at the time of angiography continued on a down-hill course; while others, less ill, showed improvement. Thus it is unlikely that angiography was responsible for the worsening or improvement of their neurologic status. Nevertheless these figures are included in this study for completeness. Four patients who received meglumine iothalamate were worse. One patient died. Seven patients receiving meglumine diatrizoate were worse. One patient died. Ten patients who had received meglumine iothalamate and 6 who had received meglumine diatrizoate showed improvement. Two technical complications ascribable to angiography occured in patients who had received meglumine diatrizoate. One patient had claudication of the hand, which subsequently cleared on conservative therapy and 1 patient developed a large hematoma in the neck which had nearly resolved at the time of the 1 week follow-up.

## DISCUSSION

Complications of angiography can at times be ascribed to technical factors; however, a certain number of neurologic complications occur in what appears to be technically perfect examinations. Fortunately, recent improvements in contrast media have markedly reduced these complications and patient discomfort. Com-

TABLE IV

CONTRAST VISUALIZATION STUDY QUALITY

| Contrast Visualization | Conray | Meglumine Hypaque |
|---|---|---|
| Excellent | 32 | 28 |
| Good | 101 | 95 |
| Fair | 10 | 25 |
| Poor | 1 | 5 |

parative clinical studies between two relatively safe media require random double-blind techniques and enough patient material to make the study statistically valid.

## CONCLUSION

It seems apparent from this study that cerebral angiography is a relatively safe, definitive diagnostic procedure even in seriously ill patients. There is no significant difference between meglumine diatrizoate and meglumine iothalamate on the basis of subjective patient complaints or objective complications. The only parameter studied which demonstrated a significant difference between the 2 compounds was brachial artery spasm, which occurred nearly twice as often with meglumine iothalamate as it did with meglumine diatrizoate. Thus meglumine diatrizoate appears, on the basis of this clinical trial, to be as acceptable as meglumine iothalamate for cerebral angiography.

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# EXPERIMENTAL EVALUATION OF A NEW ANGIOGRAPHIC CONTRAST MEDIUM— ISOPAQUE 440*

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PROGRESS in vascular surgery in the past 15 years has been accompanied by widespread application of arteriographic procedures. Contrast angiography has become a routine part of the diagnostic armamentarium of radiographic facilities for the study of a wide variety of disorders. Currently available contrast media are relatively free of toxic side effects if the dosage and number of injections are limited. However, there are certain areas such as the coronary arteries and the thoracic aortic arch where adverse reactions are encountered even with the safest media.[3] Studies from our laboratory have shown that of the currently available concentrated angiographic contrast media the diatrizoates (hypaque M-90%, renografin 76% and renovist 69%) and the iothalamates (angioconray 80%) have the least neurotoxicity, nephrotoxicity and systemic toxicity.[1,2,7] However, even with these media repeat injection into the canine abdominal aorta is attended by a high incidence of neurotoxicity and nephrotoxicity.[5,6] The search for safer media must continue if the full potential of contrast angiography is to be realized.

Recently reported studies have indicated that the N-methylglucamine (NMG) salts of the currently available contrast media have less myocardial toxicity than the sodium salts of the same media.[4] It has also been suggested that the adverse effects of the sodium ion might be counteracted by the addition of calcium and magnesium ions to the solution. These observations led to the formulation of a "quad" salt medium containing the Ca, Mg, NMG, and Na salts of metrizoic acid.† It contains the salts in the following ratios: Na 47%, Ca 2.5%, Mg 0.8% and NMG 32%. The iodine content is 440 mg./ml. This medium has been submitted to a group of standardized toxicity tests in the dog. These standardized toxicity tests have previously been used to evaluate all of the currently available angiographic contrast media. Experiences with the animal toxicity testing of isopaque 440 prompt this report.

## METHODS

The standardized toxicity tests in dogs have been previously described and will be summarized briefly.

*The Aortic Injection Test.* Under general anesthesia (intravenous veterinary nembutal, 30 mg./kg.) and using sterile technique the canine abdominal aorta was exposed at laparotomy. The tip of a No. 18 needle was inserted in the abdominal aorta at the level of the renal arteries. Fifteen milliters of isopaque 440 was injected over a period of 5 seconds. The animal was observed for evidence of immediate neurologic reaction (*i.e.*, hyperextension, hyperreflexia or seizure of hind limbs). After obtaining hemostasis at the puncture site, the abdominal wound was closed. The animal was then observed daily for evidence of hind limb weakness or paralysis. The serum nonprotein nitrogen values were determined every other day for comparison with preinjection level. At the end of one week the animal was sacrificed and bilateral renal biopsy obtained.

*The Repeat Aortic Injection Test.* In a second group of animals the Aortic Injec-

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† Supplied as Isopaque 440 by Winthrop Laboratories, 90 Park Avenue, New York, New York 10016.

tion Test was done and followed in 15 minutes by another identical injection. The same observations were made as outlined above.

*The Intravenous Injection Test.* In this test a P.E. 240 catheter was inserted in a femoral vein, under local anesthesia, and advanced into the thoracic inferior vena cava. Isopaque 440 was injected rapidly at the rate of 12.5 ml./second, the dosage ranging from 5 to 9 ml./kg. body weight. The animals were observed for changes in neuromuscular activity, cardiopulmonary status and the occurrence of rapid death.

*Selective Renal Artery Injection Test.* Renal toxicity was assessed by the injection of contrast medium into the isolated renal arteries. The abdominal aorta was exposed under general anesthesia using sterile precautions. The segment of aorta from which the renal arteries arise was mobilized from all surrounding structures. A curved vascular clamp was placed across the aorta such that the aorta was transiently occluded above and below the origin of the renal arteries. A volume of 15 ml. of isopaque 440 was injected into the isolated aortic segment manually; the injected solution passed undiluted into the renal arterial bed. Following injection the vascular clamp was immediately removed and flow to the renal arteries reestablished; the period of aortic occlusion was less than 1 minute. The aortic puncture bleeding was controlled by digital pressure following which the laparotomy incision was repaired. Serial serum nonprotein nitrogen determinations were performed preoperatively and on alternate postoperative days until demise or sacrifice of the animals at 2 weeks. A rise of the nonprotein nitrogen to a value greater than 50 mg. per cent was arbitrarily termed *azotemia*. Elevation of the nonprotein nitrogen to a value greater than 100 mg. per cent was arbitrarily termed *severe azotemia*. Renal specimens obtained at autopsy were fixed, mounted and stained for histologic examination. The histologic changes were graded according to the severity of renal tubular damage. *Minimal* changes consisted of scattered hydropic degeneration of the tubular epithelium without necrosis. *Moderate* changes consisted of diffuse degeneration with tubular proteinacious casts. *Severe* changes consisted of massive tubular necrosis. When the changes in one kidney were more severe than those in the other, the more severe designation was recorded.

## RESULTS

*The Aortic Injection Test.* Two of the 10 dogs had evidence of an immediate neurologic reaction in the form of hyperreflexia and hind limb extensor rigidity. None of the animals had a hind limb seizure (Table I). In the postanesthetic period there was no evidence of neurologic deficit in any animal. Similarly the serum blood urea nitrogen remained normal and histologic study of the kidneys at the end of 1 week revealed no evidence of contrast medium injury.

*The Repeat Aortic Injection Test.* Eleven of 14 dogs evidenced an immediate neurologic reaction in the form of hind limb hyperreflexia or extensor rigidity; 3 of the 11 with an immediate reaction had a hind limb seizure (Table II). In the postanesthetic period there was no evidence of a neurologic deficit in any animal, nor was there a single instance of azotemia. The histologic study of the kidneys 1 week postinjection showed no evidence of significant renal tubular or glomerular damage.

*The Intravenous Injection Test.* The 25 animals receiving rapid injection of isopaque 440 in dosages of 5–9 ml./kg. body weight into the thoracic inferior vena cava all survived (Table III). Typically the animals developed tachypnea 1–2 minutes following the injection and 10–20 minutes following the injection a marked bradycardia occurred and persisted for the next 30 minutes. None of the animals had a seizure or other evidence of neurologic disorder. There were no deaths.

*Selective Renal Artery Injection Test.* Eighteen dogs were subjected to isolated renal artery injection as described. Six animals succumbed within 48 hours and did

TABLE I

THE AORTIC INJECTION TEST—ISOPAQUE 440

| Dog No. | Weight (kg.) | Neurologic Reaction | | | | | Serum Blood Urea Nitrogen | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Immediate | Postanesthetic | | | | Preinjection | Day | | |
| | | | Day 1 | 3 | 5 | 7 | | 2 | 4 | 6 |
| 22K | 12.0 | o | o | o | o | o | 14 | 12 | 16 | 14 |
| 23K | 11.2 | o | o | o | o | o | 15 | 14 | 16 | 13 |
| 29K | 9.8 | o | o | o | o | o | 18 | 16 | 14 | 15 |
| 30K | 8.8 | o | o | o | o | o | 20 | 21 | 18 | 19 |
| 38K | 10.0 | o | o | o | o | o | 14 | 12 | 10 | 8 |
| 40K | 11.6 | ++ | o | o | o | o | 15 | 13 | 14 | 12 |
| 42K | 10.5 | o | o | o | o | o | 16 | 14 | 14 | 14 |
| 43K | 12.4 | o | o | o | o | o | 18 | 16 | 15 | 12 |
| 44K | 11.4 | + | o | o | o | o | 12 | 14 | 15 | 13 |
| 45K | 10.8 | o | o | o | o | o | 11 | 12 | 14 | 12 |
| | | 2/10 | 0/10 | | | | | 0/10 | | |

o = No reaction.
+ = Immediate reaction (1–4 plus scale).

not survive long enough to permit postoperative evaluation of renal function. Twelve animals survived beyond 2 days. One animal succumbed as a result of progressive azotemia on the fifth postoperative day. All other animals survived to be sacrificed 2 weeks postoperatively.

The nonprotein nitrogen value reached a peak greater than 50 mg. per cent in 10 out of the 12 surviving animals (Table IV). The

TABLE II

THE REPEAT AORTIC INJECTION TEST—ISOPAQUE 440

| Dog No. | Weight (kg.) | Neurologic Reaction | | | | | Serum Blood Urea Nitrogen | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Immediate | Postanesthetic | | | | Preinjection | Day | | |
| | | | Day 1 | 3 | 5 | 7 | | 2 | 4 | 6 |
| 98K | 11.8 | +++ | o | o | o | o | 15 | 12 | 14 | 14 |
| 69K | 12.8 | +++ | o | o | o | o | 17 | 16 | 14 | 13 |
| 74K | 11.2 | + | o | o | o | o | 20 | 18 | 14 | 15 |
| 75K | 10.8 | + | o | o | o | o | 19 | 15 | 13 | 16 |
| 520J | 11.8 | + | o | o | o | o | 15 | 14 | 12 | 13 |
| 705J | 13.4 | + | o | o | o | o | 16 | 15 | 13 | 15 |
| 99K | 12.0 | o | o | o | o | o | 22 | 21 | 22 | 21 |
| 101K | 10.2 | + | o | o | o | o | 22 | 21 | 19 | 20 |
| 450J | 10.0 | o | o | o | o | o | 20 | 13 | 12 | 15 |
| 813J | 9.9 | o | o | o | o | o | 12 | 15 | 15 | 18 |
| 58K | 10.5 | + | o | o | o | o | 15 | 13 | 19 | 14 |
| 59K | 8.6 | + | o | o | o | o | 19 | 14 | 14 | 15 |
| 60K | 11.8 | ++ | o | o | o | o | 13 | 12 | 15 | 16 |
| 126K | 11.2 | + | o | o | o | o | 17 | 15 | 12 | 15 |
| | | 11/14 | 0/14 | | | | | 0/14 | | |

o = No reaction.
+ = Immediate reaction (1–4 plus scale).

azotemia was fatal in the one animal mentioned previously. The average peak nonprotein nitrogen value was 98 mg. per cent. In most animals the nonprotein nitrogen had stabilized and had begun to fall by the time of sacrifice 2 weeks postoperatively. Histologic grading of the kidneys of the 12 animals that survived to permit evaluation of renal function revealed minimal pathologic changes in 5 animals, moderate changes in 6 animals and severe changes in only 1 animal (Table IV).

## DISCUSSION

The results obtained with the Repeat Aortic Injection Test and the Intravenous Injection Test show isopaque 440 to be considerably less toxic than angioconray or hypaque M-90%.[5,6,7] The Selective Renal Artery Injection Test indicated that isopaque 440 is slightly less toxic than either angioconray or hypaque M-90%. However, it should be noted that isopaque 440 contains less iodine (20–40 mg./ml.) than angioconray or hypaque M-90%.

Previous studies with single Aortic Injection Test showed that urokon 70% will produce virtually a 100 per cent incidence of paraplegia, azotemia and renal tubular necrosis.[2] In contrast, angioconray and hypaque M-90% were free of any neurotoxic or nephrotoxic effects under exactly the same conditions. However, when these same media were used in the Repeat Aortic Injection Test, a high incidence of neurotoxicity was encountered and angioconray also had transient nephrotoxic effects in a significant percentage of the animals.[5] Thus under these conditions isopaque 440 was far less toxic than angioconray or hypaque M-90%. Neurologic reactions to isopaque 440 were limited to immediate neurologic reactions; none of the animals had a postanesthetic neurologic deficit. With either hypaque M-90% or angioconray at least 50 per cent of the animals had either hind limb paralysis or weakness.

With the Intravenous Injection Test, injection of hypaque M-90% resulted in 100 per cent mortality when the dosage exceeded 4 ml./kg. and angioconray resulted in 26, 70, and 73 per cent mortality at the 5, 6, and 7 ml./kg. dosages.[1,7] In contrast isopaque 440 produced no significant abnormality and no deaths even at the 9 ml./kg. dosage. This would seem to indicate an extremely low cardiopulmonary toxicity and may indicate suitability for angiocardiography, coronary arteriography and thoracic aortography.

The Selective Renal Artery Injection Test is an extremely severe one, but is analogous to the inadvertent injection of the entire abdominal aortographic dosage of a contrast medium into a renal artery. Under the circumstances isopaque 440 proved to be less toxic than other commonly employed radiopaque media.[6]

TABLE III

THE INTRAVENOUS INJECTION TEST—ISOPAQUE 440

| Dosage (ml./kg. body weight) | Number of Animals | Lived | Died |
|---|---|---|---|
| 5 | 5 | 5 | 0 |
| 6 | 5 | 5 | 0 |
| 7 | 5 | 5 | 0 |
| 8 | 5 | 5 | 0 |
| 9 | 5 | 5 | 0 |

TABLE IV

RESULTS OF ISOLATED INFUSION OF ISOPAQUE 440 INTO THE RENAL ARTERIES

| Animal | Peak Nonprotein Nitrogen (mg.%) | Outcome (2 wk.) | Histologic Changes |
|---|---|---|---|
| 65 | 58 | Survival | +2 |
| 67 | 225 | Survival | +2 |
| 68 | 225 | Survival | +3 |
| 71 | 105 | Survival | +2 |
| 72 | 69 | Survival | +1 |
| 76 | 48 | Survival | +1 |
| 79 | 55 | Survival | +1 |
| 80 | 68 | Survival | +1 |
| 86 | 56 | Survival | +1 |
| 87 | 87 | Survival | +1 |
| 90 | 156 | Death* | +2 |

* Succumbed on the fifth postinjection day as the result of progressive azotemia.

## CONCLUSION

Experimental canine toxicity testing of isopaque 440 shows it to be less toxic than any of the currently employed concentrated angiographic contrast media as regards nephrotoxicity, neurotoxicity and cardiopulmonary toxicity.

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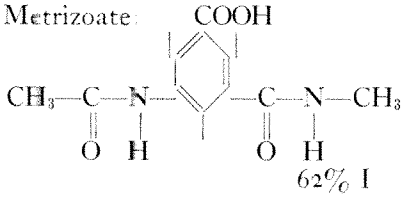
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# ISOPAQUE 440 (METRIZOATE); A NEW CARDIOVASCULAR CONTRAST MEDIUM*

## EXPERIENCE WITH 100 CONSECUTIVE CASES

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ISOPAQUE 440* is a contrast medium which contains a mixture of the sodium, calcium, magnesium, and N-methylglucamine salts of metrizoic acid. Its chemical formula is as follows:




The ratio of salts is Na 47%, Ca 2.5%, and N-methylglucamine 32%; the iodine content is 440 mg./ml. The Ca and Mg appear in the same ratio as that normally found in plasma. The N-methylglucamine was added to the compound in order to keep the Na at a reasonably low level to maintain a low viscosity. The addition of Mg and Ca was made to counteract any adverse reaction to the Na ion. For isopaque 440, the acute intravenous $LD_{50}$ in mice, rats and rabbits is 17,000–19,000 mg./kg. (a low degree of toxicity). In many pharmacologic tests on dogs that simulated the clinical situation, isopaque 440 was found to have less of an effect upon the cardiovascular system than compounds such as hypaque (diatrizoate) and angiocon ray (iothalamate) (currently used in angiocardiography).[1]

### METHOD

Beginning on September 21, 1965, 100 consecutive patients were injected with a contrast medium containing a mixture of the sodium, calcium, magnesium and N-methylglucamine salts of metrizoic acid (isopaque 440). Studies of the cardiovascular system were performed in ambulatory and hospitalized patients of all ages and both sexes. The ages ranged from 1 week to 81 years; there were 60 males and 40 females (Table I). The patients were instructed to abstain from food in the morning, or, if the examination was to be performed later in the day, the afternoon meal was to be omitted. The dosage for intravenous angiocardiography was 1 ml./kg. in 75 patients, and 1 to 1.5 ml./kg. in 25 patients (aortography) (Table II). All of the patients had advanced cardiac, pulmonary, and/or mediastinal disease. Although every patient was questioned regarding previous reaction to contrast agents or allergies, none was denied the examination. The contrast agent was injected percutaneously or via a cutdown with the single arm pressure

TABLE I

DISTRIBUTION OF AGES OF PATIENTS ADMINISTERED ISOPAQUE 440

| Age Range (yr.) | Number of Patients |
|---|---|
| 0–9 | 3 |
| 10–19 | 4 |
| 20–29 | 3 |
| 30–39 | 9 |
| 40–49 | 10 |
| 50–59 | 30 |
| 60–69 | 24 |
| 70–79 | 16 |
| 80–89 | 1 |
| 90–100 | — |
| Total | 100 |

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TABLE II

DISTRIBUTION OF STUDIES PERFORMED USING ISOPAQUE 440

| | Intravenous Angiocardiography | Intravenous Aortography |
|---|---|---|
| Number of Cases | 75 | 25 |

technique.[7] A preliminary test injection of 1 ml. was negative in the first few patients to whom it was administered; subsequently it was not routinely employed.

## RESULTS

No serious reaction occurred in any of the 100 patients to whom isopaque 440 was given. Minor reactions, such as urticaria, gave little discomfort. Indeed, in 5 patients no treatment was required because the urticaria was mild, transient, and well tolerated. Only 3 patients required antihistamine treatment (intramuscular or intravenous) with benadryl. Other reactions (headache, heat, cough, dizziness, chills, vomiting, sneezing, tachycardia, and hypotension) occurred only rarely, and were of no clinical importance (Table III).

All patients were monitored by continuous electrocardiographic recordings. Regularly, inversion of the T waves and tachycardia were present during intracardiac passage of the contrast material,[4] especially when it reached the coronary arterial system. That other electrocardiographic changes occur became evident from study of continuous electrocardiographic tracings made during the injection of isopaque 440 of 3 patients. Since similar electrocardiographic changes occur transiently during injection of other contrast media (hypaque and angioconray), additional electrocardiographic tracings were not made.

Although a positive history of urticarial reactions to foods, pollens and drugs was ascertained in 22 patients, it did not hinder the injection of isopaque 440 (Table IV). Only the first 4 patients had a preliminary dose of 1 ml. isopaque 440 intravenously. All were negative, and because this type of testing has never been found of value in predicting reactions, it was not used in the subsequent 96 cases.

No relation of the injection of isopaque 440 to concomitant drug therapy was present even though the majority of the patients were receiving drug treatments on the day of the contrast study.

Twenty subjects had a repeat study (2 injections) soon after the films of the first study were viewed. One patient had a series of 3 injections—2 for angiocardiography in frontal and lateral views, and the third for biplane supravalvular aortography. In 79 patients, a single injection was sufficient for diagnosis (Table V). This experience warrants the conclusion that isopaque 440, like the other commercially available contrast media (hypaque and angioconray), may be safely reinjected. At our center, despite the claims made by other investigators, no evidence of the emergence of sensitivity by repeated injections of contrast material has ever been found. More important than allergy is the danger of dehydration after injecting large dosages of concentrated contrast material.

## DISCUSSION

Isopaque 440 gave excellent visualization of the cardiovascular structures in 85 per cent of the cases (Table VI). Since a cleansing enema or cathartic is not a routine preliminary for patients undergoing angiography and abdominal aortography, the excellent rating might have been even higher had these procedures been made. However, the discomfort produced by these measures hardly seems worth the effort. Because so many other factors, as for example the quality of the roentgenographic technique, the development of the film, and the efficiency of the injection apparatus enter into the angiographic study of a patient, such a record is indeed remarkable. However, this experience with isopaque 440 did not differ from the results secured after other contrast agents. It should be

TABLE III

ADVERSE REACTIONS TO ISOPAQUE 440

| Case No. | Electrocardiographic Changes | Skin Reactions | Respiratory Reactions | Neurologic Reactions | Other |
|---|---|---|---|---|---|
| 1 | Tachycardia during injection with ST depression and isoelectric T waves | | | | Chills |
| 3 | | | | | Localized thrombophlebitis |
| 11 | | | Sneezing | Headache | |
| 14 | | Urticaria—given benadryl | | | |
| 16 | Tachycardia, T wave inversion | | | | |
| 31 | | | Wheezing | | |
| 37 | | Urticaria—no treatment | | | |
| 40 | | | | Dizziness | |
| 45 | | Urticaria—no treatment | "Croupy" cough | | |
| 50 | | | | Severe headache lasting 10 minutes | |
| 58 | | Urticaria—given benadryl | | | |
| 59 | | | Sneezing | | |
| 61 | Extra systoles (ventricular), prolonged PR intervals | | | | |
| 63 | | | | Vomiting | |
| 72 | | Urticaria—no treatment | | Severe heat | |
| 74 | | Urticaria—no treatment | | | |
| 87 | | Urticaria—no treatment | | | |
| 90 | | Urticaria—given benadryl | | | |

TABLE IV

HISTORY OF ALLERGIC REACTIONS IN PATIENTS GIVEN ISOPAQUE 440

| | Drug Allergies | Other Allergies |
|---|---|---|
| Number of Cases | 14 | 8 |

attributed rather to the proficiency of the angiography team (doctor, technician and nurse), and to the use of superb, modern electronic roentgenographic and accessory equipment.

Because all of the injections of isopaque 440 were made with a mechanical injector, it was not possible to accurately determine the value of the low viscosity of the compound. With the injector apparatus, delivery of contrast substance to the vein, cardiac, or arterial structure was effective in every case.

## SUMMARY AND CONCLUSIONS

In a series of 100 cases, injection of isopaque 440, with dosages of 1–1.5 ml./kg. for visualization of the cardiovascular system proved to be safe and without major reactions. Minor reactions, such as urticaria, headache, nausea, vomiting, dizziness, and hypotension, were rare and transient. The usual minor electrocardiographic changes, common with contrast media, such as tachycardia, bradycardia, and inversion of the T waves occurred when the left heart structures were opacified. A history of allergy to drugs, foods or pollens and preliminary testing with the compound were valueless for predicting reactors.

Excellent opacification of the cardiovascular structures under examination was secured in 85 per cent of the cases. In 15 per cent, it was good. Although direct comparison with other contrast agents, hypaque and angioconray, was rarely possible, it would appear that isopaque 440 is equal in contrast to other commercially available contrast media. A high degree of skill of the angiographic team, excellence of modern, high-powered roentgenographic equipment, and ancillary mechanical devices, rather than the contrast substance, merit praise for this accomplishment.

Because no hand injections were made, the value of the low viscosity of isopaque 440 was not determined. No difficulty, however, was encountered with passage of the contrast compound through the special 12-gauge Robb-Steinberg cannula or cardiac catheters inserted into the heart or great vessels.

Therefore, it would appear that isopaque 440 is a relatively safe and easily administered contrast agent. Although serious reactions were absent in 100 cases, and minor reactions transient and unharmful, cardiac and pulmonary arrest, and ventricular fibrillation are always possible. Accordingly, every patient receiving contrast material should have electrocardiographic monitoring during the injection of contrast material. The angiographic team should also be trained to resuscitate patients who develop major reactions. Maintenance of the airway and respiratory and defibrillatory apparatus should be immediately available to prevent fatality following major reactions. Certainly, isopaque 440 (metrizoate) is a valuable addition to the list of contrast agents available for angiography. It is equal in radiopacity to the other commercially produced compounds for opacification of the cardiovascular structures. Although isopaque 440 is a highly satisfactory contrast agent for

TABLE V

NUMBER OF INITIAL AND REPEATED INJECTIONS WITH ISOPAQUE 440

| | 1 | 2 | 3 |
|---|---|---|---|
| Number of Cases | 79 | 20 | 1 |

TABLE VI

QUALITY OF VISUALIZATION WITH ISOPAQUE 440

| | Excellent | Good or Satisfactory |
|---|---|---|
| Number of Cases | 85 | 15 |

angiography, search for the ideal, completely innocuous, highly concentrated, and nonreactive radiopaque medium should continue.

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# A COMBINED FLUSHING AND PULSE-PRESSURE MONITORING SYSTEM FOR PERCUTANEOUS CATHETERIZATION AND ANGIOGRAPHY*

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ANGIOGRAPHY utilizing percutaneous arterial catheterization has become an indispensable radiodiagnostic method and is now widely employed in community hospitals as well as in large medical centers. Although complications of the technique are infrequent, they may, on rare occasion, be serious.

The purpose of the authors is to describe a simple system for continuous monitoring of the arterial pulse contour and pressure at the catheter tip, combined with a convenient catheter flushing system, the use of which, we believe, adds some measure of safety to percutaneous catheterization and angiography.

We have employed this system routinely at the Mayo Clinic for the past 7 years. It is in a large measure due to the interest shown in its use and the many questions posed by visiting radiologists in regard to the details of this system that this description is submitted.

## DESCRIPTION

Periodic flushing of the catheter during percutaneous catheterization for angiography is necessary to ensure that clotting does not occur in the catheter tip. For this purpose, the catheter may simply be attached to a saline filled Luer-Lok syringe fitted with a stopcock, from which small amounts of saline can be injected at intervals through the catheter. A drip system also has been employed by some. Although such flushing systems are simple, they do not permit monitoring of the arterial pulse contour and pressure.

The system herein described is illustrated in Figures 1, 2 and 3. It permits periodic flushing of the catheter, as well as continuous oscilloscopic display of the pulse contour. The approximate arterial pressure also may be displayed as a function of the height of the systolic and diastolic peaks of the pulse curve. By calibration of the oscilloscope from 0 to a point above the patient's arterial systolic pressure, using midchest reference position, the pressure can be measured oscilloscopically within $\pm 5$ mm. of mercury of true arterial pressure. The electrocardiogram (ECG) may be displayed simultaneously on the dual-trace oscilloscope, if this is desired, although the pulse curve itself will serve as a monitor of cardiac rate and rhythm.

Subsequent to insertion of the catheter into the artery for angiography, the catheter is connected, via a rotating adapter stopcock with 360° swivel and male Luer-Lok connector, to a strain gauge, which is connected to the oscilloscope. A flushing bypass is interposed between the rotating adapter stopcock and the strain gauge. A flush bottle containing heparinized saline solution is pressurized (above arterial pressure) by means of a Tycos bulb and gauge. The pressurized saline bottle is connected by a length of Tygon tubing and a stopcock to the flushing bypass, with an air trap interposed between the flush bottle and the bypass to ensure that no air is inadvertently injected.

The catheter may be flushed by simply turning the flushing bypass stopcock on and off, and the flush response is observed on the oscilloscope. The quick return of the flush to base line indicates that the catheter is open and free of obstruction.

In the case of intramural passage of the catheter tip, the pulse contour may or may not be altered significantly, but there will

* From the Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

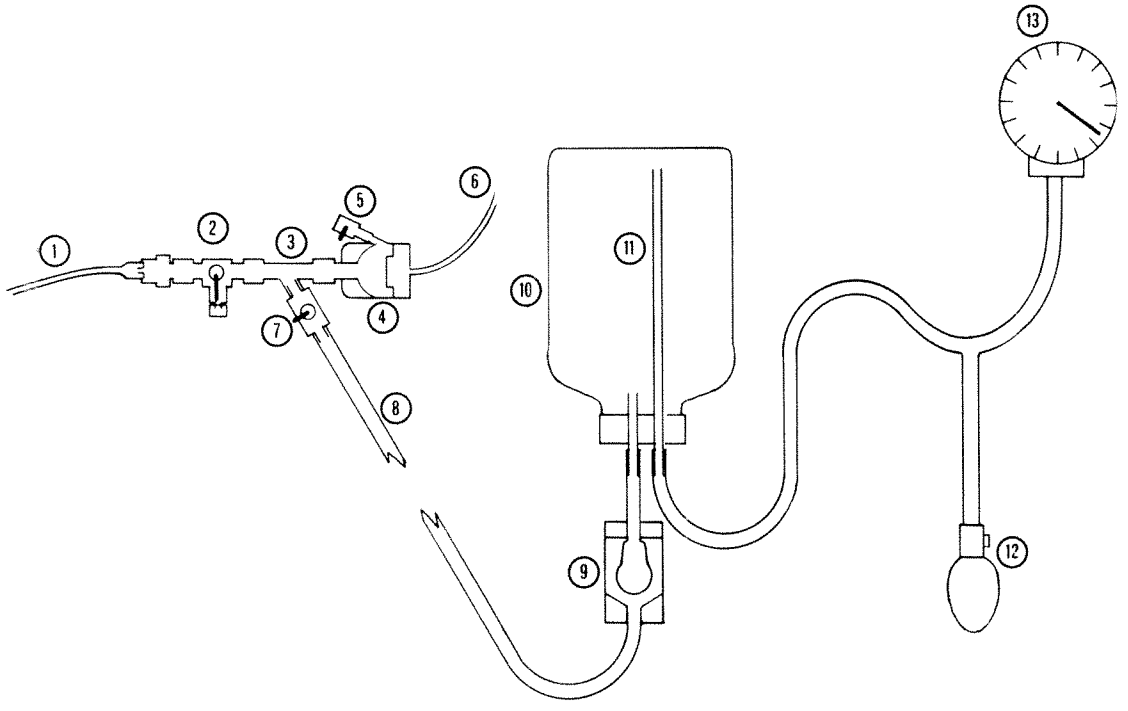



FIG. 1. Combined flushing and pulse-pressure monitoring system: (1) catheter; (2) three-way rotating adapter stopcock; (3) flushing bypass; (4) strain gauge; (5) one-way stopcock for cleaning and sterilizing strain gauge; (6) wire to strain-gauge power supply and oscilloscope; (7) one-way flushing stopcock; (8) 3/16 inch ID Tygon tubing from flush bottle to flushing bypass; (9) air trap; (10) flush bottle; (11) air inlet; (12) Tycos bulb; and (13) Tycos manometer.

be some drop in pressure as indicated by the lowering of the systolic and diastolic peaks of the displayed pulse curve. Clotting within the catheter or, in the case of selective catheterization, wedging of the catheter tip in a branch artery is signaled by immediate damping or disappearance of the pulse curve and a slow return of the oscilloscopic beam to base line after flushing of the catheter.

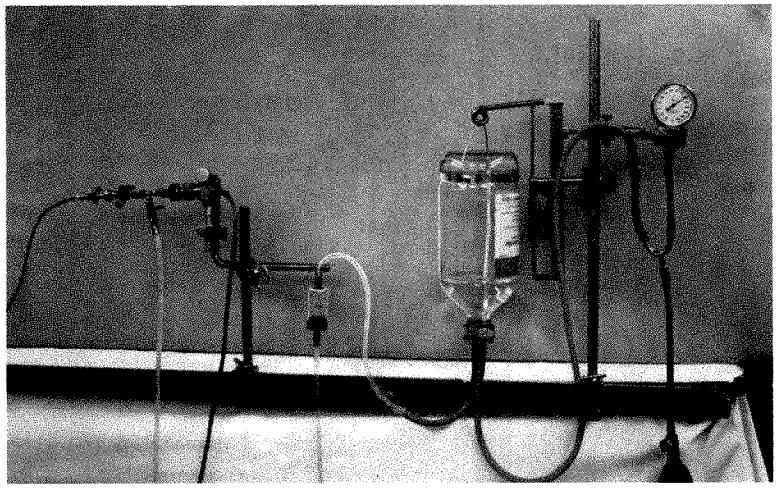


FIG. 2. System attached to side rail of table.

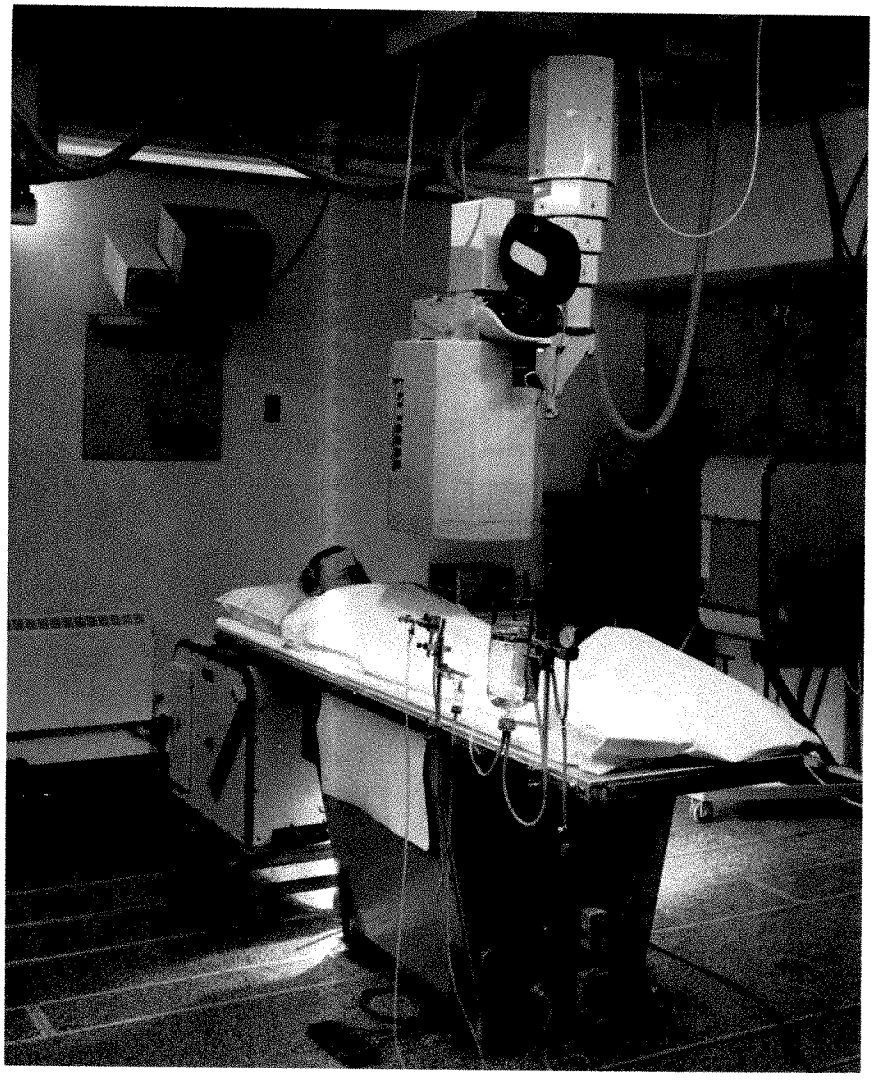


FIG. 3. Room and equipment showing system attached to side rail of table. Oscilloscope and fluoroscopic TV monitor mounted in background. Tabletop and image intensifier in position for fluoroscopy. Ceiling-mounted overhead roentgenographic tube is out of picture to left.

The additional female side arm on the rotating adapter stopcock permits the connection of a small Luer-Lok syringe for the test injection of contrast medium without disconnecting the catheter.

The entire system, except for the oscilloscope, the ECG, and strain-gauge D-C power supply, is mounted with screw clamps on the table rail and moves with the patient when the table top is moved. The use of a fairly long piece of tubing between flush bottle and the flushing bypass and a wire of generous length between strain gauge and its power supply permits the strain-gauge assembly to be moved to various positions on either side of the table without moving the rail-mounted flush bottle. The oscilloscope itself may be mounted in any convenient location in the room. We have found it most convenient to mount it alongside the fluoroscopic television monitor.

As long as the flush-bottle pressure is maintained above arterial pressure during the procedure, no contamination of the flushing system should occur. If the flush-

bottle pressure is permitted to fall below the patient's arterial pressure, however, blood will enter and contaminate the strain-gauge assembly and connecting tubing. If contamination occurs, the flush-system tubing and strain-gauge assembly are sterilized in zephiran solution (1:1,000) prior to use on the next patient. Unless such contamination occurs, the tubing and strain-gauge assembly are sterilized only at the beginning of each day's work.

## SUMMARY

A combined flushing and pulse-pressure display system for percutaneous catheterization and angiography is easy to assemble, convenient to use, and adds some measure of safety to the procedure. Clotting in the catheter, intramural passage of the catheter tip, or, in the case of selective angiography, wedging of the catheter tip in a branch artery may be detected immediately by observing the oscilloscopic display of the pulse pressure contour and the response to flushing. In addition, the pulse curve serves as a convenient monitor of cardiac rate and rhythm. The oscilloscope may also be used for display of the electrocardiogram during angiography, if more precise cardiac monitoring is desired.

The Mayo Clinic
Rochester, Minnesota 55901

# THE SHELDON CAROTID CANNULA*

*By* R. S. LEIGHTON, M.D.†
SEATTLE, WASHINGTON

IN 1964, Sheldon[3] described the development of a cannula designed to facilitate safe and dependable carotid arteriography. He reported at that time his experience with more than 1,200 examinations, with an unusually low incidence of complication. Complication in this context means not only neurological complication but also, and particularly, dissection in the vessel wall. There were no complications related to the use of the cannula.

In our hospital virtually all carotid studies are done by residents in training. While the number of dissections occurring in their hands was not unusually large, we felt this complication to be far commoner than it should be. Considerable experimentation with various devices had left us unsatisfied so the system introduced by Sheldon was tried with most gratifying results.

Briefly, the system consists of a blunt cannula; 18 G thin wall, 7–8 cm. in length, a very sharp inner stylet for puncturing the artery; and a short flexible guide wire of the Seldinger type (Fig. 1).* The puncture of the artery is performed with the sharp stylet held firmly in the cannula. The stylet is withdrawn as soon as the artery has been punctured. The cannula is very slowly withdrawn until a good arterial flow is obtained. The hub of the cannula is then slightly depressed to place it in the axis of the vessel and the guide is introduced. We have found this convenient if the hub of the cannula is held with the thumb and middle finger of the left hand for the withdrawal, leaving the index finger free to stop blood flow from the cannula when the lumen is entered. Meanwhile the guide is held ready in the right hand. When the guide has been inserted to a depth of 1–2 cm. beyond the cannula it is held firmly in the right hand and the cannula advanced along it with the left hand. The guide is then withdrawn and the cannula con-

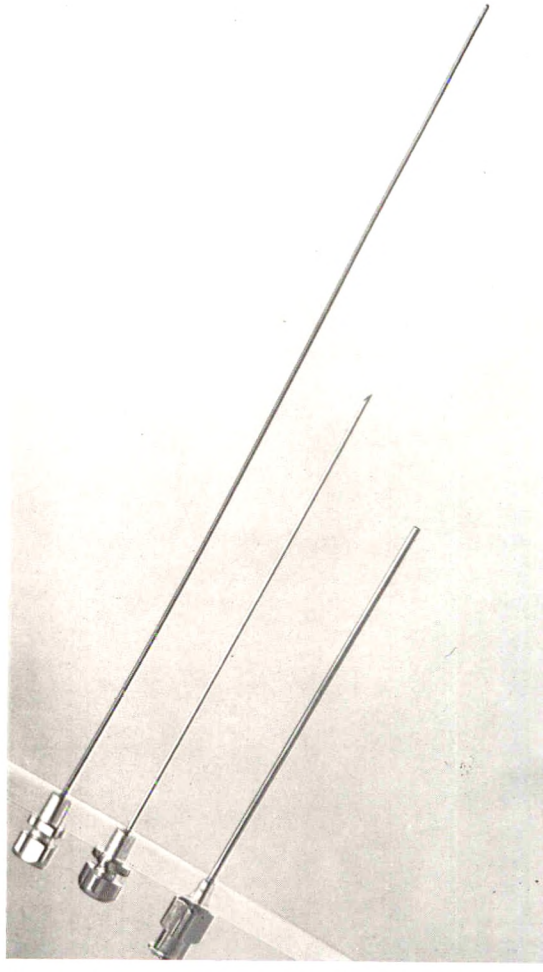


FIG. 1. The cannula set showing blunt cannula, sharp inner stylet and flexible guide wire.

* A carotid artery set composed of cannula, sharp stylet and short guide was prepared for us by Electro Catheter Corporation, Linden, New Jersey. It differs from Sheldon's original set only in that it uses Luer fittings and does not have an adjustable chuck on the guide. These differences do not appear important. The set will shortly be marketed by the manufacturer.

* From Radiology Service, Veterans Administration Hospital, Seattle, Washington.
† Chief, Radiology Service, Veterans Administration Hospital, Seattle, Washington; Associate Professor of Radiology, University of Washington Medical School.

nected to a plastic tube-syringe set up containing irrigating solution. If the puncture is made in the common carotid artery, selective cannulation of the internal or external branches is usually quite easy. This is facilitated by injecting a small amount of contrast material into the common carotid and observing the position and character of the carotid bifurcation with the image intensifier and altering the angle of the cannula slightly so that insertion of the guide into the desired branch can be accomplished directly. That the cannula carries little risk of trauma to the artery when properly used is indicated by the fact that several times it has been advanced farther than necessary and has entered small branches of the external carotid (occipital, lingual) (Fig. 2) and an injection of contrast medium made. No harm was detectable to artery or patient.

We have used this system in over 300 examinations with great satisfaction. Only one instance of dissection of the arterial wall has occurred and in this case the physician who performed the study stated that he was sure he had inserted the guide too vigorously (*vide infra*). As in all methods of arterial puncture, certain precautions are mandatory.

1. A satisfactory central puncture of the artery is necessary.

2. The guide must be inserted with extreme gentleness. When it protrudes only millimeters beyond the cannula it is splinted thereby. If it does not go in easily, either the position of the cannula must be modified slightly or a new puncture made.

3. The cannula must be inserted only far enough to accomplish the purpose of the examination. Too deep an insertion into the internal carotid may give rise to arterial spasm.[4] Too deep insertion into the external carotid may result in the cannulation of a branch (*vide supra*).

4. The inner stylet must be kept very sharp. When this is done the puncture becomes easier than with the usual carotid needle since the passage of the cannula through the arterial wall is readily felt.

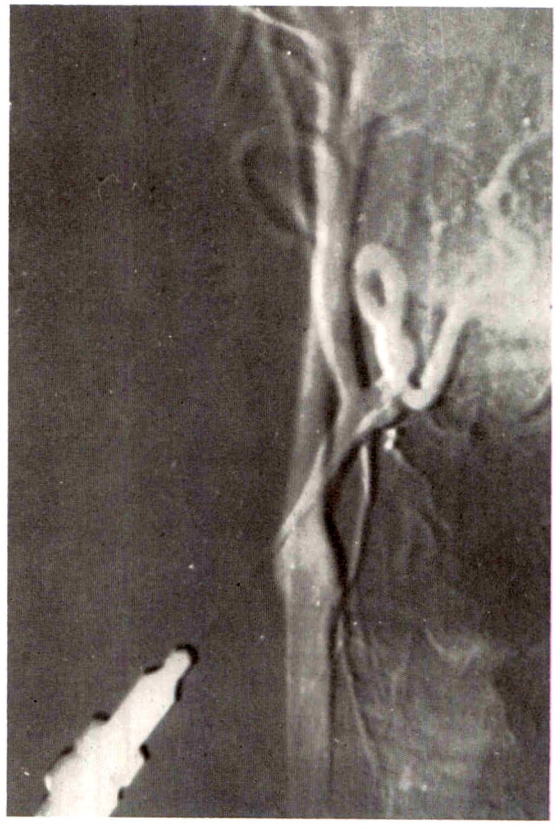


Fig. 2. Offset reproduction of roentgenogram showing the cannula tip in the lingual artery.

5. The cannula must never be inserted beyond the end of the guide. Even a blunt cannula can puncture the wall of a vessel with surprising ease.

We now use this needle for all our arteriographic work including catheter studies since the use of a long guide is easier and less worrisome when one knows that the cannula is threaded well into the arterial lumen.

For examinations in which a pressure injection through the cannula is to be used (*e.g.* aortic arch studies per axillary artery), the hub of the cannula may be sutured to the skin but in most applications this is unnecessary. Generally, changes in position of the patient into oblique or submental vertical posture are done with ease and security without suture.

VOL. 101, NO. 1

# THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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*Sixty-eighth Annual Meeting:* Washington Hilton Hotel, Washington, D. C., Sept. 26–29, 1967.

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*Representative on the Board of Chancellors of the American College of Radiology:* George G. Stetson.

The Fiftieth Annual Meeting of the Society will be held at the Hotel Fontainebleau in Miami Beach, Fla., April 7–11, 1968.

# EDITORIALS

## INSTRUCTION COURSES OF THE SIXTY-EIGHTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

PRESIDENT-ELECT C. Allen Good, Chairman of the Program Committee, asked the Section on Instruction that it continue the fruitful policy of the past by arranging a series of Instruction Courses also for the Sixty-eighth Annual Meeting of the American Roentgen Ray Society, to be held at the Washington Hilton Hotel, Washington, D. C., September 26–29, 1967.

As is known, these courses were originated nearly three decades ago by Dr. Edward L. Jenkinson, President-Elect for 1939, who at the Fortieth Annual Meeting of the Society in Chicago, with the Executive Council's approval, founded the Section on Instruction. The courses arranged by this Section had at once proven so successful, that since then every President-Elect requested their uninterrupted continuation as an essential part of the overall program of the Annual Meeting. The JOURNAL over these years gave faithful expression, through its pages, to the many important successive phases characterizing the scientific and educational facets of these courses. Even a cursory perusal of these pages reveals the tremendous advancement that has taken place in all branches of clinical radiology and the allied sciences of physics, radiobiology, radiochemistry, technology and other disciplines of timely interest. Dissemination of knowledge relating to these amazing achievements has become an ever broadening task of the Section on Instruction.

Since 1939, 3 outstanding Directors guided in shaping the pattern for fulfillment of this task: Dr. B. R. Kirklin, 1939–1948; Dr. H. M. Weber, 1948–1957; and Dr. Harold O. Peterson, 1957–to the present (with exception of 1964, his tenure of office as President-Elect, when Dr. Donn G. Mosser substituted).

The Director of the Section on Instruction for the Sixty-eighth Annual Meeting is again Dr. Harold O. Peterson, and the Associate Director, Dr. Donn G. Mosser. As usual, they have selected a distinguished Faculty of Instructors who, in conforming with Dr. Peterson's intent, will conduct the courses insofar as possible in a relatively informal manner, epitomizing the latest contributions to the various subjects chosen.

The Section this year will present a total of 39 courses of the following categories: (1) Diagnostic Roentgenology: 19 single period, 9 double period, and 1 triple period courses; (2) Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology: 8 single period, 1 double period, and 1 quadruple period courses. This latter course is sequential and will be given on special invitation by Robert Gibb, M.D., Ch.B., F.F.R., Deputy Director of Radiotherapy, Christie Hospital and Holt Radium Institute, Manchester, England.

A complete list of the Faculty and a brief abstract of each course, as well the schedule of the courses and the registration form with the necessary instructions, have been published in the August issue of the JOURNAL. An early registration is advisable.



# GILBERT W. HEUBLEIN, M.D.
## 1908-1967

DR. GILBERT WHIPPLE HEUBLEIN died in Carton, Connecticut on May 27, 1967, from a primary malignant chest tumor after an illness of one year. He is survived by his wife, the former Anne A. Kritz, a sister, Mrs. W. H. Sterg O'Dell of Devon, Pennsylvania, and two sons, two daughters, and five grandchildren. He was the son of another prominent radiologist in Hartford, Connecticut, Dr. Arthur C. Heublein, who founded the Department of Radiology at Hartford Hospital and who developed techniques for body spray irradiation at Memorial Hospital in New York City prior to his death in 1932.

Dr. Gilbert Heublein graduated from the Medical School at Yale University in 1934 after receiving a B.S. degree from Yale in 1931. He then served an internship at Hartford Hospital after which he was trained in radiology under Dr. Eugene Pendergrass at the University of Pennsylvania where he received the degree of D.Sc. (Radiology) in 1939. He then returned to

Hartford where he remained in the private practice of radiology and on the staff of Hartford Hospital for the remainder of his life except for an interval during the war years between 1942 and 1946. During that interval he was stationed at Percy Jones General Hospital, where he became Chief of the X-Ray Department. He was discharged from the army as a Lieutenant Colonel in 1946.

The attainments of Dr. Heublein were numerous. He was the author of a number of scientific publications. He was on the staff of the Institute of Living, where he was Chief of the X-Ray Department. He was also Chief of the X-Ray Department at Rocky Hill Veterans Hospital from 1946 until 1956. He was on the staff of the Newington Hospital for Crippled Children, and he was on the Senior Staff at Hartford Hospital. He was a consultant radiologist at U.S. Veterans Hospital in Newington, Connecticut, and at Windham Memorial Hospital in Willimantic, Connecticut. He was Visiting Professor of Radiology at Jefferson Medical College in Philadelphia. He belonged to all the national radiological societies and he was a Fellow of the American College of Radiology. He was a founder and the first President of the Connecticut Valley Radiological Society. He was on the Council of the American College of Radiology and a Director of the Connecticut State Radiological Society. It is hardly necessary to list any more of his numerous connections.

Gil, as he was familiarly known to everyone, enjoyed especially the convivial company of men as well as outdoor activities. He was an expert fisherman and he was a member of the Farmington Country Club, the Hartford Gun Club, and the Limestone Fish and Game Club. Although financially independent, it is greatly to his credit that he was a hard working conscientious and very capable radiologist who seldom found time to go fishing and to indulge in the other outdoor activities which he enjoyed so much.

WENDELL C. HALL, M.D.

Hartford Hospital
Hartford, Connecticut

# NEWS ITEMS

## TWENTY-FIFTH ANNIVERSARY OBSERVANCE OF THE FIRST NUCLEAR CHAIN REACTION

The University of Chicago

Six Nobel Laureates will be among the speakers at the Twenty-fifth Anniversary Observance of the First Nuclear Chain Reaction to be held December 1 and 2, 1967, at The University of Chicago.

They are Melvin Calvin, University of California at Berkeley (Chemistry, 1961); Sir John Cockcroft, Churchill College, Cambridge, England (Physics, 1951); Willard F. Libby, University of California at Los Angeles (Chemistry, 1960); Glenn T. Seaborg, Chairman of the U. S. Atomic Energy Commission (Chemistry, 1951); Emilio Segre, University of California at Berkeley (Physics, 1959); and Eugene P. Wigner, Princeton University (Physics, 1963).

Twelve other distinguished scientists and scholars have been invited to participate in the two-day observance program.

The participants will be welcomed by George Wells Beadle, President of The University of Chicago and a 1958 Nobel Laureate in Medicine and Physiology.

Chairman of the committee planning the 25th Anniversary Observance is Herbert L. Anderson, Professor of Physics and in the Enrico Fermi Institute for Nuclear Studies at The University of Chicago. Professor Anderson was one of the group of 40 scientists and technicians who, under the leadership of the late Enrico Fermi, designed and executed the experiment which produced the world's first self-sustaining nuclear reaction on December 2, 1942.

Retrospective and prospective views will be presented during the observance. These include the implications of nuclear biology and chemistry, nuclear medicine in its diagnostic and therapeutic phases, and the use of nuclear energy as a power source for industry and public utilities.

A highlight of the observance will be the dedication of a sculpture "Nuclear Energy," a work by the famous British artist Henry Moore, which was commissioned for the occasion. The 12 foot sculpture is being cast in bronze by Hermann Noack in Hamburg, Germany and will be placed on the site of the 1942 experiment in Stagg Field on the University campus.

Featured at the end of the observance will be the world premiere of a documentary film on the developments which led to the first nuclear chain reaction. The film is being produced by the motion picture unit of Argonne National Laboratory, in cooperation with the U. S. Atomic Energy Commission.

It was 3:25 P.M. on December 2, 1942, when Fermi ordered the cadmium-plated control rod withdrawn from Chicago Pile Number One. For 28 minutes a nuclear chain reaction continued within its depths. The total energy produced was only about one-half watt—enough to alter the course of history. When the pile was shut down the late Arthur Holly Compton, Nobel Laureate physicist in charge of Manhattan Project operations at Chicago, telephoned the Project's over-all scientific chief, James B. Conant, President of Harvard University. "The Italian navigator has landed in the New World," he told Conant. "How were the natives?" Conant asked. "Very friendly," replied Compton.

Their improvised exchange has become perhaps the best known secret code of the 20th century.

The co-ordinator for the program of the 25th Anniversary Observance is Mrs. Judith Reiffel of the Office of Special Events at The University of Chicago.

For further information please contact The Office of Public Relations, The University of Chicago, Chicago, Illinois 60637.

## MUSEUM OF HISTORICAL RADIOLOGY

The Museum of Historical Radiology will definitely open in October, 1967, in

some 2,000 square feet of air conditioned space on the 29th floor (adjacent to the Home Office of the American College of Radiology) at 20 North Wacker Drive, Chicago, Illinois.

The origin of medical museums is obscured by antiquity. Evidence indicates that there was such a museum in Alexandria as early as 300 years before Christ. The Museum of Historical Radiology will be the newest in a long succession and will embody the best as determined by the Museum Committee under the Chairmanship of Dr. M. H. Poppel and the Curator, Mr. Robert T. Morrison.

The museum is now a member of the American Museum Association. It will also have formal liaison and reciprocal relationships with the National Library of Medicine at Bethesda, Md.

It is the prime project of the American College of Radiology Foundation, which has now opened a subscription drive for membership.

For further information please write to the American College of Radiology Foundation, 20 North Wacker Drive, Chicago, Illinois 60606.

## TWELFTH ANNUAL CLINICAL CONFERENCE
### Neoplasia in Childhood

"Neoplasia in Childhood" will be the subject of the Twelfth Annual Clinical Conference to be held at The University of Texas M. D. Anderson Hospital and Tumor Institute at Houston on November 9 and 10, 1967. Co-sponsor of the conference with Anderson Hospital is The University of Texas Graduate School of Biomedical Sciences at Houston, Division of Continuing Education.

Chairman for the two-day conference is Dr. H. Grant Taylor, Chief of the Section of Pediatrics at Anderson Hospital and Dean of The University of Texas Graduate School of Biomedical Sciences at Houston, Division of Continuing Education. Co-chairman is Dr. W. W. Sutow, Associate Pediatrician and Associate Professor of Pediatrics at Anderson Hospital.

The conference will comprise sessions on four topics: (1) current concepts basic to the management of childhood cancer; (2) techniques for approaches and evaluation; (3) specific tumor therapy of 5 of the more common childhood solid tumors (Wilms' tumor, neuroblastoma, rhabdomyosarcoma, retinoblastoma, and bone cancer); and (4) specific therapy for the leukemia-lymphoma group of diseases.

The visiting speakers include: Dr. Sidney Farber, Boston, Massachusetts; Drs. Robert Miller and Frank Rauscher, Jr., Bethesda, Maryland; Dr. William Clatworthy, Jr., Columbus, Ohio; Dr. William G. Thurman, Charlottesville, Virginia; Drs. Merle K. Loken and Giulio J. D'Angio, Minneapolis, Minnesota; Dr. Isaac Djerassi, Philadelphia, Pennsylvania; Sir Stanford Cade, London, England; Dr. Joseph H. Burchenal, New York, New York; Dr. Derek Jenkin, Toronto, Canada; and Dr. John S. Stehlin, Jr., Houston, Texas.

For further information please write to the Publications Department, The University of Texas, M. D. Anderson Hospital and Tumor Institute, at Houston Texas Medical Center, Houston, Texas 77025.

## SYMPOSIUM ON CLINICAL ASPECTS OF ACUTE LEUKEMIA AND BURKITT'S TUMOR

This Symposium, sponsored by the American Cancer Society and the National Cancer Institute, is intended to present the latest clinical information as well as the most current research findings on acute leukemia and Burkitt's tumor.

The Symposium will be held Wednesday, September 20, 1967, at the Museum of Science, Boston, Massachusetts.

For further information, please write: Dr. Jack W. Milder, Research Department, American Cancer Society, Inc., 219 East 42 Street, New York, New York 10017.

## SIXTH ROCHESTER (N.Y.) SYMPOSIUM ON CINERADIOLOGY

The Sixth Rochester Symposium on Cineradiology will be held Thursday evening, Friday, and Saturday, April 25–27,

1968, in Rochester, New York, under the auspices of the Department of Radiology, University of Rochester Medical Center. As before, the meeting should provide an opportunity for registrants to meet informally with neophytes and experts in this field.

Owing to the restricted seating capacity of the Medical School Auditorium, registration will be limited to approximately 200 individuals.

The program will be oriented toward the practical application of cineradiology in research, clinical service and teaching demonstrations as well as the latest technical developments in cineradiology and allied fields. The Thursday evening presentation will consist of the consideration of Selected Basic Technical Aspects of Cineradiology. Further discussion of technical material will be carried out in smaller seminar type groups. Hopefully this present announcement will stimulate the submission of papers from laboratories or clinics engaged in cineradiologic research, clinical service and technical development that will stress the primary usefulness of cineradiology.

The Program Committee is eager to receive applications to present papers from any individual working in cineradiology along the lines stated above. In order to be considered for inclusion, applications must be received prior to December 1, 1967.

Please direct all inquiries and applications to Raymond Gramiak, M.D., Division of Diagnostic Radiology, University of Rochester Medical Center, Rochester, New York 14620.

# BOOK REVIEWS

*Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.*

RADIOLOGY IN WORLD WAR II. Prepared and published under the direction of Lieutenant General Leonard D. Heaton, The Surgeon General, United States Army; Editor in Chief, Colonel Arnold Lorentz Ahnfeldt, MC, USA; Editor for Radiology, Kenneth D. A. Allen, M.D. Cloth. Pp. 1088, with many illustrations. Price, $8.25. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402, 1963.

For a nation and any group within that nation, it is difficult to imagine a stimulus more extreme than participation in a World War. Indeed, history views the success of the response as victory or defeat.

The Volume under consideration is a splendid account of the response of Radiology to full participation in World War II. Written by those who magnificently aided in the creation of that history, it recalls in great detail the complexities of the conversion of a medical specialty from peacetime activity to the provision of diagnostic and therapeutic service under the most stressful of conditions. Acutely feeling the lack of any recorded precedent from World War I, it becomes obvious that the authors were determined to record their experiences for future generations of radiologists in terms of the practical necessities of creating an efficient organization highly dependent on elaborate equipment and well-trained personnel capable of providing adequate radiologic services during battle. Although the changes in warfare and the state of readiness of the Armed Forces will pose new problems in the event of remobilization and obviate the necessity of solving many others, this meticulous evaluation of World War II, viewed from the perspective of 20 years provides a most valuable historical precedent. Indeed, the response in many ways would of necessity be different; however, the technique of creating that response in many ways would be similar.

This monumental volume of *Radiology in World War II* is one of a series of volumes on the official history of the Medical Department of the United States Army in World War II, prepared by the Historical Unit, United States Army Medical Service, and published under the direction of the Surgeon General, United States Army. Its distinguished Editor is Dr. Kenneth D. Allen, who under most difficult circumstances assumed this heavy responsibility after the death of the original Editor, Dr. Byrl R. Kirklin, when the work was scarcely begun. Thirty-one authors, all former medical officers, who saw service in World War II, contributed the various chapters of this volume, describing their experiences and giving radiologic information of inestimable value. To quote from the foreword of Surgeon General Heaton, "Many reviewers and other readers have expressed their approval of the United States Army Medical Department histories for just this reason. There is, naturally, no possible objection to documentation as such, but blind reliance upon it, as Bernard De Voto, himself a Pulitzer Prize winner in History, once put it, can indeed destroy the spirit that alone giveth life. We have, I think, avoided this fallacy in these historical volumes."

This history of *Radiology in World War II* has significance to us engaged in the practice of Radiology above its military interest, for the second World War is in large measure responsible for the current form of both our specialty and the society in which that specialty is practiced.

Dr. Stafford L. Warren in the final chapter of the Volume describes the participation of Radiology in the development of the atomic bomb. Faced with a nearly total void of knowledge, it was their responsibility to provide the information necessary to protect the health of those engaged in the preparation of the fissionable material used in these weapons. The solutions of these problems provided the groundwork for much of radiation biology and the necessary information for the incorporation of radioactive materials into biologic research and clinical medicine.

The use of this energy bequeathed to us by World War II should be of the utmost and urgent interest to all of us.

FREDERICK R. CUSHING, M.D.

X-Rays and Radium in the Treatment of Diseases of the Skin. By Anthony C. Cipollaro, M.D., Emeritus Clinical Professor of Medicine (Dermatology), Cornell University Medical College; Consulting Physician, New York Hospital; Past President, American Dermatological Association and American Academy of Dermatology; Former Member of the Radiation Protection Committee, U. S Bureau of Standards; Former Member of the Council on Medical Physics, American Medical Association; and Paul M. Crossland, M.D., Clinical Professor of Dermatology, Stanford University School of Medicine, Palo Alto, Calif. Fifth edition, completely rewritten. Cloth. Pp. 830, with 365 illustrations. Price, $28.50. Lea & Febiger, Philadelphia, Pa. 19106, 1967.

This completely rewritten version of the classic text on radiation therapy of the skin, primarily intended for dermatologists, will have great value for radiologists as well. For one thing, it demonstrates what an astonishing amount of radiology two dermatologists can learn if they put their minds to it. For another, it illuminates some of the intricacies and subtleties of dermatologic medicine brightly enough at least to show that this field is no place for the amateur.

The authors note the decreasing use of x rays and radium for the treatment of skin disease, but state that "Nonetheless, radiation is still one of the most therapeutic agents in dermatology." They believe that every dermatologist should be equipped with the knowledge to give radiation therapy, and clearly the standards they set for such knowledge are high.

The first half of the book is devoted to the basic sciences of radiology. There is an exceptionally rich history section. The physics treatment is comprehensive, and appropriate to present practice in dermatology; it eschews such recent innovations as the rad and the solid dosimeter. The classical dicta of radiobiology are meticulously listed.

Although the basic sections of the book are excellent for their intended readers, the radiologist, who is familiar with this material from other sources, will find his excitement in the clinical chapters. The treatment of skin cancer occupies about 20 per cent of the clinical text space. The approach displays unusually wide perspective, and if the student finds himself confused by the great variety of techniques mentioned, his reward is to know that any single pattern he may learn is but one of a multitude which have proven successful in experienced hands. It is in the remaining sections on nonmalignant disease that most radiologists will find themselves wallowing in the depths of an esoteric and profound specialty concerned with the most superficial organ. The therapeutic regimens proposed are empirical and sometimes seem to border on the occult, although the radiation prescriptions are conservative to a degree that should satisfy almost everyone except perhaps the geneticists. The reviewer is inclined to recommend that this book be on the shelf of every radiologist, if only to remind him not to attempt to practice amateur dermatology.

A. Raventos, M.D.

## BOOKS RECEIVED

Radiology as an Art and Other Essays. By John L. McClenahan, M.D., Visiting Radiologist, Hospital of the University of Pennsylvania, Philadelphia, Pa. Cloth. Pp. 89. Price, $5.75. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill. 62703, 1967.

Radiological Atlas of Bone Tumours. Volume I. The Netherlands Committee on Bone Tumours. Cloth. Pp. 267, with many illustrations. Price, $35.00. The Williams & Wilkins Company, Baltimore 2, Md., 1966.

Les Carcinomes Bronchiques Primitifs. By Maurice Bariéty, Professeur de Clinque Médicale à l'Hôtel-Dieu de Paris, Membre de l'Académie de Médecine; Jacques Delarue, Professeur d'Anatomie Pathologique à la Faculté de Médecine de Paris; Membre de l'Académie de Médecine; Jean Paillas, Professeur Agrégé d'Anatomie Pathologique à la Faculté de Médecine de Paris; and Roger Rullière, Médecin des Hôpitaux de Paris. Cloth. Pp. 642, with 221 figures. Price, 180 F. Masson et Cie Éditeurs, 120 Boulevard Saint-Germain, Paris, 1967.

Radiations Ionisantes, Radioprotecteurs et Syndrome Gastro-intestinal. By Dr. J.-R. Maisin, Chef du Département de Radiobiologie du Centre d'Étude de l'Énergie Nucléaire de Mol, Belgique. Paper. Pp. 194, with 68 figures. Price, 35 F. Masson et Cie, Éditeurs, 120 Boulevard Saint-Germain, Paris, 1966.

A Biological Consideration of Treatment of Breast Cancer. By George Crile, Jr., M.D., F.A.C.S., Head, Department of General Surgery, Cleveland Clinic, Cleveland, Ohio. Cloth, Pp. 189, with some illustrations. Price, $8.00. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill. 62703, 1967.

Daily Growth in Length of Diaphysis Measured by Oxytetracycline in Rabbit Normally and After Medullary Plugging. By Lars Ingvar Hansson, Department of Histology and the Department of Orthopaedics, University of Lund, Sweden. Paper. Pp. 199, with some illustrations. Acta Orthopaedica Scandinavica, Supplementum No. 101. Munksgaard, Copehagen, 1967.

Surgery of the Paralysed and Flail Shoulder. By A. K. Saha. Paper. Pp. 90, with some illustrations. Acta Orthopaedica Scandinavica, Supplementum No. 97. Munksgaard, Copenhagen, 1967.

Spondylosis Deformans in the Dog: A Morphologic Study with Some Clinical and Experimental Observations. By Joe P. Morgan, Department of Clinical Radiology, Royal Veterinary College, Stockholm, Sweden. Pp. 88, with some illustrations. Acta Orthopaedica Scandinavica, Supplementum No. 96. Munksgaard, Copenhagen, 1967.

Microradiographic Investigation of Bone Grafts in Man. By Gabriele Stringa and Giuseppe Mignani, Florence, Italy. Paper. Pp. 78, with some illustrations. Acta Orthopaedica Scandinavica, Supplementum No. 99. Munksgaard, Copenhagen, 1967.

Tensile Properties of the Human Lumbar Annulus Fibrosus. By Jorge O. Galante, Department of Orthopaedic Surgery, University of Gothenburg, Sweden. Cloth. Pp. 91, with some illustrations. Acta Orthopaedica Scandinavica, Supplementum No. 100. Munksgaard, Copenhagen, 1967.

Experimental Skeletal Tuberculosis in the Guineapig: A Method for Producing Local Lesions and an Autoradiographic Study of Their Accessibility to Tritium-labelled Dihydrostreptomycin. By Lars Lindberg, Department of Orthopaedic Surgery, Malmö General Hospital, University of Lund, Malmö, Sweden. Paper. Pp. 80, with some illustrations. Acta Orthopaedica Scandinavica, Supplementum No. 98. Munksgaard, Copenhagen, 1967.

Studies on Muscle Metabolism of Glycogen and Active Phosphate in Man with Special Reference to Exercise and Diet. By Eric Hultman, Clinical Central Laboratory, Sct Eriks Sjukhus, Stockholm, Sweden. Paper. Pp. 63, with some illustrations. The Scandinavian Journal of Clinical & Laboratory Investigation. Volume 19, Supplementum 94. Universitetsforlaget, Oslo, 1967.

Hyaline Membrane Disease: A Radiological Investigation in Rabbits. By Klas Rosengren, Roentgen Department II, Sahlgrenska Sjukhuset, Gothenburg, Sweden. Paper. Pp. 64, with some illustrations. Price, Sw.Kr.35:-. Acta Radiologica, Supplementum 262, Stockholm, 1967.

Angiography in Tumours of the Urinary Bladder. By Jan Nilsson, Roentgen Diagnostic Department, University Hospital, Lund, Sweden. Paper. Pp. 130, with some illustrations. Price, Sw.Kr.35:-. Acta Radiologica, Supplementum 263, Stockholm, 1967.

Postmortal Changes of the Lung: A Roentgenographic, Microscopic and Bacteriological Follow-up Study on a Pediatric Series and on Animals with Experimental Pneumonia. By Per-Erik Heikel, Children's Hospital, University of Helsinki, Helsinki, Finland. Paper. Pp. 104, with some illustrations. Price, Sw.Kr.30:-. Acta Radiologica Supplementum 264, Stockholm, 1967.

Le Esperienze Radiografiche nella Evoluzione dello Studio Anatomico del Sistema Linfatico dell'Uomo. Relazione al XXIII Convegno della Società Italiana di Anatomia, Siena, October 14–17, 1963. By L. Oliva and C. Stuart. Paper. Pp. 393, with some illustrations. Valsalva Editrice, Firenze, 1964.

La Protezione dei Pazienti Sottoposti a Indagini o Terapie Radiologiche. Edited by Luigi Oliva and Carlo Polvani. Paper. Pp. 231, with some tables. Edizioni Minerva Medica, Torino, 1966.

Technology and Manpower in the Health Service Industry, 1965–75. Manpower Research Bulletin No. 14. Paper. Pp. 109. Single copies available free from: U. S. Department of Labor, Manpower Administration, Washington, D. C. 20210, 1967.

Ureteral Reflux in Children. A Workshop sponsored by the Committee on the Genitourinary System, Division of Medical Sciences, National Academy of Sciences-National Research Council, November 11–12, 1966. Edited by James F. Glenn, M.D. Paper. Pp. 213. Price, $2.50. The Printing and Publishing Office, National Academy of Sciences-National Research Council, 2101 Constitution Avenue, N.W., Washington, D. C. 20418, 1967.

# SOCIETY PROCEEDINGS

## MEETINGS OF RADIOLOGICAL SOCIETIES*

### UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
*Secretary*, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Washington-Hilton Hotel, Washington, D. C., Sept 26–29, 1967.

AMERICAN RADIUM SOCIETY
*Secretary*, Dr. Fernando G. Bloedorn, Division of Radiotherapy, University of Maryland Hosp., Baltimore, Md. 21201. Annual meeting: Hotel Fontainebleau, Miami Beach, Fla., April 7–11, 1968.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
*Secretary-Treasurer*, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., Nov. 26–Dec. 1, 1967.

AMERICAN COLLEGE OF RADIOLOGY
*Executive Director*, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Drake Hotel, Chicago, Ill., Feb. 6–10, 1968.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
*Secretary*, Dr. Kenneth L. Krabbenhoft, Harper Hospital, Detroit, Mich. 48201. Annual meeting: San Francisco, Calif., June 16–20, 1968.

AMERICAN BOARD OF RADIOLOGY
*Secretary*, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn.

The Fall 1967 examination will be held at the Statler Hilton Hotel, Dallas, Texas, December 4–8, inclusive. The deadline for filing applications was June 30, 1967.

The Spring 1968 oral examination will be held at the Fontainebleau Hotel, Miami Beach, Florida, June 10–14, inclusive. The deadline for filing applications is December 31, 1967. Candidates eligible for this examination will not be required to take the written examination.

The first written examination will be held the latter half of June 1968 in various centers of the country for all residents having completed 3 years of approved training as of June 30, 1968. Deadline for filing for this examination and the oral examination of December 1968 is December 31, 1967.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
*Secretary*, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
*Secretary*, Dr. J. A. del Regato, Penrose Cancer Hospital, Colorado Springs, Colo. 80907.

AMERICAN SOCIETY FOR DIAGNOSTIC ULTRASOUND
*Secretary*, Dr. Charles C. Grossman, 552 N. Neville St., Pittsburgh, Pa. 15213.

TWELFTH INTERNATIONAL CONGRESS OF RADIOLOGY
*President*, Dr. Kempo Tsukamoto, 9-1, 4-chome, Angewa, Chiba, Japan. Meeting: Hotel New Otane, Tokyo, Japan, Oct. 6–11, 1969.

NINTH INTER-AMERICAN CONGRESS OF RADIOLOGY
*Counselor for the United States*, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.
*President*, Dr. Leandro Zubiaurre, Montevideo, Uruguay. Meeting: Hotel San Rafael, Punta del Este, Uruguay, Dec. 6–12, 1967.

INTER-AMERICAN COLLEGE OF RADIOLOGY
*President*, Dr. Oscar Soto, H. Urteaga 480, Lima, Perú.

ALABAMA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Walter Brower, Birmingham, Ala. Meets time and place of Alabama State Medical Association.

ARIZONA RADIOLOGICAL SOCIETY
*Secretary-Treasurer*. Dr. George Gentner, 3435 W. Durango, Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meetingsix months later.

ARKANSAS CHAPTER OF AMERICAN COLLEGE OF RADIOLOGY
*Secretary-Treasurer*, Dr. William J. Rhinehart, St. Vincent Infirmary, Little Rock, Ark. 72205.

ARKANSAS RADIOLOGICAL SOCIETY
*Secretary*, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
*Secretary-Treasurer*, Dr. Harry Z. Mellins, S.U.N.Y. College of Medicine, Brooklyn, New York 11201. Annual Meeting: Ohio State University School of Medicine, Columbus, Ohio, May 9–11, 1968.

ATLANTA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Donald R. Rooney, Burnt Hickory Road, Marietta, Ga. Meets monthly except during three summer months, on third Tuesday, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
*Secretary*, Colonel Kurt Harrell, Landstuhl Army Medical Center, Landstuhl, Germany. Meets quarterly.

BLOCKLEY RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. R. John Gould, 141 Lombardy Rd., Drexel Hill, Pa. 19026.

BLUEGRASS RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Joe Bernard, Central Baptist Hosp., Lexington, Kentucky 40503. Meets quarterly.

BROOKLYN RADIOLOGICAL SOCIETY
*Secretary*, Dr. Skottowe DePass, Brooklyn, N. Y. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
*Secretary*, Dr. Richard Sheehan, 36 Briarlee Drive, Tonawanda, N. Y. Meets second Monday evening each month, October to May inclusive.

CALIFORNIA RADIOLOGICAL SOCIETY
*Secretary*, Dr. James J. McCort, Santa Clara Valley Med. Ctr., San Jose, Calif. Meets annually during meeting of California Medical Association.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Emmett R. White, P. O. Box 303, Rutherford College, N. C. 28671. Meets every Friday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:00 NOON.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Robert A. Bornhurst, State Univ. Hospital, 750 E. Adams St., Syracuse, N. Y. 13210. Meets first Monday each month, October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
*Secretary*, Dr. Ollie E. Southard, 2787 Tudor Rd., Columbus, O. 43209. Meets second Thursday in October, November, January, and March 15 and May 19 at Fort Hayes Hotel, Columbus, Ohio.

CHICAGO ROENTGEN SOCIETY
*Secretary-Treasurer*, Dr. Fredric D. Lake, 2548 N. Lakeview Ave., Chicago, Ill. 60614. Meets second Thursday of each month, October to April, except December, at the Pick-Congress Hotel at 8:00 P.M.

CLEVELAND RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Theodore J. Castele, 18869 Canyon Rd. Parkview Park, Ohio 44126. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

COLORADO RADIOLOGICAL SOCIETY
*Secretary*, Dr. George F. Wertz, 1801 High St. Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
*Secretary*, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, George E. Plum, 712 N. Washington Ave., Dallas, Tex. 75246. Meets monthly, third Monday, at Southwest International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
*Secretary*, Dr. Robert L. Willis, Harper Hospital, Detroit, Mich. 48201. Meets monthly, first Thursday October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

EAST BAY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Tom H. Piatt, 12 Camino Encinas, Orinda, Calif. 94563. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. T. F. Haase, Jr., 205 Medical Arts Building, Knoxville, Tenn. Meets in January and September.

FLORIDA RADIOLOGICAL SOCIETY
*Secretary*, Dr. John C. Jowett, Orlando, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Garth R. Drewry, Tampa General Hospital, Tampa 6, Fla. Meets in January, April, July and October.

GEORGIA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Al Rayle, Jr., 938 Peachtree St., N. E., Atlanta, Ga. Meets in spring and fall with Annual State Society Meeting.

GREATER CINCINNATI RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

GREATER LOUISVILLE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

GREATER MIAMI RADIOLOGICAL SOCIETY
*Secretary Treasurer*, Dr. Thomas W. Tufts, Broward General Hospital, 1600 S. Andrews Ave., Ft. Lauderdale, Fla. Meets monthly, third Wednesday at 8:00 P.M., at Jackson Memorial Hospital, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
*Secretary-Treasurer*, Dr. Alexander J. Link, 7215 Maryland, St. Louis, Mo. 63130.

HAWAII RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. R. K. Wang, P.O. Box 256, Wahiawa, Oahu 96786. Meets third Monday of each month at 7:30 P.M.

HOUSTON RADIOLOGICAL SOCIETY
*Secretary*, Dr. Zoltan Petrany, 1200 Moursund Drive, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at the Doctors' Club, 8:00 P.M., Houston, Tex.

IDAHO STATE RADIOLOGICAL SOCIETY
*Secretary*, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY
*Secretary*, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC.
*Secretary*, Dr. Richard A. Silver, 1815 N. Capitol Avenue, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

IOWA RADIOLOGICAL SOCIETY
*Secretary*, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Robert C. Lawson, 310 Medical Arts Bldg., 10th and Horne, Topeka, Kan. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER, AMERICAN COLLEGE OF RADIOLOGY
*Secretary-Treasurer*, Dr. Ralph C. Quillin, 1221 S. Broadway, Lexington, Ky. 40504. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
*Secretary*, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
*Secretary*, Dr. Samuel Gelband. Meets second Tuesday of the month in February, April, June, October and December.

LOS ANGELES RADIOLOGICAL SOCIETY
*Secretary*, Dr. Harvey I. Meyers, 2010 Wilshire Blvd., Los Angeles, Calif. 90057. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Texas 77701.

MAINE RADIOLOGICAL SOCIETY
*Secretary*, Dr. J. T. Chen, 7 Cherry Hill Terrace, Waterville, Me. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY
*Secretary*, Dr. Henry Startzman, Medical Arts Building, Baltimore, Md.

MEMPHIS ROENTGEN SOCIETY
*Secretary-Treasurer*, Dr. Vernon I. Smith, Jr., Suite 203, 1085 Madison Ave., Memphis, Tenn. 38104. Meets first Monday of each month at John Gaston Hospital.

MIAMI VALLEY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Indiana. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MID-HUDSON RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
*Secretary-Treasurer*, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Edward A. Peterson, 572 Lowry Medical Arts Bldg., St. Paul, Minn. Meets twice annually, fall and winter.

MISSISSIPPI RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Dan T. Keel, Jr., 504 Chippewa St., Brookhaven, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

MISSOURI RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. M. Shoss, Cape Girardeau, Mo.

MONTANA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Clark Grimm, Great Falls, Montana. Meets at least once a year.

NEBRASKA STATE RADIOLOGICAL SOCIETY
*Secretary*, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEVADA RADIOLOGICAL SOCIETY
*Secretary*, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.

NEW ENGLAND ROENTGEN RAY SOCIETY
*Secretary*, Dr. Morris Simon, 330 Brookline Ave., Boston, Mass. 02153. Meets third Friday of each month, October through May, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
*Secretary*, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New Mexico Association of Radiologists
*Secretary-Treasurer*, Dr. Justin J. Wolfson, Department of Radiology, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

New Mexico Society of Radiologists
*Secretary*, Dr. Phil Fox, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
*Secretary*, Dr. Arnold L. Bachman, Francis Delafield Hosp., 99 Fort Washington Ave., New York, N. Y. 10032. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M. Annual meeting to be announced.

North Carolina Chapter of ACR.
*Secretary-Treasurer*, Dr. Ira Bell, Hickory, N. C. Annual meeting to be announced.

North Carolina Radiological Society
*Secretary*, Dr. E. H. Schultz, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in the spring and fall each year.

North Dakota Radiological Society
*Secretary*, Dr. A. Ohrt, 408 Medical Arts Bldg., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
*Secretary*, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
*Secretary*, Dr. Anthony J. Tabacco, 621 Central Ave., Albany 6, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
*Secretary-Treasurer*, Ivan D. Siddons, 3701 J. St., Suite 106, Sacramento, Calif. 95816. Meets fourth Monday of Sept., Nov., Jan., March and May at the Sutter Club in Sacramento.

Northwestern Ohio Radiological Society
*Secretary*, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society
*Secretary*, Dr. Mortimer Lubert, Mt. Sinai Hospital, Cleveland, Ohio. Annual meeting to be announced.

Oklahoma State Radiological Society
*Secretary*, Dr. Robert Sukman, 1200 N. Walker, Oklahoma City, Okla. Meets in January, May and October.

Orange County Radiological Society
*Secretary*, Dr. George W. Logan, 301 Newport Blvd., Newport Beach, Calif. Meets fourth Tuesday of every month at Orange County Medical Association Building.

Oregon Radiological Society
*Secretary-Treasurer*, Dr. Irving J. Horowitz, 2311 N.W. Northrup Str., Portland, Ore. 97210. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
*Secretary*, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
*Secretary-Treasurer*, Dr. Willis Taylor, 1118 9th Ave., Seattle, Washington. Annual meeting to be announced.

Pennsylvania Radiological Society
*Secretary*, Dr. T. Frederick Weiland, 619 Ridgeway Ave., Grove City, Pa. 16127. Annual meeting to be announced.

Philadelphia Roentgen Ray Society
*Secretary*, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
*Secretary*, Dr. Edward R. Seitz, 601 Jenkins Bldg., Pittsburgh, Pa. 15222. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiological Society of Connecticut, Inc.
*Secretary-Treasurer*, Dr. Orlando F. Gabriele, 1450 Chapel St., New Haven 11, Conn. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
*Secretary*, Dr. Harold N. Margolin, 6159 Tulane Road, Cincinnati, Ohio. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
*Secretary*, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
*Secretary*, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
*Secretary*, Dr. Lester W. Eavenson, 2700 Napoleon Ave., New Orleans 15, La. Meets semiannually, during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey
*Secretary*, Dr. John W. Marquis, 12 Hawthorne Ave., East Orange, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island
*Secretary-Treasurer*, Dr. John M. Vesey, 1196 Elmwood Ave., Cranston, R. I.

Radiological Society of South Dakota
*Secretary-Treasurer*, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
*Secretary-Treasurer*, Dr. Gerald M. McDonnel, U.C.L.A. Medical Center for Health Sciences, Los Angeles, Calif. 90024. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
*Secretary-Treasurer*, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y.

Redwood Empire Radiological Society
*Secretary*, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
*Secretary*, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
*Secretary*, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
*Secretary-Treasurer*, Dr. Robert W. Lackey, 4200 E. Ninth Ave., Denver, Colo. Annual meeting to be announced.

San Antonio-Military Radiological Society
*Secretary*, Dr. Hugho F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday of each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
*President-Secretary*, Charles R. Henkelmann, 3909 Palm Drive, Bonita, Calif. 92002. Meets first Wednesday of each month at the Town & Country Motel.

San Francisco Radiological Society
*Secretary*, Dr. H. Joachim Burhenne, Children's Hospital and Adult Medical Center, 3700 California St., San Francisco, Calif. 94119. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco, Calif. 94118.

Santa Clara County Radiological Society
*Secretary*, Dr. Bill G. Karras. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

Section on Radiology, California Medical Association
*Secretary*, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
*Secretary-Treasurer*, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
*Secretary*, Dr. Andrew F. Giesen, Jr., White-Wilson

Clinic, Fort Walton Beach, Fla. 32548. Annual meeting: Miami Beach, Fla., Nov. 13–16, 1967.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
*Secretary*, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
*Secretary*, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
*Secretary*, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Washington Hilton Hotel, Wash., D.C., Sept. 25, 1967.

SOCIETY OF NUCLEAR MEDICINE
*Secretary*, Mr. C. Craig Harris, Oak Ridge National Laboratories, Oak Ridge, Tenn. *Administrator*, Mr. Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting to be announced.

SOUTH BAY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

SOUTH CAROLINA RADIOLOGICAL SOCIETY
*Secretary*, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

SOUTH DAKOTA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Donald J. Peik, 1417 S. Minnesota Ave., Sioux Falls, S. Dak. Meets in spring with State Medical Society and in fall.

SOUTHERN RADIOLOGICAL CONFERENCE
*Secretary-Treasurer*, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. 36604. Annual meeting: Grand Hotel, Point Clear, Ala. 36504, Jan. 26–28, 1968.

SOUTHWESTERN RADIOLOGICAL SOCIETY
*Secretary*, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

TENNESSEE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Marion E. Spurgeon, Memorial Hosp., Clarksville, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

TEXAS RADIOLOGICAL SOCIETY
*Secretary*, Dr. Herman C. Sehested, 815 Medical Arts Bldg., Fort Worth 2, Tex. Annual meeting to be announced.

TRI-STATE RADIOLOGICAL SOCIETY
*Secretary*, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

UPPER PENINSULA RADIOLOGICAL SOCIETY
*Secretary*, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

UTAH STATE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Jack J. Davis, Salt Lake Clinic, 333 South Ninth East, Salt Lake City, Utah 84102. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

VERMONT RADIOLOGICAL SOCIETY
*Secretary*, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

VIRGINIA RADIOLOGICAL SOCIETY
*Secretary*, Dr. John M. Ratliff, Mary Immaculate Hospital, Newport News, Va.

WASHINGTON STATE RADIOLOGICAL SOCIETY
*Secretary*, Dr. Owen Marten, 930 Terry Avenue, Seattle, Wash. Meets quarterly.

WEST VIRGINIA RADIOLOGICAL SOCIETY
*Secretary*, Dr. George G. Green, Morgantown, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

WESTCHESTER COUNTY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Arnold Gerson, Medical Arts Bldg., Mt. Vernon, N. Y. Meets on third Tuesday of January and October and on two other dates.

WISCONSIN RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Harold F. Ibach, 2400 W. Villard Ave., Milwaukee, Wis. 53209. Meets twice a year, May and September.

WYOMING RADIOLOGICAL SOCIETY
*Secretary*, Dr. J. D. Grant, Memorial Hosp., Sheridan, Wyo. Meets in fall with State Medical Society and in spring on call of President.

## CUBA, MEXICO, PUERTO RICO AND CENTRAL AMERICA

ASOCIACIÓN DE RADIÓLOGOS DE CENTRO AMERICA Y PANAMÁ. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
*Secretary-General*, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

SOCIEDAD DE RADIOLOGÍA DE EL SALVADOR
*Secretary*, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador.

SOCIEDAD DE RADIOLOGÍA DE GUATEMALA
*Secretary*, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPÍA CUBANA
*Secretary*, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

SOCIEDAD COSTARRICENSE DE RADIOLOGIA
*Secretary*, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

SOCIEDAD MEXICANA DE RADIOLOGÍA, A.C.
Coahuila No. 35, México 7, D. F.
*Secretary-General*, Dr. Armando L. Rodríguez
Meets first Monday of each month.

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA
*Secretary*, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

SOCIEDAD RADIOLÓGICA PANAMEÑA
*Secretary*, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

SOCIEDAD RADIOLÓGICA DE PUERTO RICO
*Secretary*, Dr. José T. Medina, Apt. 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

## BRITISH COMMONWEALTH OF NATIONS

ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC
*Secretary*, Dr. R. Robillard, Notre-Dame Hospital, 1560 Sherbrooke St., East, Montreal, Que., Canada. Meets four times a year.

BRITISH INSTITUTE OF RADIOLOGY
*Honorary Secretary*, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May. Annual meeting to be announced.

CANADIAN ASSOCIATION OF PHYSICISTS, DIVISION OF MEDICAL AND BIOLOGICAL PHYSICS.
*Honorary Secretary-Treasurer*, Paul M. Pfalzner, Dept. of Therapeutic Radiology, University of Western Ontario, London, Ont., Canada. Annual meeting to be announced.

EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY
*Secretary*, Dr. B. V. Evans, 105 Northgate Bldg., Alberta, Canada. Meets second Tuesday of each month in various Edmonton Hospitals.

FACULTY OF RADIOLOGISTS
*Honorary Secretary*, Dr. J. N. Pattinson, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting to be announced.

FACULTY OF RADIOLOGISTS, ROYAL COLLEGE OF SURGEONS IN IRELAND
*Registrar*, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
*Honorary Secretary-Treasurer*, Dr. Maurice Dufresne, *Associate Honorary Secretary-Treasurer*, Dr. F. Robert MacDonald, 1555 Summerhill Ave., Montreal 25, Que., Canada. Thirty-first Annual Meeting, Chateau Frontenac, Quebec, Que., March 4–9, 1968.

Montreal Radiological Study Club
*Secretary*, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
*Secretary*, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie
*Secretary General*, Dr. Jacques Lespérance, 5415 Boul. L'Assomption, Montreal 26, P. Q., Canada. Meets every third Tuesday from October to April. Annual meeting: Oct. 12–14, 1967.

Toronto Radiological Society
*Secretary*, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

College of Radiologists of Australasia
*Honorary Secretary*, Dr. T. P. Loneragan, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

## South America

Asociación Argentina de Radiología
*Secretary*, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
*Secretary*, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
*Secretary-General*, Dr. Miguel Mario Céntola, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiologia
*Secretary-General*, Dr. Jorge Enrique Barragué, Azeuénaga 888, Buenos Aires. Meetings are held monthly.

Sociedad Boliviana de Radiología
*Secretary*, Dr. Héctor Fernández Ferrufino, Casilla 1192, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
*Secretary*, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
*Secretary*, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
*Secretary*, Dr. Patricio Barriga, Casilla 13426, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
*Secretary-General*, Dr. Armando Uribe, Hospital Militar Central, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
*Secretary*, Dr. Luis Blum, P.O. Box 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
*Secretary*, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
*Secretary-General*, Dr. Augusto Cáceres G. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 318, Lima.

Sociedad de Radiologica del Atlantico
*Secretary*, Dr. Raul Fernandez, Calle 40 #41–110, Barranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología Cancerología y Física Médica del Uruguay
*Secretary-General*, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedad de Radiología de El Salvador
*Secretary*, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedade de Radiología de Pernambuco
*Secretary*, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
*Secretary-General*, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad Venezolana de Radiología
*Secretary-General*, Dr. Luis F. Muro, Apartado No. 9362 Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

## Continental Europe

Österreichische Röntgen-Gesellschaft
*President*, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. Annual meeting to be announced.

Société Belge de Radiologie
*General Secretary*, Prof. Simon Masy, Louvain, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
*Permanent Secretary*, Dr. Jaques Sauvegrain, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France.
*General Secretary*, Dr. H. Ludin, Department of Roentgenology, Basler Kinderspital, Basel, Switzerland. Annual meeting to be announced.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
*Secretary-General*, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
*Secretary*, Dr. Robert Poch, Praha 12, Srobárova 50, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
*Secretary*, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut, Lagenbeckstr. 1, Mainz, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
*Secretary*, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
*Secretary*, Dr. H. F. O. Stricker, Schalklaar, Netherlands.

Scandinavian Radiological Society
*Secretary-General*, Dr. C-E. Unnérus, Hagalund-Tapiola, Havsvindsvägen 5 C., Finland. Annual meeting: Copenhagen, Denmark, 1968.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
*Secretary*, Juan Gomez Lopez, Villanueva, 11, Madrid 1. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid. Annual general meeting to be announced.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
*Secretary*, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

## Asia

Indian Radiological Association
*Secretary*, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

Indonesian Radiological Society
*Secretary*, Professor Sjahriar Rasad, Taman Tjut Mutiah I Diakarta, Indonesia

Iranian Radiological Society
*Secretary*, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

# ABSTRACTS OF RADIOLOGICAL LITERATURE

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## ROENTGEN DIAGNOSIS

### BLOOD AND LYMPH SYSTEM

GUILFOIL, PAUL H., and CHRISTIANSEN, THORVALD. An unusual vascular complication of fractured clavicle. *J.A.M.A.*, April 3, 1967, *200*, 72–73. (Address: Paul H. Guilfoil, M.D., 4100 W. Third Street, Dayton, Ohio 45428.)

This is the report of a case of a 55 year old man who was admitted to hospital in an alcoholic stupor complaining of pain in his left hand. It was cool, the fingernails were cyanotic and no pulse could be palpated in the left upper extremity. Aortography showed complete occlusion of the left subclavian artery.

Operation 16 hours after admission revealed an ununited fracture of the left clavicle with a tight fibrous band between the fragments, compressing the subclavian artery. The artery was found to contain an organized clot and also a large amount of soft red clot. These were removed and after suturing, the radial and axillary pulses returned and were still present 8 weeks later. Histologic examination of the clot showed evidence of an old, partially organized mural thrombus with revascularization and other areas of fresh thrombosis.

It is thought that the vascular injury was related to the old fracture of the clavicle. It is also thought that the eventual complete occlusion resulted from the stuporous patient having positioned his left upper extremity so as to cause injury to the artery by pressure of the mobile ends of the bone. The authors have not been able to find any similar case in the literature.—*Arthur E. Childe, M.D.*

SERATT, ERNEST M., DORAN, MORTON L., and BAIRD, RONALD J. False aneurysms in the lower extremity. *Surg., Gynec. & Obst.*, March, 1967, *124*, 562–566. (From: Department of Surgery, University of Toronto, Toronto Western Hospital, and the Toronto General Hospital, Toronto, Ontario, Canada.)

Thirty-three patients with false aneurysms in the lower extremity were treated at the Toronto Western and the Toronto General Hospitals between 1956 and 1966. The commonest site of false aneurysm was the groin and 2 major causes were identified.

(1) In 7 patients, trauma produced the aneurysm. In 5 of these, the aneurysm developed at the site of arterial needle puncture or femoral arterial catheterization.

(2) In 26 patients, the aneurysm developed following primary arterial surgery and 21 of these followed femoro-popliteal by-pass grafting procedures. Teflon, nylon and autogenous vein grafts have all been involved.

The usual symptoms consisted of aching or throbbing discomfort, the development of a pulsatile mass, and a systolic bruit at the site of aneurysmal formation.

In the absence of sepsis at the site of the false aneurysm, a second reconstructive surgical procedure usually effected a cure. In 6 of 7 patients with sepsis at the aneurysmal sites, repeated subsequent surgical procedures failed and resulted in amputation of a portion of the involved extremity because of ischemia and necrosis. In the 7th patient with an infected aneurysm, a radical procedure with by-pass of the infected site resulted in eventual saving of the lower extremity.—*John H. Harris, M.D.*

LEVIN, HUGH S., MCDERMOTT, PAUL H., RUNCO, VINCENT, CARNAZZO, ANTHONY J., and BOOTH, RICHARD W. Petechiae following angiography: evidence for increased capillary permeability. *Radiology*, Feb., 1967, *88*, 253–257. (From: Department of Medicine, Division of Cardiology, Creighton University School of Medicine, Omaha, Neb.)

The use of modern radiodiagnostic contrast media has resulted in a decrease in the fatal complications following angiography, although a number of adverse reactions of varying severity still occur. The development of petechiae following angiography has been reported only once previously (LANGE, E. K., *J. Urol.*, 1963, *90*, 603), although the occurrence of hemorrhagic phenomena in patients receiving other iodine containing compounds is well known. During a 12 month period the authors have observed this postangiography complication in 3 patients.

The case reports of the 3 patients who developed extensive petechiae after angiography are presented in detail. In each instance there was no history of iodine or contrast medium allergy. Two of the patients received sodium acetrizoate as the contrast medium and 1 received sodium iothalamate. In Case I, the petechiae appeared about 4 hours after injection, progressively involved the entire body, began to fade in 2 days, and disappeared within 4 days. The extensive hematologic evaluation was normal. In Case II, the diffuse petechiae appeared about 24 hours after angiography and cleared within 2 days. A hematologic evaluation in this instance was not obtained. In Case III, the petechiae appeared about 19 hours after transfemoral catheter abdominal aortography and dramatically involved only the lower trunk and lower extremities, reflecting the body area receiving the high concentration of contrast medium. In this patient, a biopsy of a petechial area was performed 8 hours after the appearance and revealed bland diapedesis of red cells without vasculitis or capillary thrombosis. Again, the extensive hematologic evaluation for clotting defects was normal and all petechiae had disappeared in 10 days.

Previous investigations of petechiae due to inorganic iodides have shown the cause to be nonthrom-

bocytopenic vasculitis or thrombocytopenia secondary to the destruction of circulating platelets, presumably idiosyncratic. Careful studies of the effects of iodinated organic contrast media indicate that many of their adverse effects can be attributed to capillary endothelial damage. This effect is largely related to hyperosmolarity which results in red cell crenation, clumping and agglutination, and increased viscosity of blood flow with subsequent ischemia of the endothelium. This damage interferes with the integrity of the endothelium and leads to increased permeability of the vessel wall. In addition, the contrast medium may have a direct and independent chemotoxic action.

The clinical and laboratory observations in these 3 cases do not indicate a clotting defect or an allergic vasculitis as a cause of the petechiae. Instead, the authors theorize that, because of normal variability in regional blood flow, some vessels were exposed to the hyperosmotic and chemotoxic effects of the medium while shunting protected other capillaries. As a result of the local endothelial damage there was increased capillary permeability, allowing the transudation of blood and the formation of petechiae.—*Edward B. Best, M.D.*

DÜX, A., BÜCHELER, E., and THURN, P. Die indirekte Splenoportographie: Methodik, Indikationen und Ergebnisse. (Indirect splenoportography: methods, indications and results.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1967, *106*, 183–197. (Address: Priv. Doz. Dr. A. Düx, Institut für Röntgenologie und Strahlenheilkunde der Universität Bonn, 53 Bonn—Venusberg, Germany.)

Two methods are available for studying the portal circulation: the direct splenoportography with injection of contrast material into the splenic pulp and the indirect method through high abdominal aortography or selective angiography of the celiac axis, superior mesenteric artery and splenic artery. The indirect method is only possible with an adequate dose of contrast material if there is no delay of its passage. The splenoportal vessels must be patent and the blood flow must be in the direction toward the liver.

Opacification of the portal circulation with the indirect method normally occurs from 6 to 9 seconds following the rapid injection of contrast material. The results are good with hypervascularization of the spleen but rather poor when the spleen is hypovascularized.

The indirect method is indicated in all mass lesions of the left upper quadrant requiring arteriography, such as tumors of the pancreas, the stomach and splenomegaly, also in atypical vascular murmurs, arteriovenous fistulas, and portal hypertension. Further indications are portal hypertension following splenectomy, recurrence of bleeding varices, splenic vein occlusion, small sized spleen, and atypical location of the spleen. It is also indicated in rapid increase of a small cirrhotic liver for excluding a primary malignant tumor.

The results were best when the tip of the catheter was lodged in the splenic artery and under normal conditions, but the portal vein was only faintly visualized when the circulation was delayed. The density of the contrast depended not as much on the size of the spleen as on the speed of blood flow. Selective gastric arteriography was inadequate for demonstrating the portal vein. Selective injection into the superior mesenteric artery, however, was satisfactory in normal cases, but when circulation was slow, visualization of the portal vein was delayed as much as 20 seconds. In cirrhosis of the liver, the splenic vein could not be visualized because of portal hypertension with reversed blood flow.

Comparison of the two methods revealed that the indirect method was good in demonstrating various tumors in the epigastrium but insufficient in depicting the vasculature of a large tumorous liver. The direct splenoportography is distinctly superior in ruling out metastatic seeding of the liver. When the spleen is too small for this method, isotope scanning rather than arteriography can demonstrate the metastatic lesions. Selective angiography is the method of choice in splenomegaly of unknown etiology, in questionable splenic cyst, hematoma of the spleen, systemic lesions, and in malignant tumors of the spleen. This indirect method is also superior in arteriovenous fistulas and arterioportal fistulas.—*Ernest Kraft, M.D.*

## GENERAL

GERSHON-COHEN, J., HABERMAN-BRUESCHKE, J. D., and BRUESCHKE, E. E. Medical thermography. *J. de radiol., d'électrol. et de méd. nucléaire*, Jan.–Feb., 1967, *48*, 12–24. (Address: J. Gershon-Cohen, M.D., 255 S. 17th Street, Philadelphia, Pa. 19103.)

The increasing value of thermography is outlined in this paper. The authors first review the biophysics of thermography and then briefly compare the available instruments and methods of examination. Examples of the current application of thermography are given for each of the clinical specialties.

In 64 patients with carotid occlusive lesions the over-all accuracy of diagnosis using thermography was found by Wood to be 89 per cent. The value of thermography in localizing early metastases and early breast carcinoma is demonstrated. In obstetrics and gynecology it is possible to localize the placenta thermographically with great accuracy. In orthopedics it is possible to demonstrate herniated disks, fractures, sprains and contusions. Thermography also has diagnostic value in peripheral vascular disease, dermatology, and urology.

The conclusion reached is that thermography has made significant advances in its early stage of development and holds great promise for the future.—*Harry G. Barr, M.D.*

## RADIATION THERAPY

Williams, I. G. "Let there be light"—the treatment of advanced retinoblastoma by external irradiation. *Proc. Roy. Soc. Med.*, Feb., 1967, *60*, 189–196. (From: Radiotherapy Department, St. Bartholomew's Hospital, London, England.)

In this, the President's Address, the history of radiotherapy in the treatment of retinoblastoma is traced from 1929 until the present day. It was originally practiced at St. Bartholomew's Hospital by Moore, and currently by Stalland and Williams. Their present policy is to treat small lesions with $Co^{60}$ disks applied directly to the sclera, reserving external irradiation for those patients with bilateral tumors in whom an attempt is made to preserve useful vision in the less affected eye. The patients reported had advanced disease in both eyes or, having had one eye removed, the growth in the remaining eye was too large for the application of radioactive disks.

In arriving at the rationale for their technique consideration is given to these 5 factors: (1) natural history of the disease; (2) tolerance of normal tissue; (3) radiosensitivity of the tumor; (4) dose necessary to destroy it; and (5) advantages of concomitant chemotherapy.

With these considerations in mind treatment is given utilizing $Co^{60}$ teletherapy through a single anterior field centered directly over the cornea. A dose of 900 or 1,050 r is delivered weekly in 3 sessions to a total dose of 3,500 r (3,000–4,000 r) in 28 days. In addition oral cyclophosphamide is administered in maintenance doses for 1–2 years.

Forty-six patients have been treated in this manner from 1962 through 1965; 31 have been followed for 2–4 years. Thirty-five of the 46 are alive and well with vision, 2 have died of metastases, 2 are blind with neoplasm destroyed, 5 have had enucleation and 2 have been lost to follow-up.

Twelve patients have developed cataract which has been accepted as an almost certain complication and the author anticipates the development of many more. He divides these into 2 categories: (1) a stippled opacity 1–1.5 mm. in diameter which does not progress; and (2) those that progress over 1 to 3 years and require a discission operation. This operation is not especially difficult, the only disadvantage to a child being lack of accommodation and the remote possibility of retinal detachment in 10 to 20 years. Even this latter hazard is deemed unlikely after irradiation since the retina and choroid may adhere in some places.—*Howard West, M.D.*

Elkington, S. G., and McKissock, Wylie. Pituitary adenoma: results of combined surgical and radiotherapeutic treatment of 260 patients. *Brit. M. J.*, Feb. 4, 1967, *1*, 263–266. (Address: S. G. Elkington, M.D., Senior Medical Registrar, St. Thomas's Hospital, London, England.)

Several surgical and radiotherapeutic techniques have been employed in the treatment of pituitary adenoma varying from the intracranial route of Horseley and the endonasal route of Cushing as surgical approaches, to primary treatment of the tumors and postoperative irradiation for reduction of recurrences in radiotherapeutic efforts. The authors' series comprises 260 patients over the period from 1938 to 1962 inclusive, who were treated by the same technique surgically (transfrontal route) followed, with few exceptions, by radiotherapy. Immediate (or within 6 weeks) postoperative deaths were approximately 10 per cent, with a mortality of 33 per cent in patients with large or extensive tumors and just under 5 per cent in patients with small tumors.

Of 63 deaths in the series occurring later than 6 weeks, only 6 were attributed to radiation damage to the cerebral hemispheres and 1 due to recurrence of the tumor.

During the early years of the series, 10 patients were given radiotherapy in doses now recognized as carrying a risk of radiation damage to nervous tissue. Nine of the patients died between 10 months to 7 years after completion of the course of therapy with radiation damage demonstrated in 3 at autopsy; probable damage on clinical grounds was present in 2 more.

Tumor recurrence was observed in 7.5 per cent of patients, most occurring within 5 years of the operation. The results in 7 patients who underwent a second operation for tumor recurrence were disappointing.

Vision was improved by treatment in 58 per cent of the survivors examined and unchanged in 26 per cent.—*James R. Knapp, M.D.*

Einhorn, Jerzy, and Wikholm, Gunnel. Hypothyroidism after external irradiation to the thyroid region. *Radiology*, Feb., 1967, *88*, 326–328. (From: Radiumhemmet, Stockholm, Sweden.)

Reports of a pronounced tendency toward development of hypothyroidism long after $I^{131}$ therapy for hyperthyroidism prompted an examination of the thyroid function many years after heavy external irradiation of the normal thyroid gland.

There had been reports in the literature of cases of hypothyroidism appearing after irradiating tumors of the neck, but they were incomplete as to frequency of occurrence or to length of follow-up. Thus the present study consists of patients who had received

irradiation to their neck lesions at least 10 years previously.

Sixty-three patients who received external irradiation at Radiumhemmet for laryngeal and hypopharyngeal carcinoma between 1921 and 1951 were living and not more than 75 years of age in 1961. Of the 63, 41 were available for study with a mean period of follow-up after treatment of 18 years. None of these had any known disorders of the thyroid prior to irradiation. An additional 20 patients with tumors in regions other than the neck served as controls.

The thyroid function was assessed by: (1) clinical examination; (2) radioiodine uptakes with thyrotropin stimulation; (3) protein bound iodine determination; and (4) basal metabolic rate measurement. The controls were also subjected to these tests.

The results showed that the response to thyrotropin was weaker in the patients who had received radiotherapy for laryngeal and hypopharyngeal carcinomas than in the control group. Four of the 41 patients in the former group exhibited clinical signs of hypothyroidism, and the diagnosis of this condition was supported by the above laboratory tests. One case of hypothyroidism was quite severe, the other 3 cases were mild. (These 4 patients were placed on thyroid substitution and have been followed for 3 years; 3 of them improved.) In 28 of the 41 patients, there was laboratory evidence of thyroid dysfunction but no clinical evidence of hypothyroidism. In none of the 41 patients was any enlargement or nodularity of the thyroid gland observed, nor were there any signs of thyroid carcinoma.

The authors point out that the thyroid gland is fairly radioresistant, but for only a limited follow-up period, and that a decade after heavy external irradiation there is reduction of the thyroid reserve and resultant mild hypothyroidism is probably not uncommon. Histologic examination of the thyroid gland post irradiation discloses extensive vascular damage. Impairment of the reproductive ability of the thyroid cells (in rat studies) and histologic evidence of defective cell division (in cases treated with $I^{131}$) have been observed. Low-titer thyroid antibodies were noted in 19 of 20 cases treated in which hypothyroidism developed after $I^{131}$ treatment of hyperthyroidism and in 5 euthyroid patients who developed hypothyroidism after external irradiation to their necks.

This information is insufficient in the authors' opinions to decide if the development of hypothyroidism many years after irradiation of the thyroid gland is due to vascular damage, impairment of regeneration, or immunologic reactions.—*P. M. Kroening, M.D.*

DELARUE, NORMAN C., PETERS, VERA, ANDERSON, W. S., and STARR, J. A re-evaluation of the place of major extirpation in the management of patients with metastatic mammary carcinoma. *Canad. M. A. J.*, March 18, 1967, *96*, 637–651. (From: Department of Surgery, Toronto General Hospital, and the Ontario Cancer Institute incorporating the Princess Margaret Hospital, Toronto, Ontario, Canada.)

In an effort to assess the value of the two major extirpative procedures in metastatic breast carcinoma (adrenalectomy and hypophysectomy) the authors have analyzed a series of 222 cases—133 adrenalectomies and 89 hypophysectomies.

Since only about 40 per cent of patients are benefitted by such a procedure the results were studied first to determine what criteria are valid in predicting a response to such surgery, and secondly to decide what point in the course of the disease it can most profitably be employed. Numerous clinical and histopathologic variables were evaluated as were the endocrine and menstrual status of the patients. Their response to previous addititive hormonal therapy as well as castration were also studied.

Among the several conclusions which resulted from this detailed analysis were these 6:

(1) This type of surgery may be beneficial in all but menopausal women having the disease for less than 2 years.

(2) Extirpation should be used soon after the appearance of metastases rather than at the end of a program of sequential therapy.

(3) It is of particular value when there is asymptomatic involvement of vital organs such as lung or liver.

(4) Oophorectomy is an effective screening procedure in the premenopausal patient. Women who do not respond to oophorectomy are not likely to benefit from either of these procedures.

(5) Early use of this procedure does not preclude subsequent response to other modalities of treatment.

(6) There was no significant differences in the remission rate between the 2 procedures but adrenalectomy is the more practical operation.—*Howard West, M.D.*

ANDERSON, W. A. D., and GUNN, SAMUEL A. The vaginal irrigation smear: reliability in detection of premalignant and malignant conditions of the uterine cervix in asymptomatic and symptomatic patients. *J.A.M.A.*, April 10, 1967, *200*, 166–167. (Address: W. A. D. Anderson, M.D., 1700 NW 10th Avenue, Miami, Fla. 33136.)

The method of patient-obtained cytologic specimen (the vaginal irrigation smear) for detection of cancer of the cervix was compared with the office-obtained cytologic smear.

The population used was from the outpatient

clinics of Jackson Memorial Hospital. A total of 4,494 patients were included in the survey. The patient-obtained specimen proved to be correct in 85 per cent of the time for carcinoma *in situ*, and 100 per cent of the time for all cases of invasive cervical cancer. In severe dysplasia of the cervix, the irrigation smear detected 79 per cent in symptom-free cases, and 83 per cent in symptomatic cases.

From this study the authors conclude that the vaginal irrigation smear technique appears to be sufficiently reliable even in asymptomatic women to warrant its use when a cervical scrape specimen cannot be obtained for some reason or another.—*David C. Alftine, M.D.*

Tow, W. S. H., and CHENG, W. C. Recent trends in treatment of choriocarcinoma. *Brit. M. J.*, March 4, 1967, *1*, 521–523. (From: Department of Obstetrics and Gynaecology, University of Singapore, Singapore.)

The successful chemotherapy of choriocarcinoma has cast some doubt on the value of hysterectomy in this disease and some have stated that it may impair response to this type of therapy. The authors report their results in the treatment of 80 cases of choriocarcinoma seen between 1959 and 1966, 64 of which occurred in conjunction with hydatidiform mole.

Surgical procedures consisted of total abdominal hysterectomy in 59, excision of vaginal nodules in 9 and resection of solitary lung lesions in 5. Hysterectomy was considered palliative in 4 and was the primary treatment for hydatidiform mole in 17 of the 59.

Methotrexate and mercaptopurine became available in September, 1960. Two standard regimens were used, methotrexate plus mercaptopurine and methotrexate alone. Actinomycin-D was available after January, 1963, and was used in those cases which proved resistant to the other two drugs.

Of the 80 cases 49 were cured, 30 died and 1 was still under treatment. Cure was defined as no evidence of disease for 6 months or longer: 17 were cured by hysterectomy alone and 2 by hysterectomy plus excision of vaginal nodules. Thirty-five were treated with hysterectomy plus chemotherapy; there were 23 cures in this group. Of those treated with chemotherapy alone 7 were cured and 6 died. Twelve received only symptomatic treatment and all expired.

The authors conclude that hysterectomy in those cases with tumors largely or entirely localized to the uterus is still a valuable therapeutic measure.—*Howard West, M.D.*

## RADIOISOTOPES

HISADA, KIN-ICHI, OHBA, SATORU, and MATSUDAIRA, MASAMICHI. Isosensitive radioisotope scanning. *Radiology*, Jan., 1967, *88*, 124–128. (From: Department of Radiology, School of Medicine, Kanazawa University, Kanazawa, Japan.)

The authors describe the development of a medical universal human counter which has several clinical applications. This medium level human counter employs four 3×2 inch NaI (Tl) crystals which may be arranged in certain configurations and combined with scanning table motion to accomplish the desired technique.

For the purpose of whole body counting, the signals from the four symmetrically placed detectors are added and analyzed by a spectrometer and a 6 decade scaler. For linear (profile) scanning the table of the scanning bed may be moved longitudinally while simultaneously recording the spectrometer counting rate. Transverse motion of one to four detectors can be combined with stepwise longitudinal bed motion to accomplish area scanning. The detector signals may be fed to four spectrometers with contrast amplifiers and then to four multidot recording heads for multinuclide area scanning.

The authors consider isosensitive scanning to be one of the most important applications of this device. For this purpose two opposed detectors with a 37 hole collimator of 15 cm. focal length and 2 inch Pb side wall shielding is used. The patient is placed on the scanning table between the detectors. Transverse detector motion is combined with stepwise longitudinal motion to accomplish the conventional scanning mode. If the patient's thickness is 20 cm., the best spatial conditions for obtaining a uniform isoresponse curve independent of depth in the body require 5 cm. of clearance for each detector.

With isosensitive radioisotope scanning, a record is obtained which is independent of depth in contrast to conventional scanning which is heavily weighted to the superficial distribution. It is, therefore, suitable for: (a) detecting deepseated abnormalities in such large organs as the liver, lung, and brain; (b) multinuclide scanning; and (c) quantitative estimation of the regional pulmonary blood flow by the lung scan. Also, the use of two detectors reduces the amount of radioisotope required for the scan. It is the authors' opinion that isosensitive scanning should be performed first to survey the entire depth and, when it is necessary to accentuate the lesion observed, laminoscanning should follow.—*L. H. Deiterman, Jr., Ph.D.*

HISADA, KIN-ICHI, HIRAKI, TATSUNOSUKE, OHBA, SATORU, and MATSUDAIRA, MASAMICHI. Simultaneous performance of isosensitive scanning and bilaminoscanning. *Radiology*, Jan., 1967, *88*, 129–134. (From: Department of Radiology, School of Medicine, Kanazawa University, Kanazawa, Japan.)

The authors describe in more detail their isosensitive radioisotope scanning technique which can pro-

vide depth independent scan information. They recommend that isosensitive scanning should be performed first to survey the entire depth and then laminoscanning in order to further resolve the abnormality. Since this procedure may be time consuming, a new technique was developed to simultaneously obtain an isosensitive scan and 2 laminoscans.

This technique employs a total of four 3×2 inch NaI (Tl) scintillation detectors. Two are opposed and are used to provide the isosensitive scan. The other two are placed obliquely above and below the patient so that their response curve intersects the isosensitive response curve of the first pair at a depth of 6 cm. All the collimators are of 37 hole honeycomb design and the focal lengths in air are 15 cm. for the isosensitive pair and 25 cm. for the laminoscanning pair. The signals from the detectors are mixed three ways: (a) addition of signals from the two opposed detectors for the isosensitive scan; (b) addition of the upper perpendicular detector and the upper oblique detector for the upper laminoscan; and (c) addition of the lower perpendicular detector and lower oblique detector for the lower laminoscan. By the inclusion of the single output of the upper perpendicular detector, the conventional scan is also obtained. These are recorded simultaneously by using the four mechanical multidot tapper heads which are servo driven. This system can be helpful in disclosing smaller lesions in an organ of thick dimensions.—*L. H. Deiterman, Jr., Ph.D.*

Christie, J. H., Gomez Crespo, G., Koch-Weser, Dieter, and MacIntyre, W. J. The correlation of clearance and distribution of colloidal gold in the liver as an index of hepatic cirrhosis. *Radiology*, Feb., 1967, *88*, 334–341. (From: Highland View Hospital, Cleveland, Ohio.)

The authors have investigated the scan pattern in 46 patients with cirrhosis, in an attempt to correlate the clearance rate of colloidal gold 198 and rose bengal $I^{131}$ with the scan pattern.

The patients were divided into 4 groups on the basis of clinical findings and liver function tests: (1) no significant decrease in hepatic function; (2) minimal decrease; (3) moderate decrease; (4) severe decrease in function. Liver scannings with gold and rose bengal were done with constant scanning speed, count rate parameters, and cut-off setting. The tracer dose was calculated on the basis of relative clearance rates for each patient, so that the dose would provide a maximum rate of 5,000 counts/minute.

It was concluded, on evaluation of gold and rose bengal scans at 25, 60, and 75 per cent cut-off levels, that the most useful criteria for determination of liver impairment were related to the configuration of the organ itself. These changes were: (1) fullness in the hilar region; (2) decrease in deposition of isotope in the liver periphery; and (3) increased accumulation in the center of the liver. The frequency of all 3 findings increased with increase in liver function impairment, with hilar fullness being the most consistent finding in all the groups.

Splenic visualization was also very frequent in patients with severe impairment of liver function.

The increase in perihilar isotope accumulation and decrease in peripheral hepatic concentration is felt to be due to diminished peripheral blood flow in cirrhosis, with increased A–V shunting.—*J. A. Cunyus, M.D.*

Parker, Jacob J., and Siemsen, Jan K. Liver regeneration following hepatectomy, evaluated by scintillation scanning. *Radiology*, Feb., 1967, *88*, 342–344. (From: Department of Radiology, University of California School of Medicine, Los Angeles, Calif.)

Three cases of liver regeneration following partial resection are reported. Two patients had 80 per cent right hepatic lobectomy necessitated by liver laceration secondary to blunt trauma. Liver scans revealed normal-sized functioning mass after 2 and 2½ months, and in 1 of these patients the regenerated liver regained a normal shape. Liver function tests returned to normal in several weeks after surgery in both cases. In a third patient, a left hepatic lobectomy was performed along with a gastrectomy for gastric carcinoma with direct invasion into the adjacent liver. Eight years later, a liver scan revealed regeneration of this left lobe.

The authors conclude that serial scanning is the method of choice for evaluation of liver regeneration; that normal liver regenerates rapidly; and that liver function returns to normal before the liver size has reached normal proportions.—*Victor Brasseur, M.D.*

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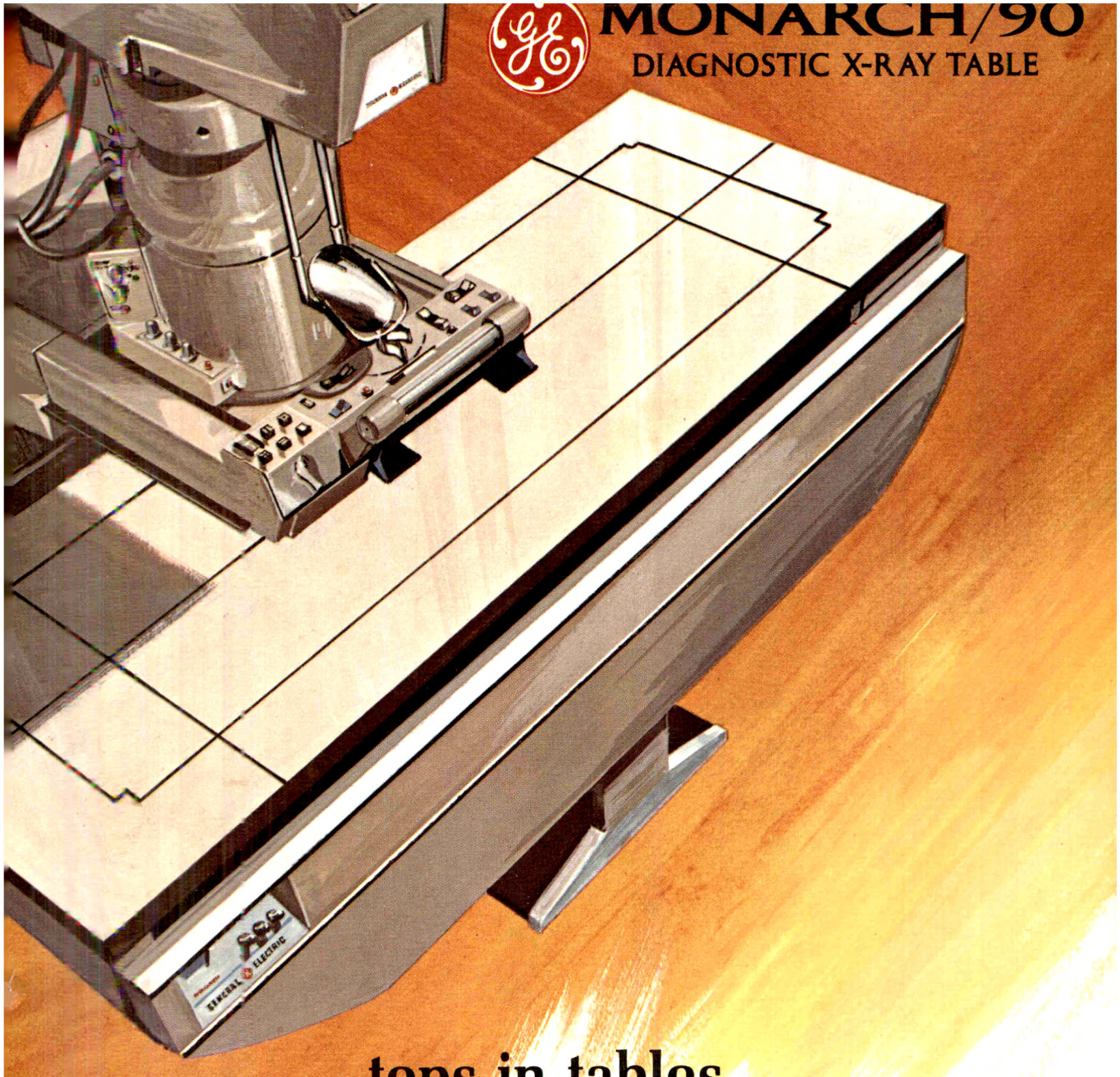
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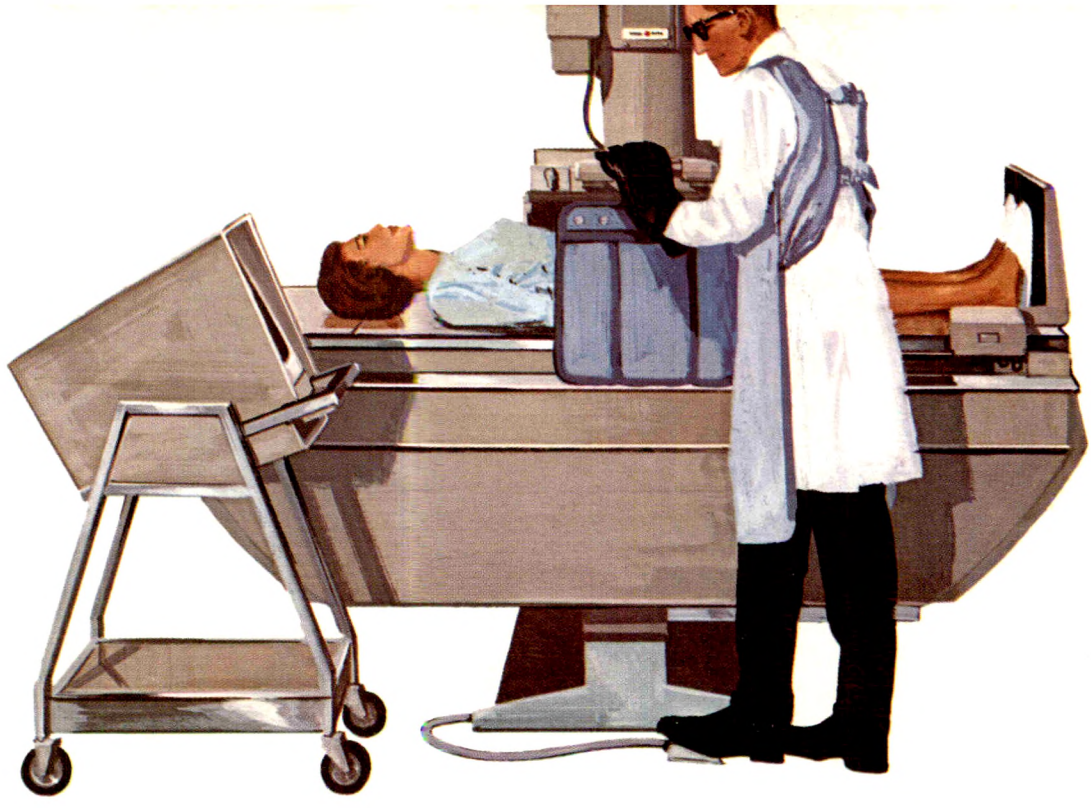
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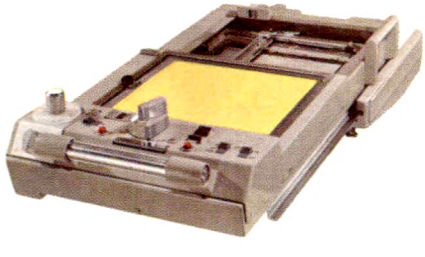
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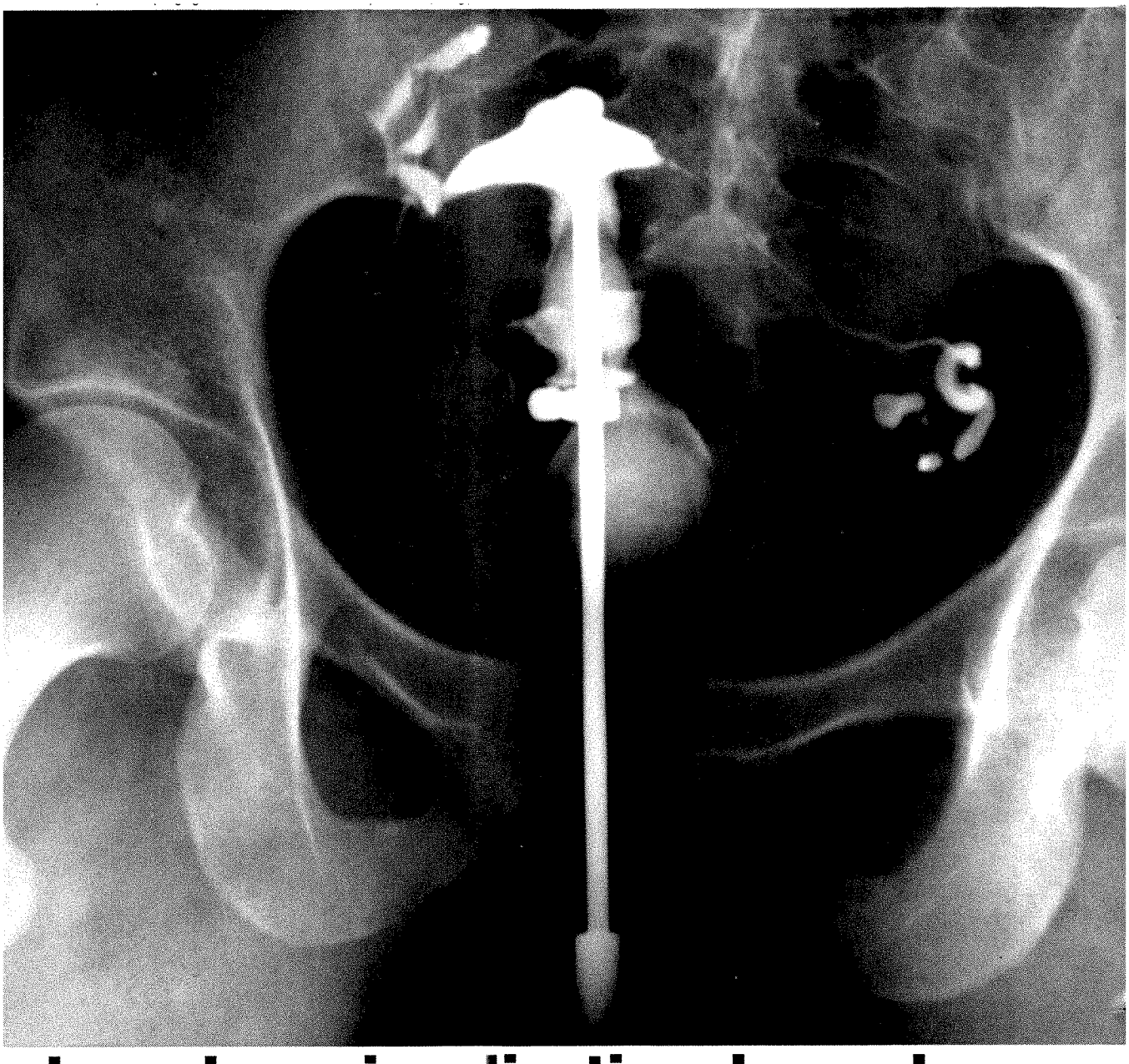
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1. Curr. Med. Pract. 7:145, 1963. 2. Am. J. Roentgenol. 81:696, 1959.

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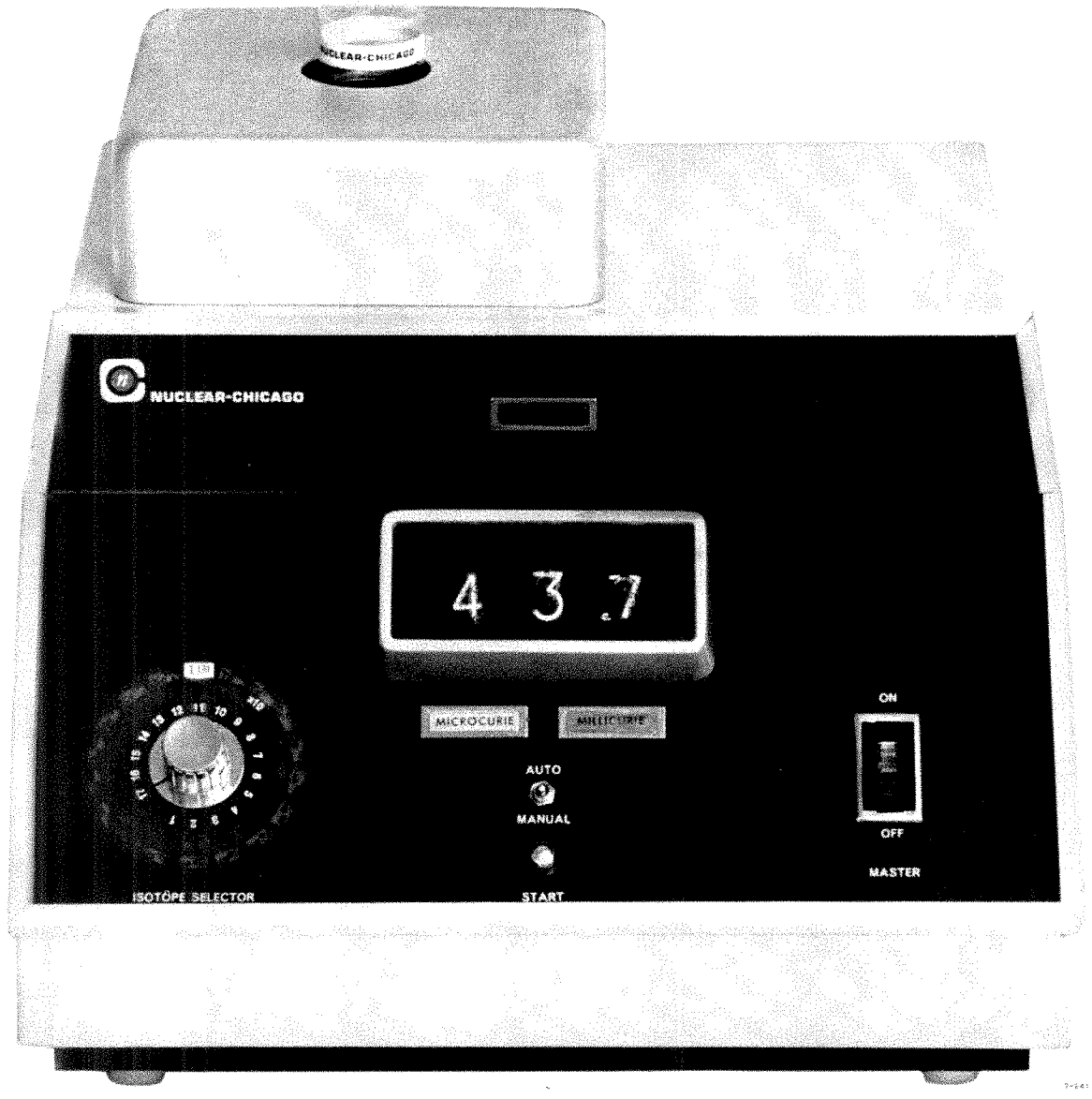
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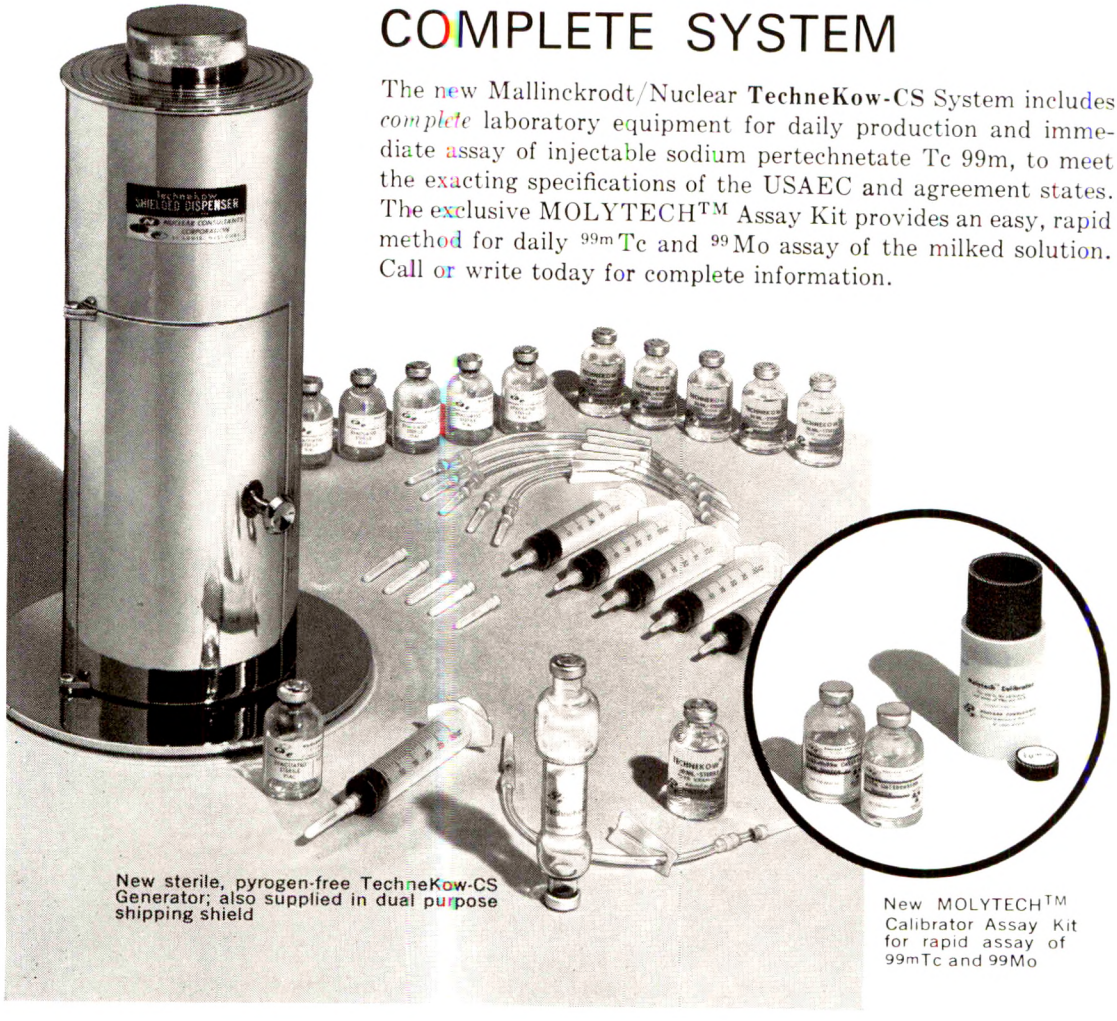
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**Adverse Reactions:** Most reactions are mild and transitory; serious side effects are very rare. Gastrointestinal effects (diarrhea, cramps, nausea, vomiting) are the most common. Usually the diarrhea consists merely of a few loose stools, although in isolated cases it may be severe. A mild stinging sensation during urination may occur, and rarely, skin rash, urticaria, pruritus, and flushing. One case of thrombocytopenia has been reported in a patient with a history of conjunctival hemorrhages. Subjective complaints have been: dryness of throat, burning on swallowing, heartburn, sore throat, dizziness, and headache.

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**Supplied:** Tablets of 500 mg., envelopes of 6 tablets, boxes of 5 and 25 envelopes; also bottles of 500.

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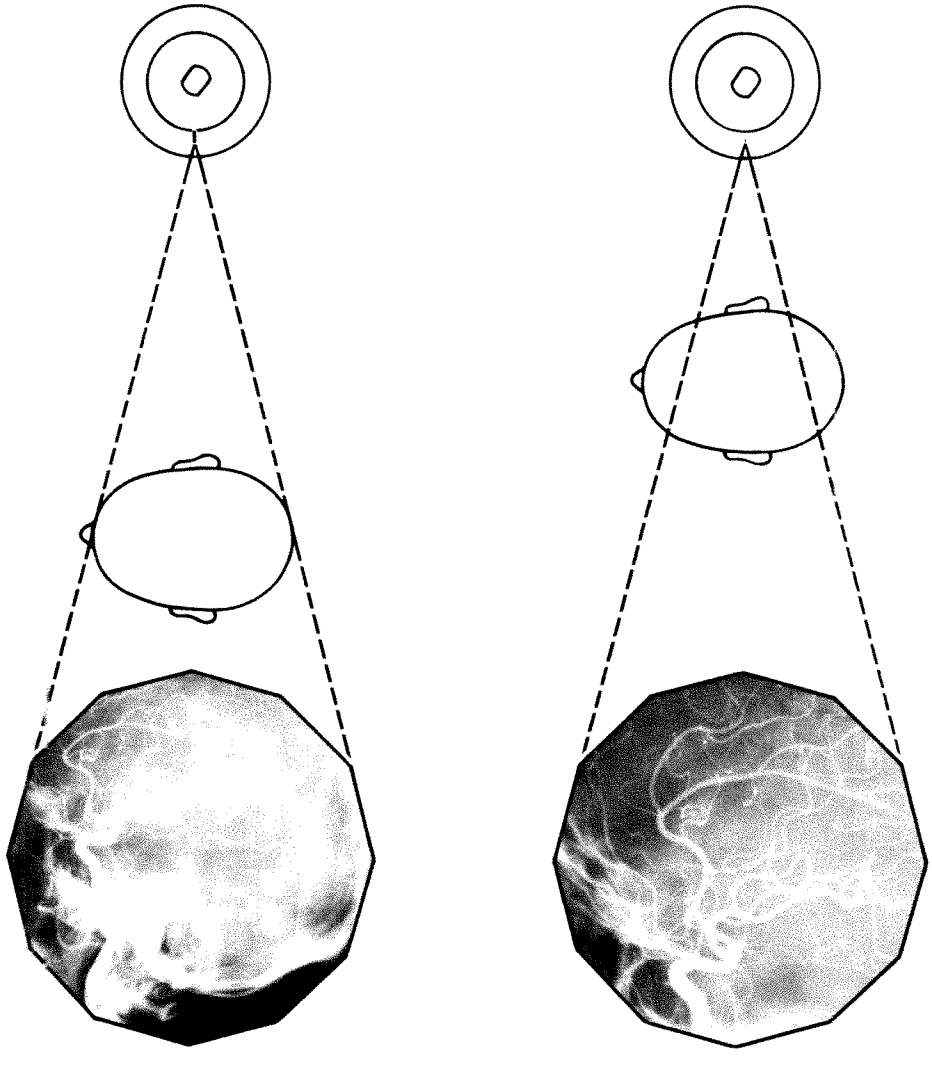
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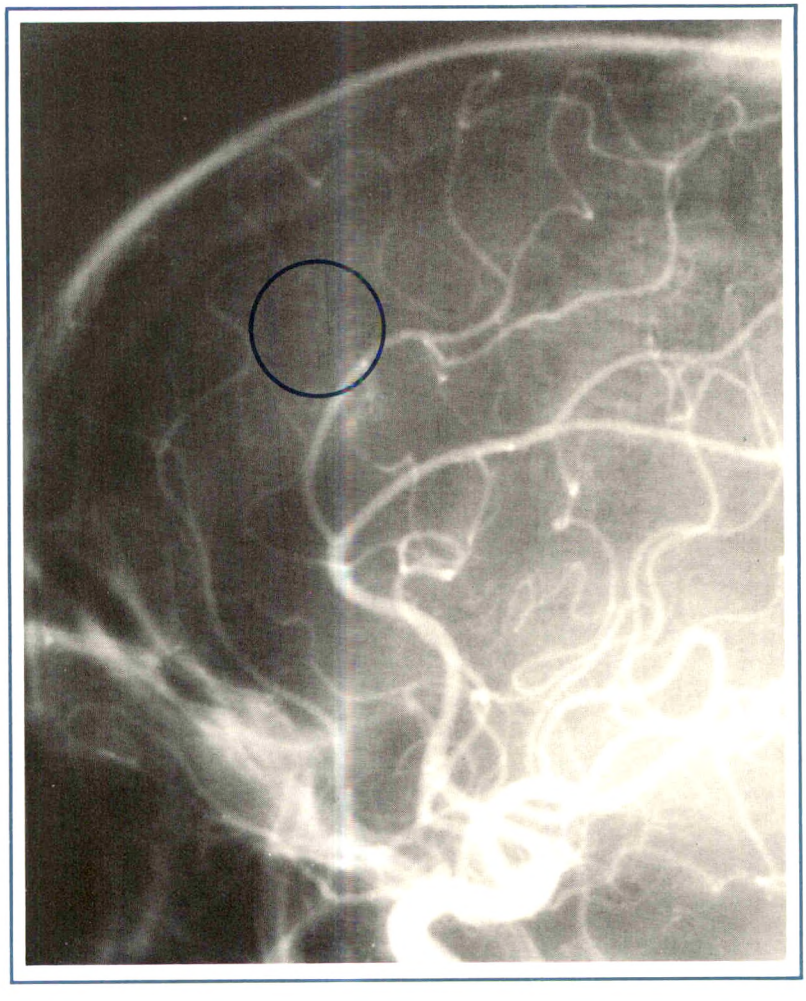
high-power angiography
1.0mm

enlargement radiography
0.3mm



The Dynamax "61" rotating anode X-ray tube is an important development in X-ray tubes for specialized roentgenographic techniques, including enlargement radiography and high-power angiography. It extends, as well, both cineradiographic and spot film capabilities.

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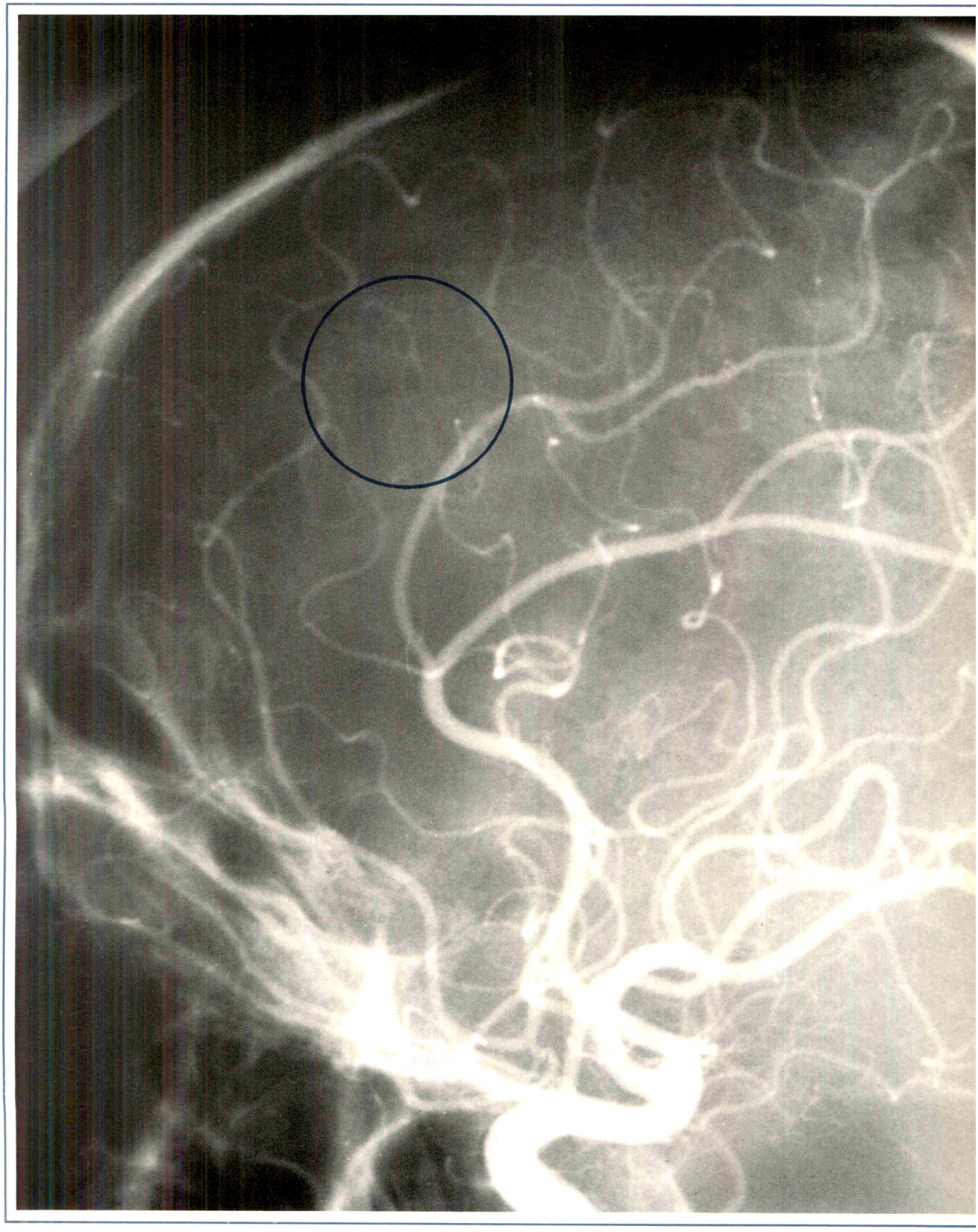


Conventional serial carotid arteriogram, cross-hatch grid. Serial sequence: 2 films per second.

Radiographs courtesy Radiology Department, Albert Einstein Medical Center, Philadelphia, Pennsylvania.

ifference

# Direct Serial Magnification

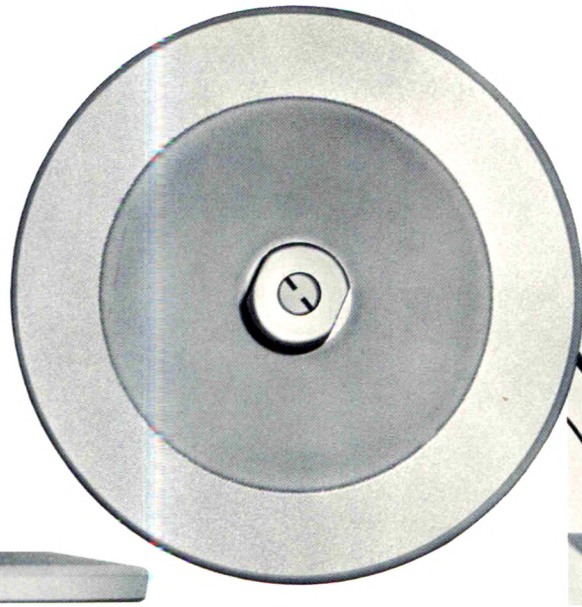


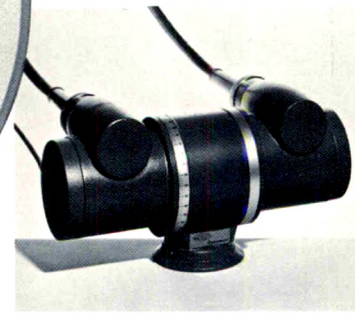
Direct serial magnification carotid arteriogram, "air gap" technique, non-grid (approximate 2X linear, 4X area enlargement). Serial sequence: 2 films per second.

Enhancement of vascular detail by the magnification technique provides additional diagnostic information to the radiologist.

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**The Dynamax "61", a specialized version of the Dynamax "60",** is designed for high-energy techniques in enlargement radiography and angiography. By incorporating an anode with a 10° target angle, the Dynamax "61" provides increased short exposure ratings approximately 60% greater than the Dynamax "60". These higher loadings, hitherto associated with larger focal spots, may be employed on the 0.3mm or 1.0mm focal spot of the Dynamax "61". As an example, with full-wave three-phase rectification, exposures of 1/10 second duration at 100 PKV may be made at 125 MA on the 0.3mm focal spot or 875 MA on the 1.0mm focal spot, with triple-speed anode rotation.

**The Dynamax "61" is available in all options** of high speed, standard speed, and grid-controlled. On grid-controlled models, both focal spots are grid-controlled, but either or both may be operated conventionally.

**Dynamax "61", general characteristics:**

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Maximum Energy Ratings

Maximum Load in MA at 100 PKV,
Full-Wave Rectified, 180 Cycles Stator Power:

| | Single Phase | | | Three Phase | | |
|---|---|---|---|---|---|---|
| Time (seconds) | 0.1 | 1.0 | 10 | 0.1 | 1.0 | 10 |
| 0.3 mm focus | 110 | 93 | 60 | 129 | 97 | 47 |
| 1.0 mm focus | 750 | 430 | 150 | 880 | 450 | 118 |

Thermal Ratings

Anode heat storage capacity: 200,000 HU
Anode cooling rate, maximum: 54,000 HU/min
Housing heat storage capacity: 1,500,000 HU

For details on the Dynamax "61" X-ray tube, write: The Machlett Laboratories, Inc., Springdale (Stamford) Connecticut 06907.

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**SQUIBB**

a research concept in contrast visualization

# for better definition of disease: documenting differences between contrast agents

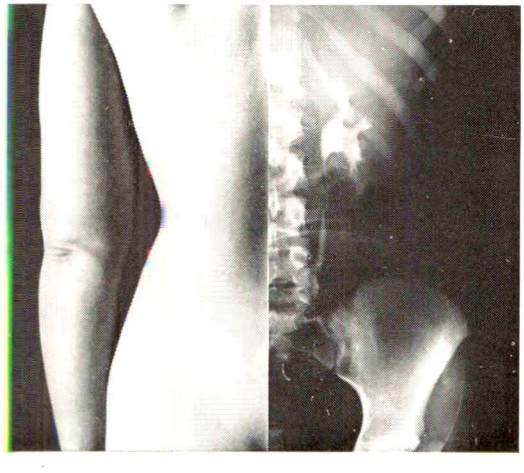
**No. 1 in a series:**

Renografin-60 (meglumine diatrizoate injection) preferred in a study of pyelographies of 2,234 patients.

In a new large scale study,[1] "...to determine which medium would produce adequate visualization of the urinary tract with the fewest toxic effects on the patient," Macht *et al.* have compared Renografin-60 with 2 other contrast agents.

Data were analyzed in 2,234 unselected (consecutive) patients, according to age, sex, and general disease group for the study population as a whole. The first 683 patients received 50% diatrizoate sodium solution, the next 921 patients received Renografin-60, and the final 630 patients received 65.8% sodium iothalamate solution.[1]

**Criteria for quality and comparative safety** The contrast agents were evaluated for quality of diagnostic films as follows: films showing a dense concentration of contrast medium with filling and visualization of all major and minor calyces, infundibula, pelves and almost all of each ureter were listed as "good"; films showing less concentration with incomplete visualization of all portions of urinary tract but sufficient to produce diagnostically adequate films were listed as "fair"; films showing unsatisfactory visualization of urinary tract, or films which could not be interpreted, as "poor." The media were also evaluated as to incidence of the following side effects: nausea, vomiting, fainting, shock or severe reaction, hiccups, hives, pain in arm, sneezing, hot flushes, stuffiness of nose or ears.[1]



The following chart* shows comparative results of the 3 media according to age category:

*quality of pyelograms and side effects expected in standard population of 1,000 patients by age, according to type of medium used*

| medium used and type of patient | standard population | quality of pyelograms good | fair | poor | with side effects |
|---|---|---|---|---|---|
| meglumine diatrizoate | | | | | |
| all ages, | 1,000 | 827 | 123 | 50 | 41 |
| 0-19† | 126 | 122 | 3 | 1 | 3 |
| 20-49 | 415 | 374 | 35 | 6 | 27 |
| 50-69 | 315 | 240 | 51 | 24 | 9 |
| 70 or older | 144 | 91 | 34 | 19 | 2 |
| diatrizoate sodium | | | | | |
| all ages, | 1,000 | 782 | 134 | 84 | 72 |
| 0-19 | 126 | 115 | 6 | 5 | 7 |
| 20-49 | 415 | 378 | 26 | 11 | 40 |
| 50-69 | 315 | 227 | 64 | 24 | 19 |
| 70 or older | 144 | 62 | 38 | 44 | 6 |
| sodium iothalamate | | | | | |
| all ages, | 1,000 | 883 | 81 | 36 | 54 |
| 0-19 | 126 | 122 | 4 | 0 | 7 |
| 20-49 | 415 | 393 | 20 | 2 | 29 |
| 50-69 | 315 | 269 | 32 | 14 | 13 |
| 70 or older | 144 | 99 | 25 | 20 | 5 |

*Adapted from Macht[1] †In some patients in this age group, higher than recommended doses were used.

**In theory:** "The choice of contrast agent should ideally be individualized according to the age, sex, and disease group of the patient in order to obtain a high probability of complete visualization of the urinary tract with low probability of adverse side effects."[1]

**In practice:** "The choice is to be made...on the basis of the agent which gives the best concentration and the fewest side effects in the greatest number of patients regardless of age, sex, or disease category."[1]

**In order of preference:** Although Renografin-60 was not rated highest in all categories, the authors feel that their preference of the contrast media for intravenous pyelography would be: first, Renografin-60; second, sodium iothalamate; third, diatrizoate sodium.[1]

For brief summary of prescribing information, please refer to the end of this advertisement.

# definition of safety...

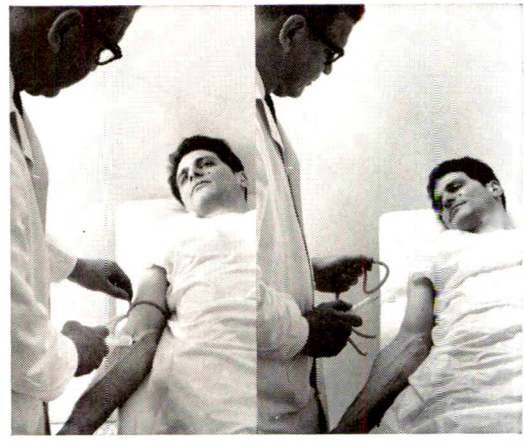
**demonstrated in animals...** Since meglumine diatrizoate is also used for cerebral angiography, toxicity studies of administration via the carotid artery are therefore of interest. Fischer and Eckstein[2] designed angiographic studies in animals in which procedures were very similar to clinical angiography, and which yielded data that was quantitative, graphic and nonsubjective.[2] According to the authors: "We selected the alterations in arterial blood pressure, venous pressure, heart rate and rhythm, the electrocardiogram and end-expiratory $CO_2$ concentration resulting from experimental cerebral angiography as refined, sensitive indications of the toxicity of a particular contrast material."[2] Their results of measured cardiovascular functions in dogs indicated that meglumine diatrizoate was far less toxic than four other contrast media.[2] As Fischer and Cornell reported in a later study: "Despite the testing of more highly concentrated solutions and larger doses, the cardiovascular responses [in dogs] from methylglucamine [meglumine] salts were much less than from sodium salts, an observation consistent with previous experiments."[3]

Other investigators have documented the comparative safety of meglumine salts in experimental studies. In order to determine reaction and tolerance of the intestines, Cooley[4] injected meglumine diatrizoate into mesenteric arteries of dogs and found no damage. Gensini *et al.*[5] reported that cardiovascular responses with it were almost identical to blood transfusions.

**theory of lower toxicity with meglumine...** Gensini and DiGiorgi have offered a hypothesis to explain their findings of lesser toxicity with experimental intravascular injections of methylglucamine (meglumine) salts. "When a relatively undiluted amount of sodium salts of a contrast agent is injected in an artery and carried by the blood stream toward the capillary bed, its molecules rapidly dissociate and readily diffuse through the capillary membrane and into the tissue. There, both the toxic effect of the iodine-containing organic radical and the increased concentration of sodium will readily manifest themselves. At equal concentrations of sodium, the end results will closely reflect the intrinsic toxicity of the iodine-containing organic radical on the tissues...."[5]

"In the case of the methylglucamine compounds, the same dissociation takes place. However the larger methylglucamine molecule, rich in hydrogen bonds, apparently either limits the migration of the organic radicals outside the vessel or at least minimizes their effects on the cellular metabolism."[5]

**proved in practice...** Paralleling similar findings in animals, clinicians have reported a generally lower



incidence of untoward reactions with Renografin-60 in urologic and cerebrovascular use. However, as with all intravascularly injected contrast agents, the possibility of severe reactions should be kept in mind (see Contraindications, Precautions and Side Effects below). In one study of over 600 urologic patients, the investigators reported that Renografin-60 produced urograms of diagnostic quality with a 6% incidence of side effects. The authors concluded: "It is hard to believe that any drug introduced intravenously could be so well borne by so many patients...."[6]

In a 74-patient study,[7] comparing Renografin-60 with diatrizoate sodium in carotid arteriography, Shealy commented: "With confused patients who are to have arteriography under local anesthesia, it is particularly desirable to have an agent that causes little pain." In this study, since "...60 per cent Renografin has resulted in considerably less pain than 50 per cent or 45 per cent [diatrizoate sodium]...we have converted to the routine use of 60 per cent Renografin for carotid arteriography; an additional 1,500 arteriograms done with 60 per cent Renografin have been quite satisfactory."[7]

**better tolerated even in pediatrics...** Citing some difficulties in administering contrast agents intravenously to children, Strasser *et al.*[8] selected Renografin-60 for intramuscular use in excretion urography in 16 pediatric patients because of the mild and relatively few reactions consistently associated with its use. The authors concluded: "The almost complete absence of any kind of local effect from its injection into the gluteal muscle and the absence of any serious reactions, local or systemic, indicate the safety of the medium."[8]

For brief summary of prescribing information please refer to the end of this advertisement.

# definition of efficacy...

**a thoroughly investigated meglumine salt** Extensively evaluated for over a decade, Renografin has been consistently shown to yield a high percentage of diagnostic quality films in many phases of contrast visualization.

Upon intravenous injection, Renografin is rapidly carried to the kidneys and is so well concentrated that renal passages—including renal pelvis, ureters and bladder—may be clearly visualized. This medium also provides high contrast vasography in visualization of the cerebral vessels and the peripheral arteries and veins.

**proved diagnostic excellence...** In a comparative study[9] of 3 contrast agents used in cerebral angiography by Doehner (comprising a cross section of an average neurosurgical practice), Renografin-60 was equal in the arterial phase and slightly superior in the venous phase of the examination.

Findings of a previously cited study by Orr *et al.*,[6] in intravenous pyelography, also attest to the diagnostic excellence of Renografin-60. "Satisfactory roentgenograms of the kidneys were obtained in 636 (97%) of the cases, demonstrating the great diagnostic value of this procedure." And, as noted previously, Shealy found the contrast agent to be "quite satisfactory" in 1,500 carotid arteriograms.[7]

**References: 1.** Macht, S. H.; Williams, R. H., and Lawrence, P. S.: Amer. J. Roentgen. *98*:79 (Sept.) 1966. **2.** Fischer, H. W., and Eckstein, J. W.: Amer. J. Roentgen. *86*:166 (July) 1961. **3.** Fischer, H. W., and Cornell, S. H.: Radiology *85*:1013 (Dec.) 1965. **4.** Cooley, R. N., *et al.*: Angiology *15*:107 (Mar.) 1964. **5.** Gensini, G. G., and DiGiorgi, S.: Radiology *82*:24 (Jan.) 1964. **6.** Orr, L. M.; Campbell, J. L., and Thomley, M. W.: J.A.M.A. *169*:1156 (Mar.) 1959. **7.** Shealy, C. N.: J. Neurosurg. *20*:137 (Feb.) 1963. **8.** Strasser, N. F., *et al.*: Radiology *79*:408 (Sept.) 1962. **9.** Doehner, G. A., and Brugger, G. E.: New York J. Med. *60*:4022 (Dec.) 1960.

**Contraindication**

A history of sensitivity to iodine per se or to other contrast media is not an absolute contraindication to the use of meglumine diatrizoate.

**Precautions and Side Effects**

Severe, life-threatening reactions are rare; when they occur they suggest hypersensitivity. A personal or family history of asthma or allergy warrants special attention and may predict, more accurately than pretesting, the likelihood of a reaction, although not the type nor severity of the reaction in the individual.

The value of any pretest is questionable. The pretest most performed is the slow injection of 0.5-1.0 cc. of the preparation into a peripheral vein. An impending reaction is often indicated by transient burning and flushing, pain, "jump-like" reactions, respiratory difficulty, faintness, sneezing, itching, nausea, vomiting or urticaria. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. Antiallergic drugs may be used to advantage. In a few cases, the reactions to the test dose have been extremely severe.

The more serious anaphylactoid reaction requires immediate treatment and may occur despite a negative sensitivity test. An emergency tray consisting of vasopressor drugs, epinephrine hydrochloride 1:1000, methoxamine (Vasoxyl) or metaraminol bitartrate (Aramine), and glucose and saline is recommended. Oxygen and instruments to guarantee a clear airway must be readily available.

Caution must be exercised, especially in cerebral angiography in extreme age, in severely debilitated patients and in those with marked or severe hypertension, advanced arteriosclerosis, cardiac decompensation, recent cerebral embolism, or thrombosis, chronic pulmonary emphysema and in cyanotic infants.

For full details the Package Insert should be read.

**Supply:** Renografin-60 is supplied in vials and ampuls as a sterile aqueous solution providing 60% meglumine diatrizoate containing approximately 29% firmly bound iodine.

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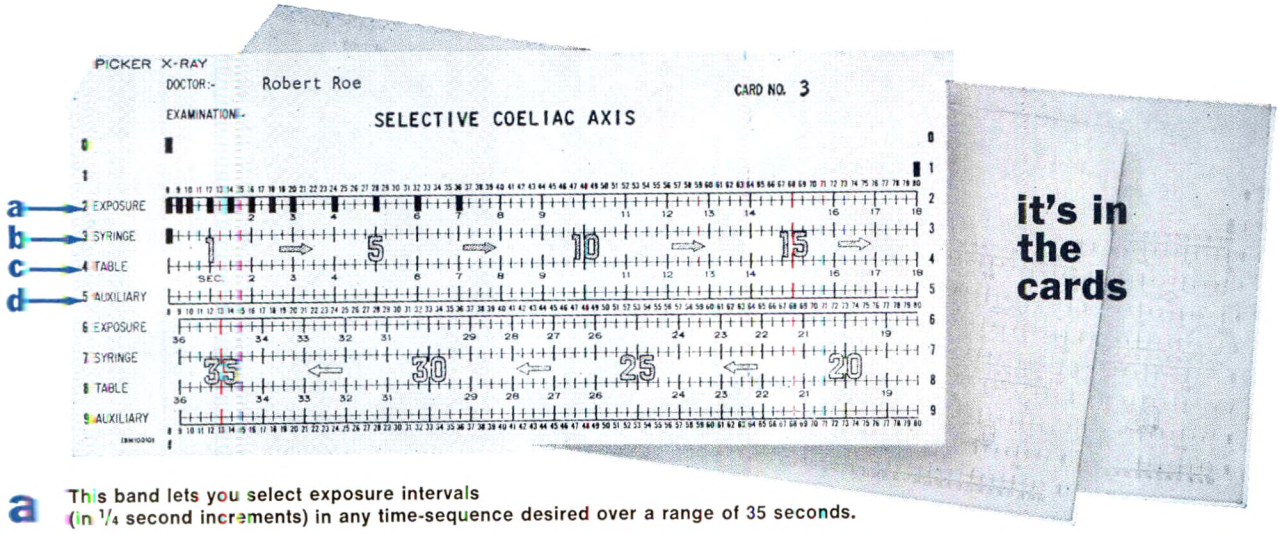
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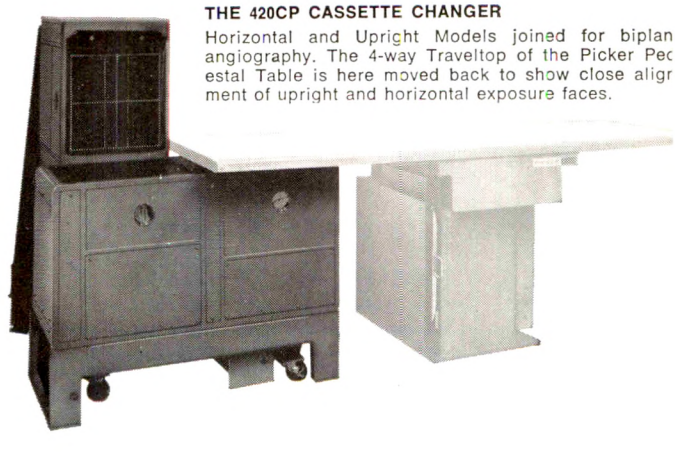
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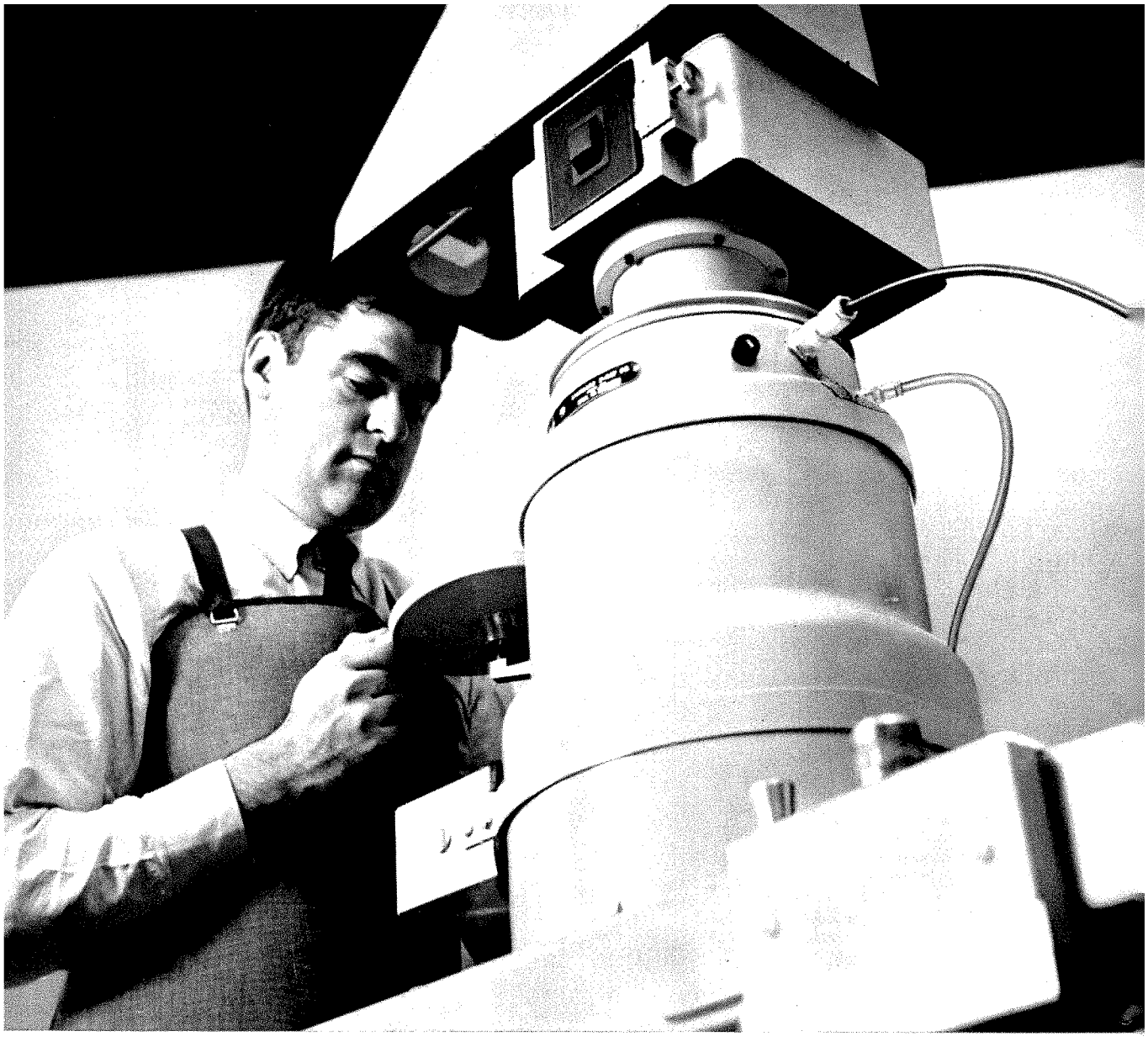
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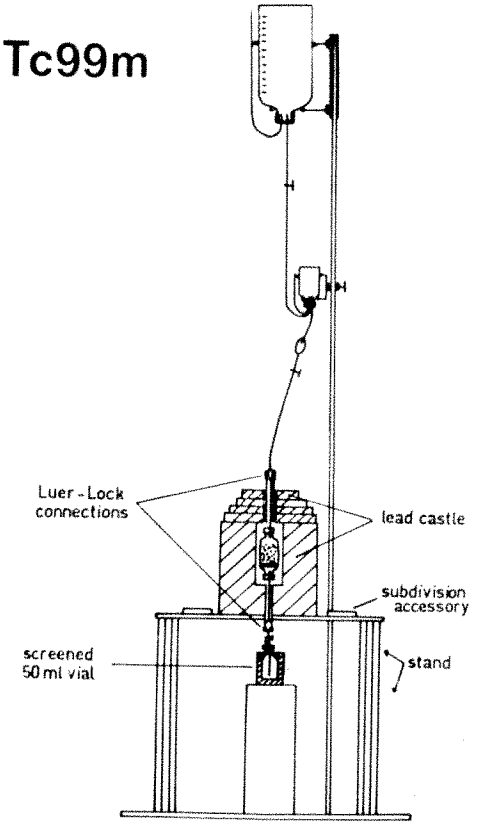
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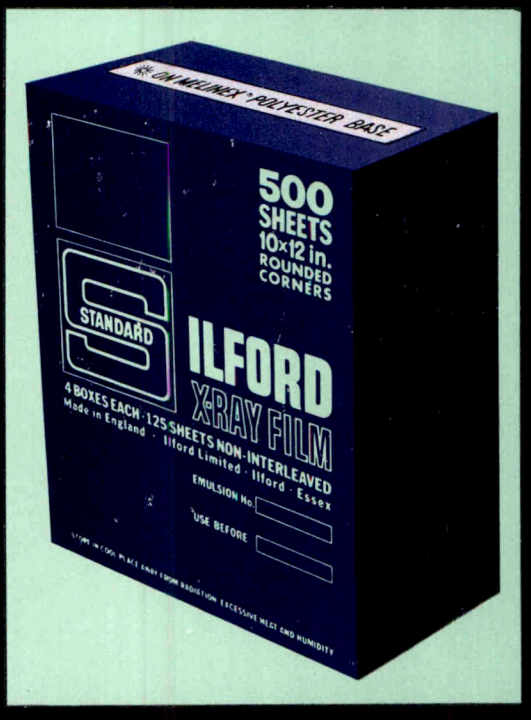
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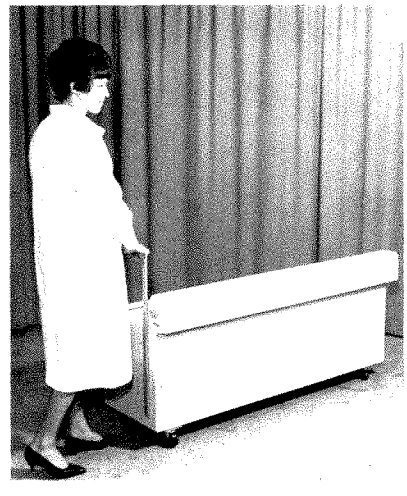
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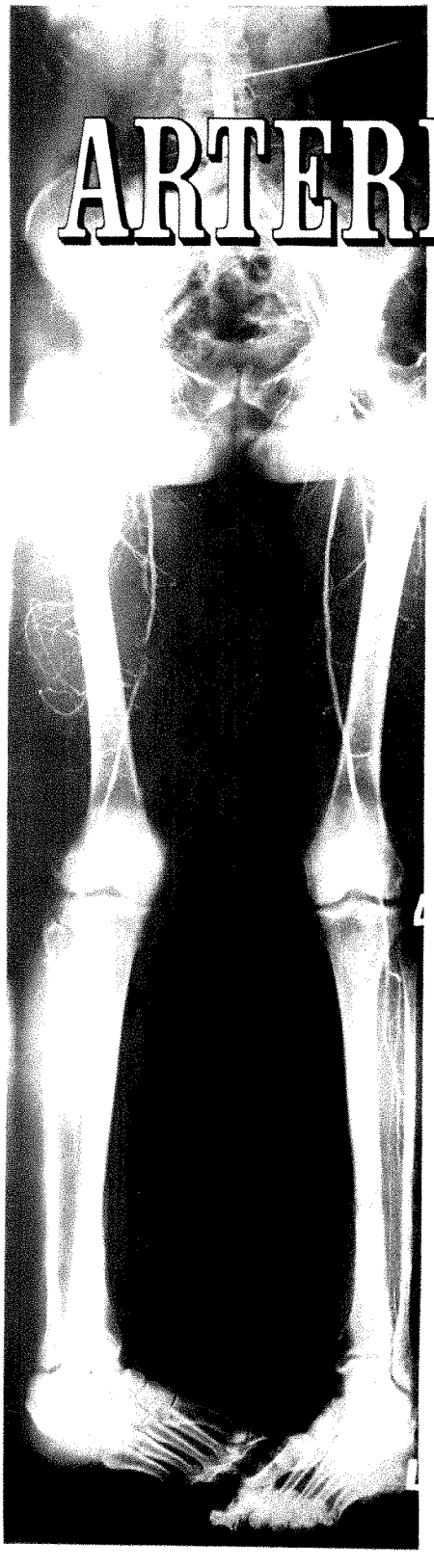
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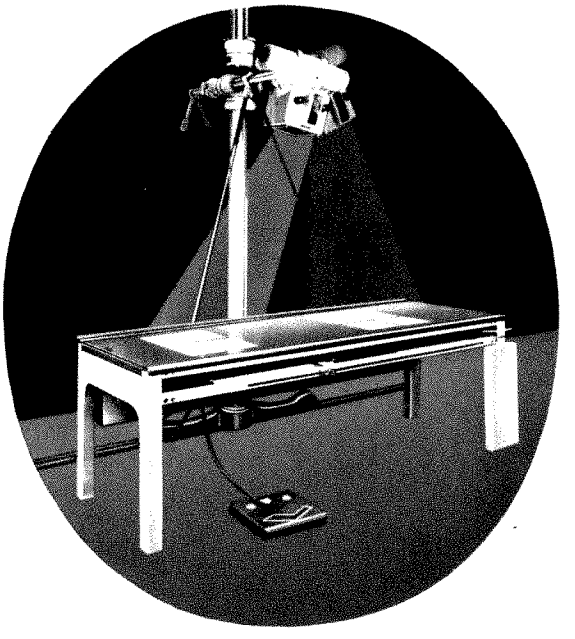
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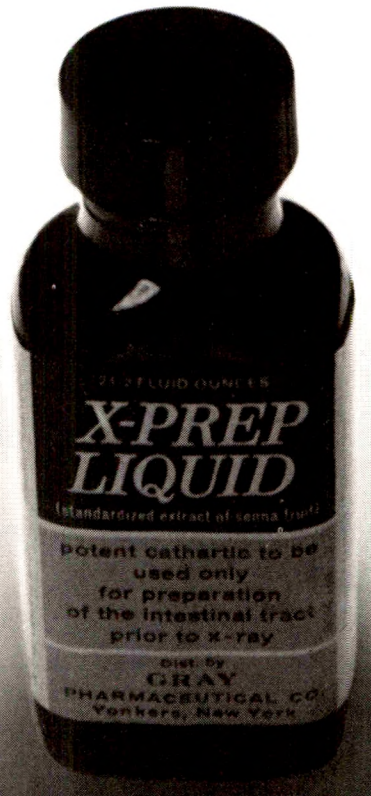
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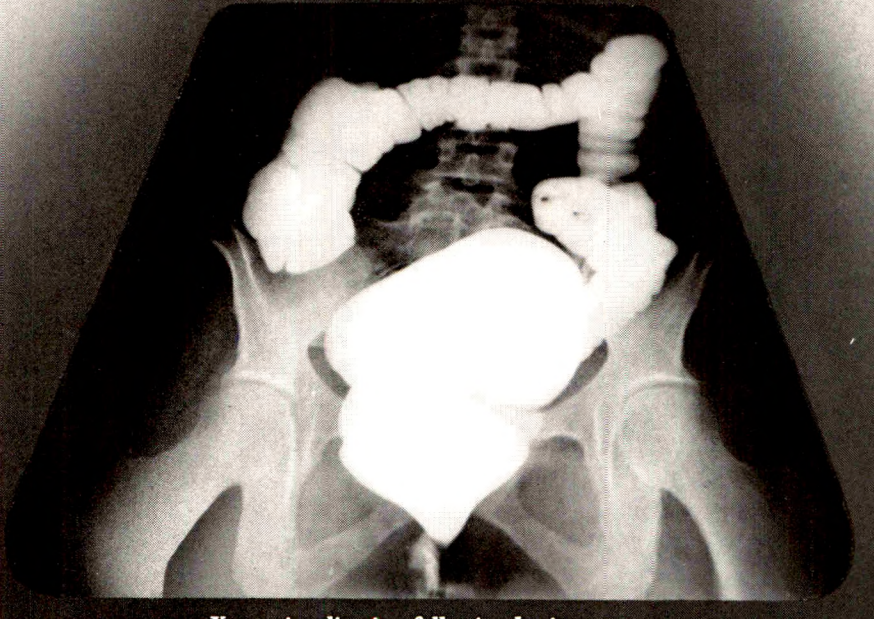
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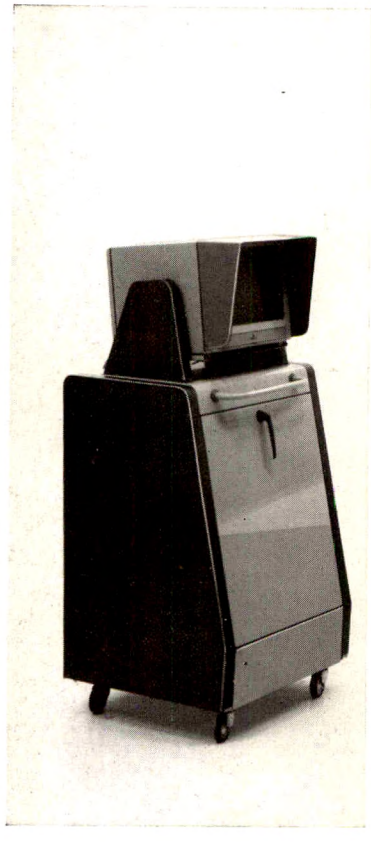


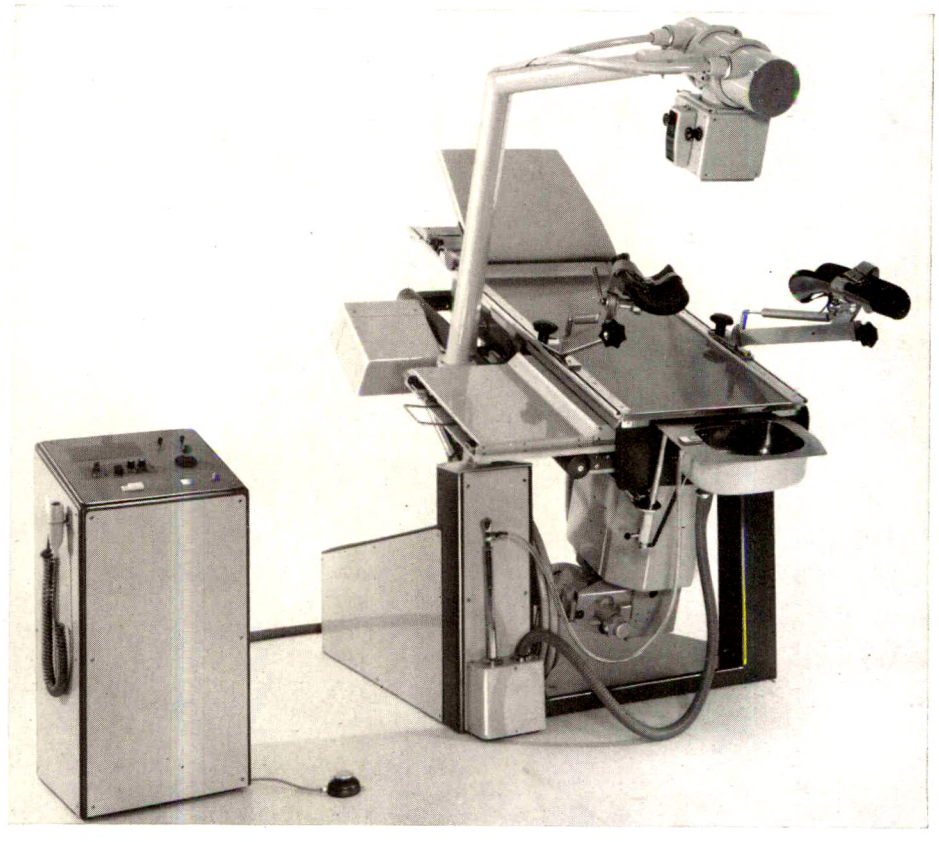



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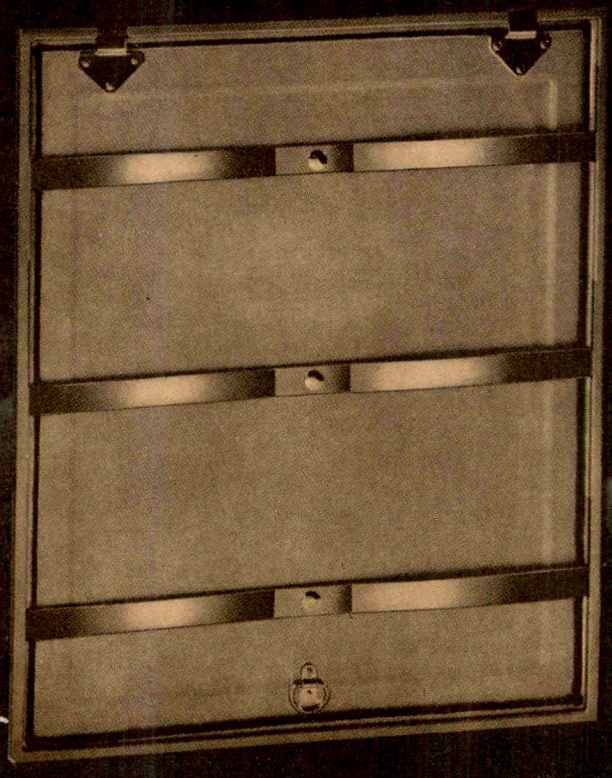
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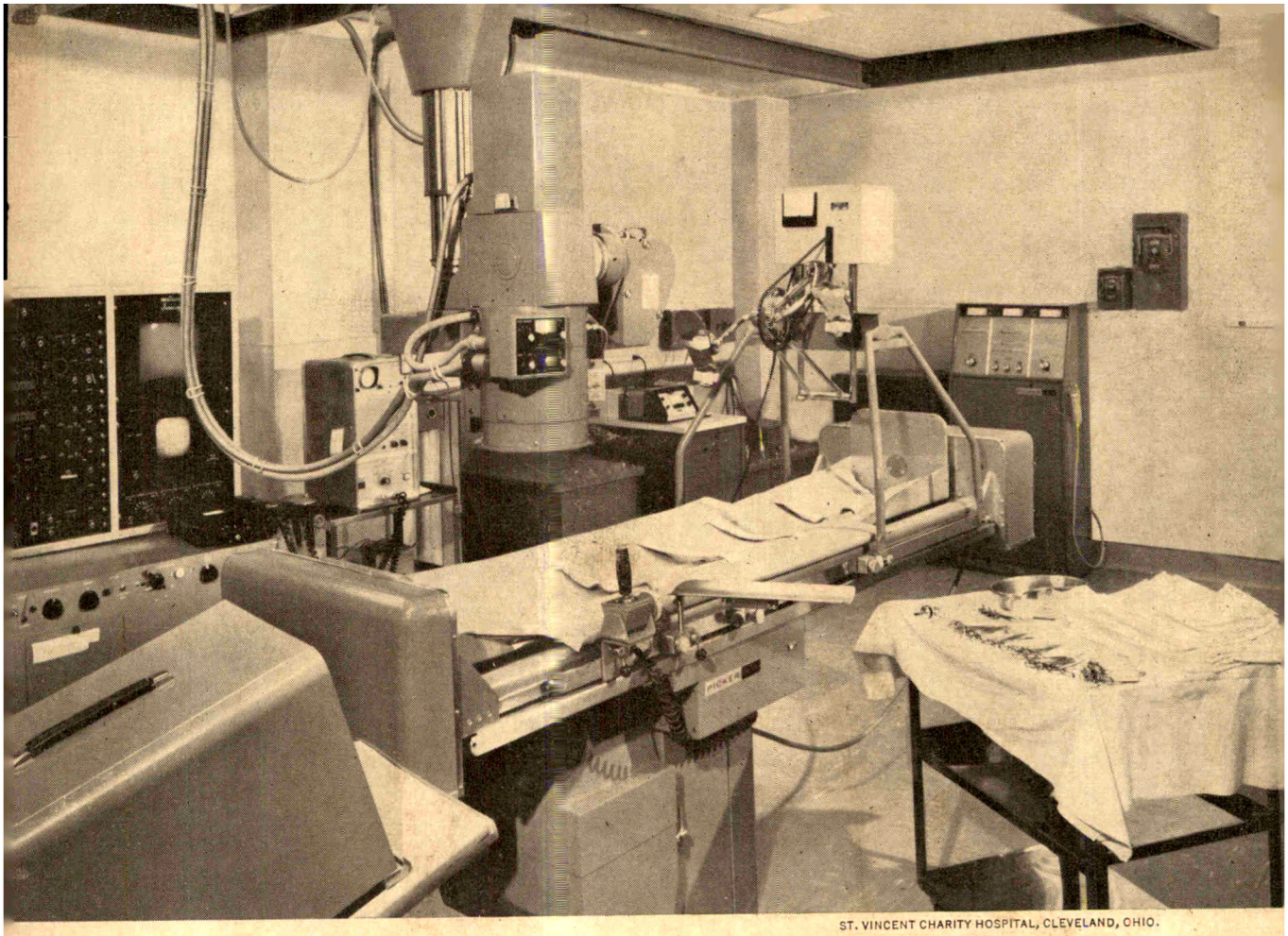
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VOLUME 101 OCTOBER, 1967 NUMBER 2

# 4 of every 5 new Departments of Nuclear Medicine get started with a Magnascanner®

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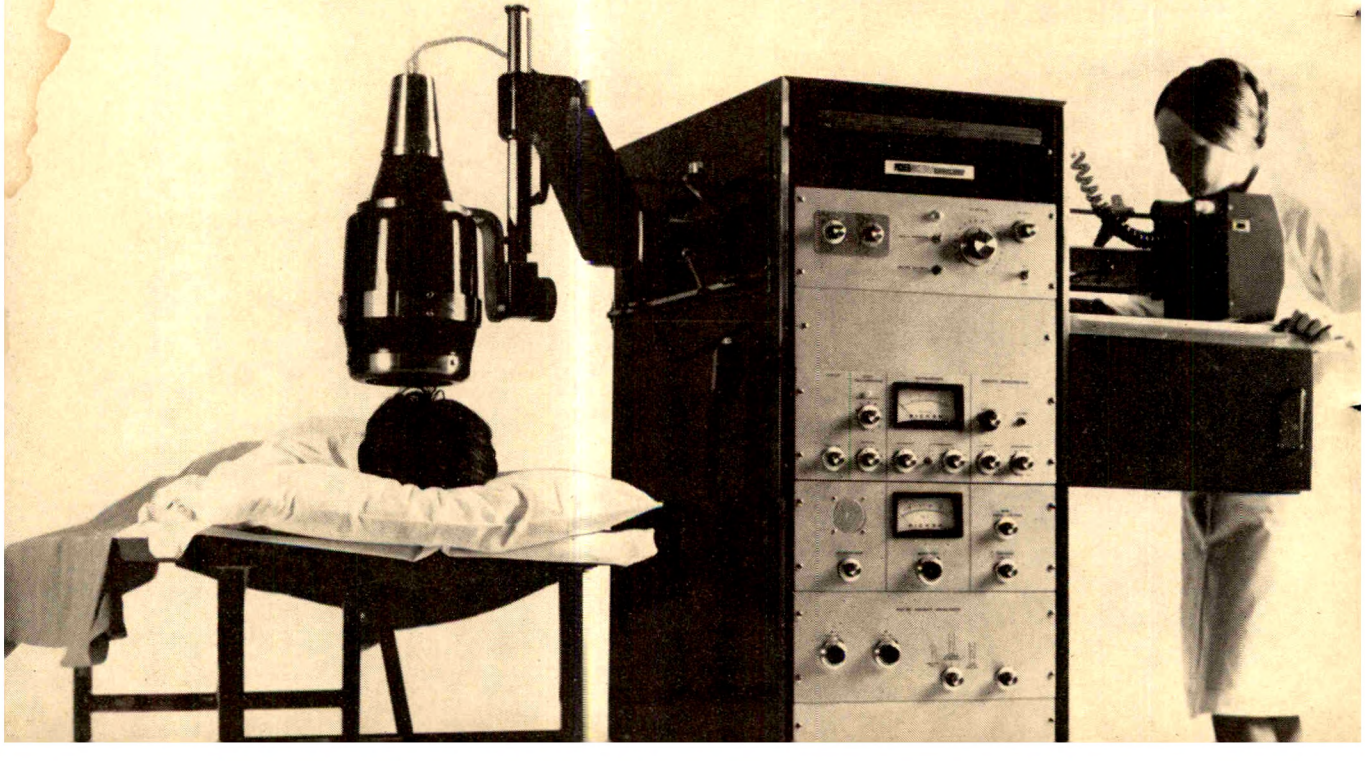
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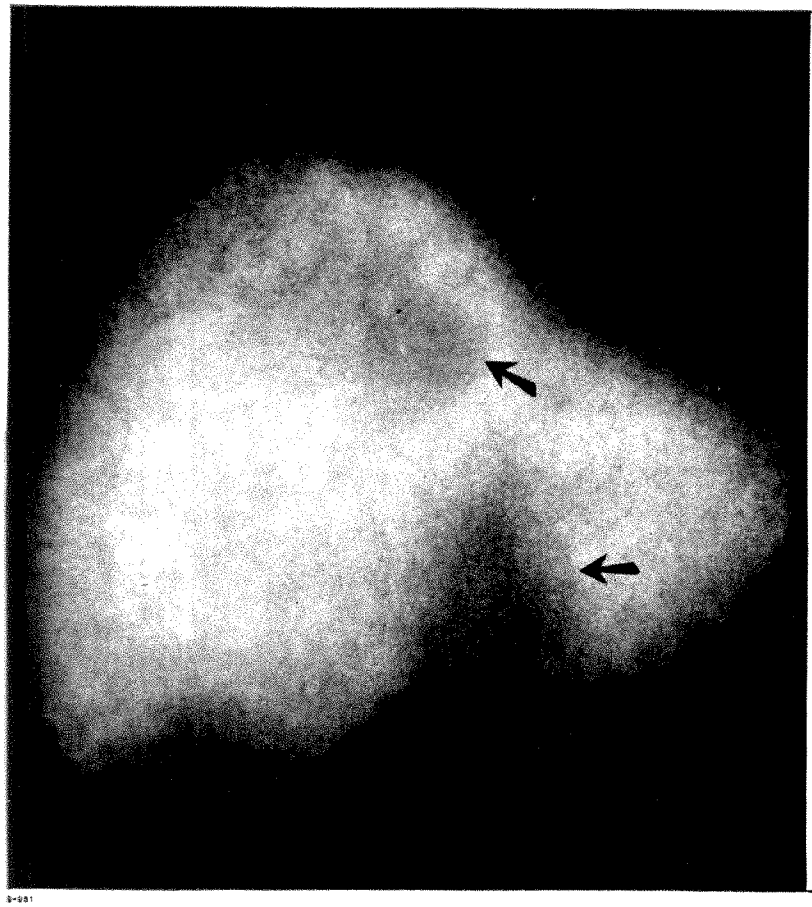
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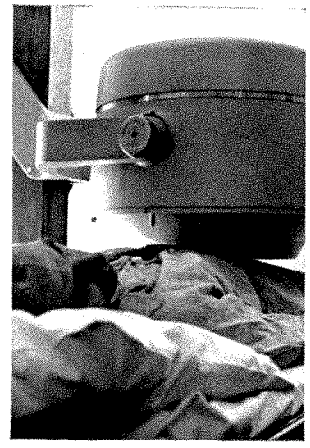
A few final words about our obligations to you. We accept the premise that our obligations don't end at time of delivery. We not only install the instrument and show you how to use it, but we feel it our obligation to help train personnel when an institution new to this field doesn't have experienced personnel on staff. We have *other* obligations to you which our people are happy to detail. But meanwhile, consider further the choice of the Magnascanner (and the Picker commitment to you) as the keystone of *your* service too by requesting our new brochure number 130X.

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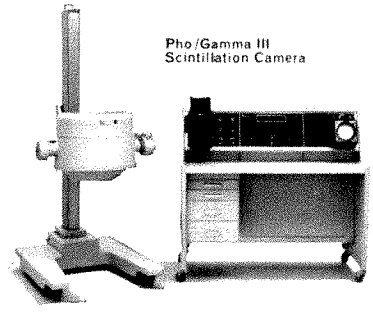
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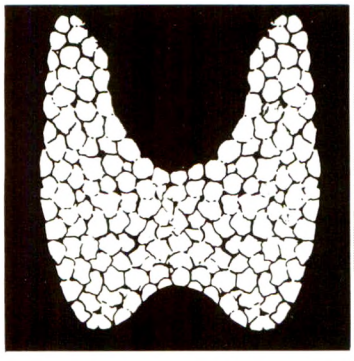
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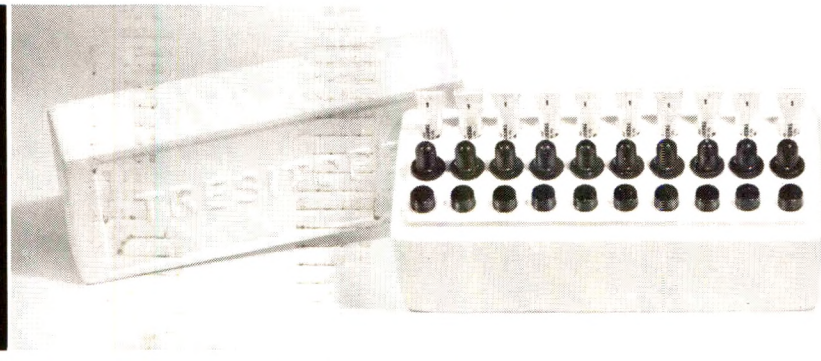
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a research concept in radiodiagnostics

# Some significant advances in thyroid-testing technique

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The technical difficulties encountered in preparing different batches of resin sponges are avoided.

Moreover, because it is an *in vitro* test, it is diagnostically significant in the presence of unrelated nonthyroidal factors that are known to complicate interpretation of other test findings. More specifically, the test is unaffected by anxiety, hypertension, congestive heart failure, or administration of mercurial agents. And it is unaffected by prior administration of most iodine-containing preparations that can completely nullify the results of other thyroid function tests for considerable periods.

**$I^{125}$ versus $I^{131}$**

The use of $I^{125}$ rather than $I^{131}$ to label the liothyronine employed in the test is also advantageous. Employing $I^{125}$ considerably lengthens the shelf life of the liothyronine because $I^{125}$ has a longer half-life and also because it emits no beta rays to affect the stability of liothyronine. The half-life of $I^{125}$ is considered to be 60 days while $I^{131}$ has a half-life span of approximately 8 days. Other advantages of $I^{125}$-labeled material include lowered radiation exposure to the technician, yet radioactivity is well within good counting range of modern standard equipment and *in vitro* counting is quite efficient.

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**NOTE:** While the resin uptake test is a very useful aid in the evaluation of thyroid function, it should not be used as the sole basis for such an evaluation. In any patient, the clinical state is probably the best indication of thyroid status, and *any* laboratory test must be interpreted with caution when test results do not agree with clinical evidence.

**Precautions**

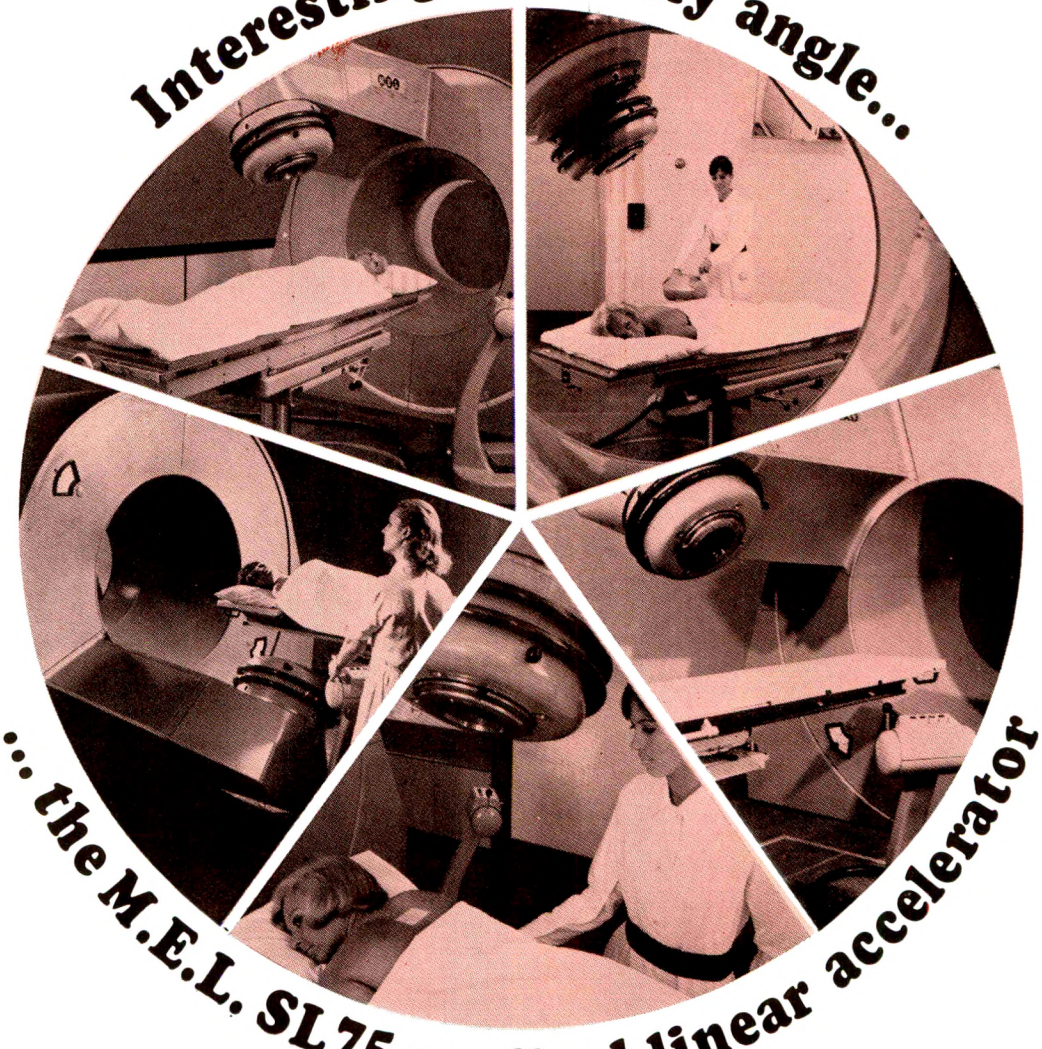
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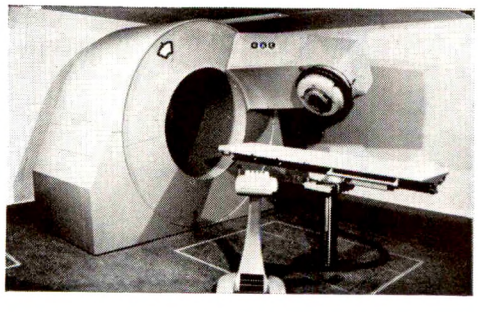
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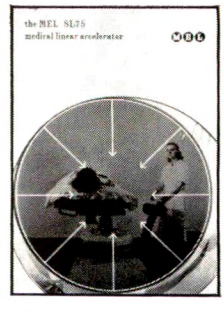
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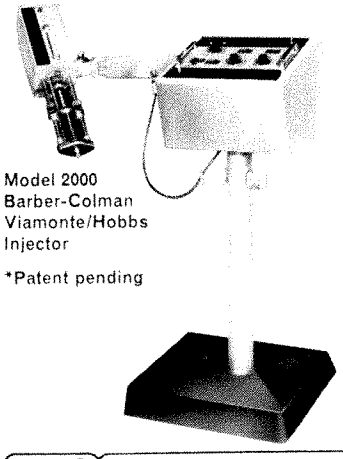
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As the originator of injectors with automatic flow rate control, we often are asked how this concept actually operates.

*True automatic flow rate control is identified by two characteristics. Both must exist for safer, more efficient angiography:*

1. Flow rate control replaces time-consuming interpolation from nomographs for catheter size, media viscosity, and other injection variables needed to estimate injection pressures. Dialing desired flow rate on the injector control panel simplifies selection procedure.

2. The control system must include a method of automatic "feedback" so that actual injection rate matches the rate you select, regardless of injection variables. This feedback action is what permits repeated accuracy. Barber-Colman Viamonte/Hobbs injectors* have flow rate control with a feedback system to maintain linear flows in ml/sec, up through those flows requiring approximately 1100 psi.



Model 2000
Barber-Colman
Viamonte/Hobbs
Injector

*Patent pending

**Significance of positive feedback**

An injector's feedback capability becomes significant when we recall that any injector is simply a power-operated syringe. Optimum x-ray contrast still depends on bolus size and delivery rate—both determined by you through either of two basic approaches:

1. *The negative approach*—measuring all variables which work *against* injection because of pressure resistance; then determining drive force (when pushing syringe piston) needed to overcome total pressure resistance.

To achieve highest accuracy when using the negative approach, you would measure resistance pressures prior to each injection. But this takes too much time, so you have consulted charts which provide *average* pressures for each variable. Using these averages, the estimated total pressure resistance could vary 5%, 15%—and even more—from the actual value.

2. *The positive approach*—making all factors work *for* you—is the function of Barber-Colman's Viamonte/Hobbs injector. It moves the syringe piston at a constant, preselected flow rate—regardless of any changes in pressure resistance—up to the power capacity of the injector.

Flow rate is kept constant by the Barber-Colman injector's servo loop feedback system described below. This not only moves the piston at your preset rate, but it also monitors piston motion continuously to insure that the piston actually *is* moving at that speed, whatever your injection variables.

**How Barber-Colman's feedback system works**

The feedback control system in a Viamonte/Hobbs injector is analogous to the human nervous system and its ability to adapt to various situations.

Speed of the motor which pushes the syringe piston is continuously sensed and "fed back" into the injector's control circuitry—much the same as neurological data is fed into the brain by human senses. And just as the brain "adjusts" human muscles in response to changing sensory impressions, the injector's control system instantly and automatically adjusts motor speed so that the syringe piston injects constantly at the rate you selected.

True automatic flow rate control must include this feedback loop between actual output and the injection rate you dial in. Without it, an injector cannot compensate for the many variables inherent in angiographic procedures. This is why any mere mechanized version of a nomograph is inadequate.

Until the advent of Barber-Colman's Viamonte/Hobbs injector, the "negative approach" was the only one used in operation of angiographic injectors. Now the picture has changed.

*If you are interested in the "positive approach" to injectors, write for latest literature.*

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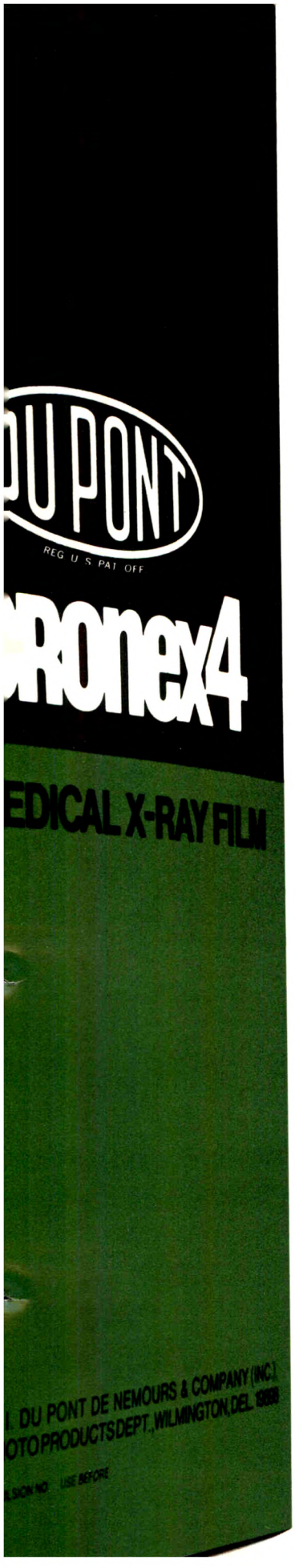
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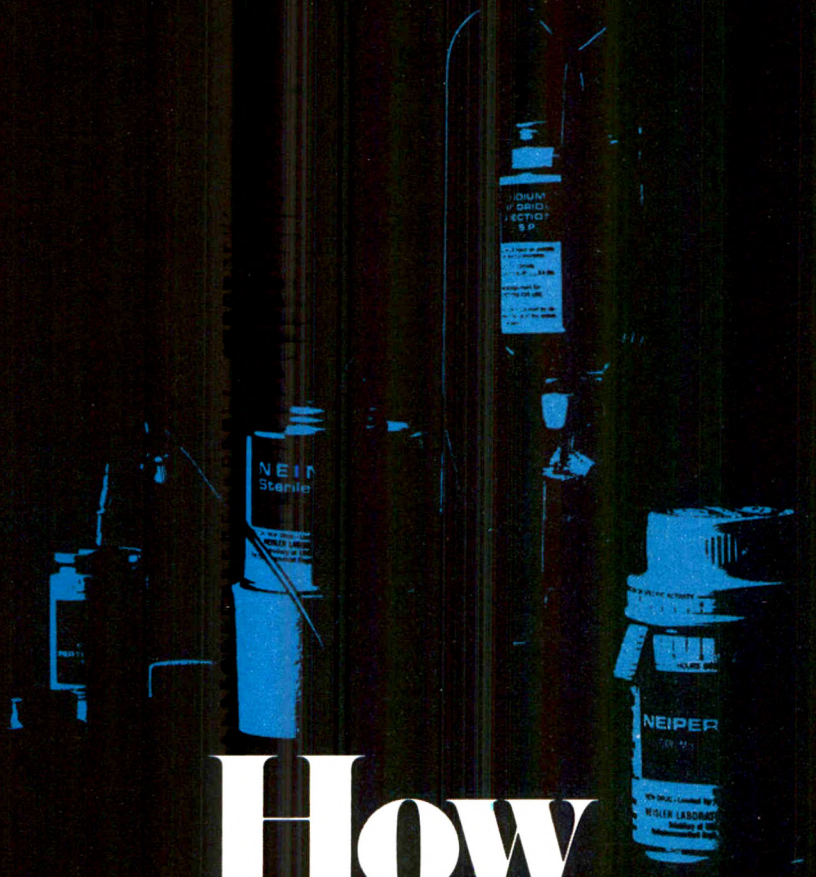
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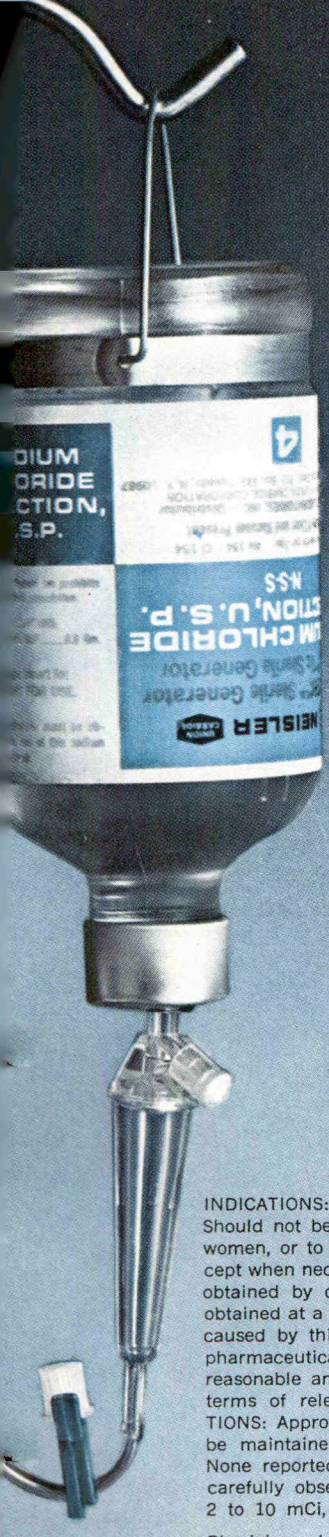
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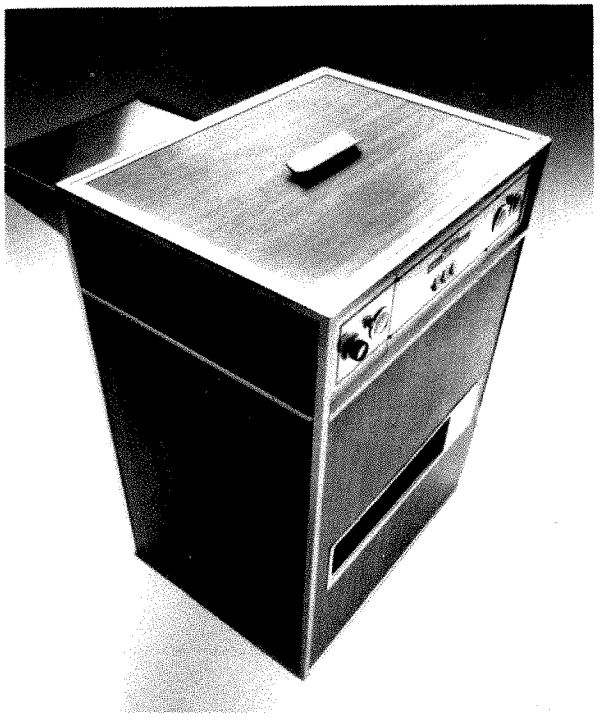
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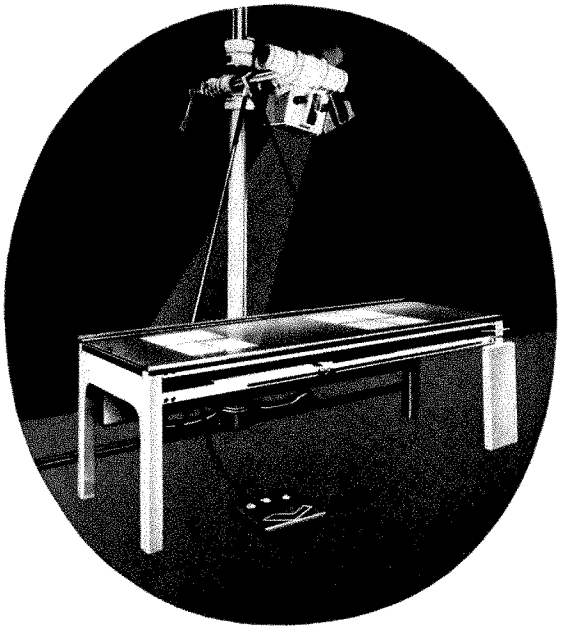
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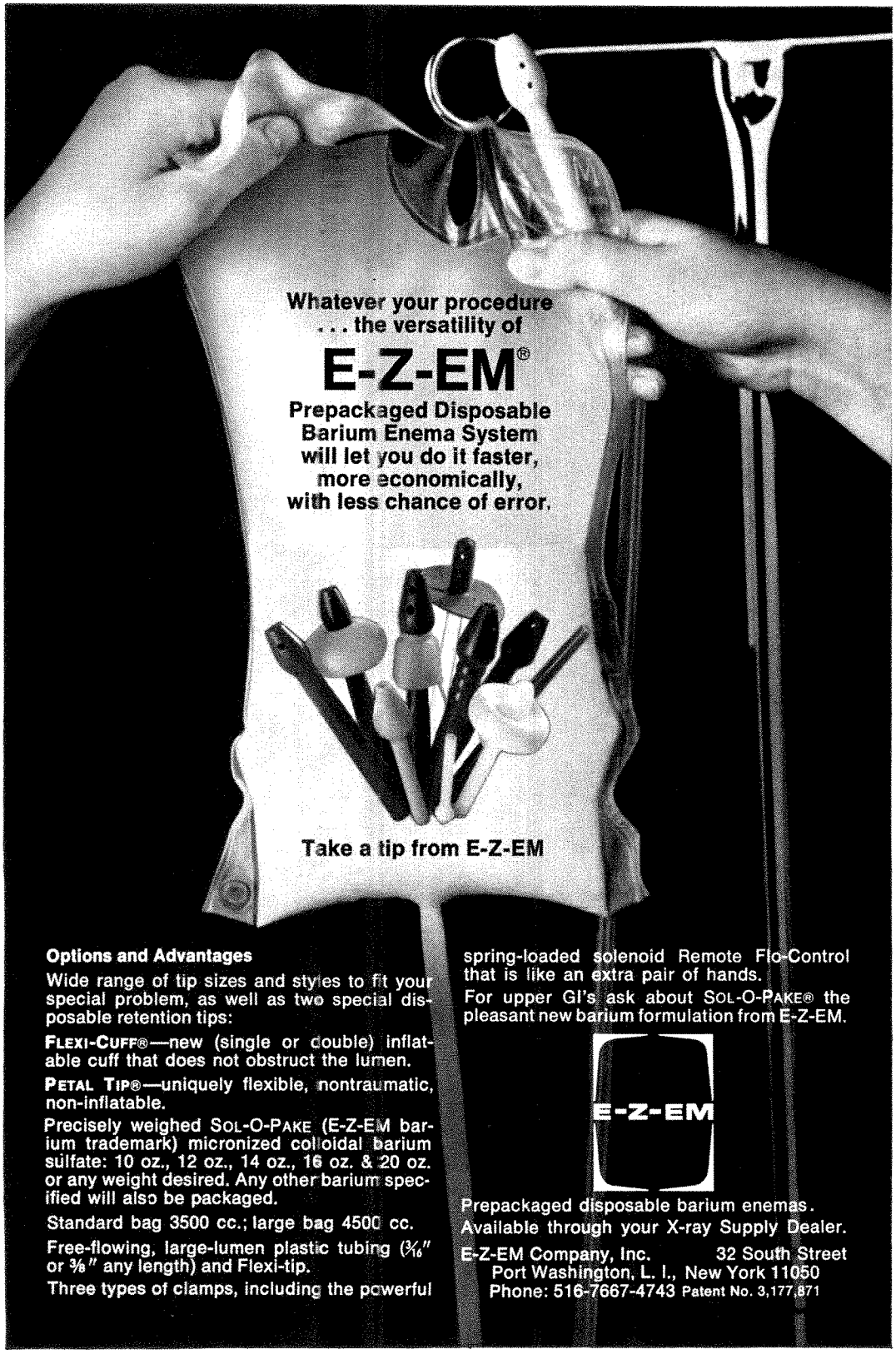


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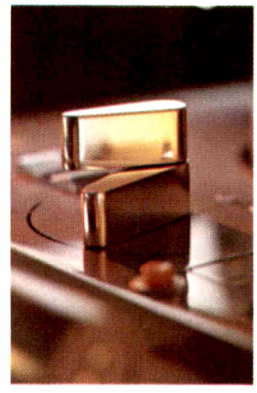
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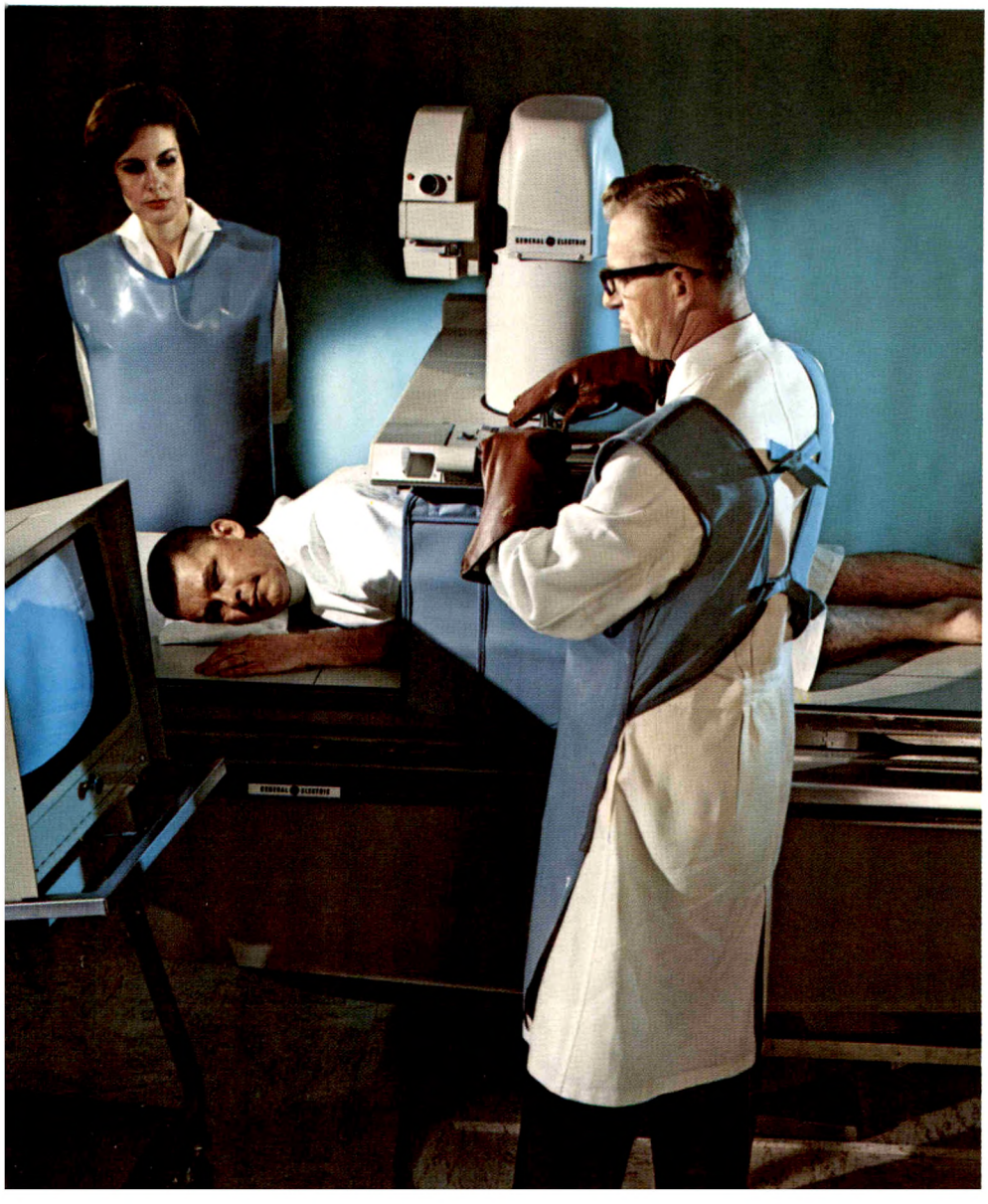

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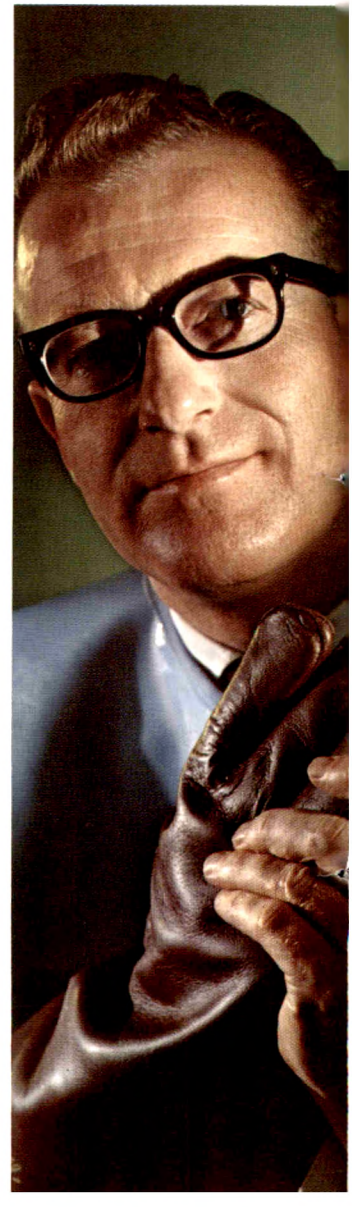
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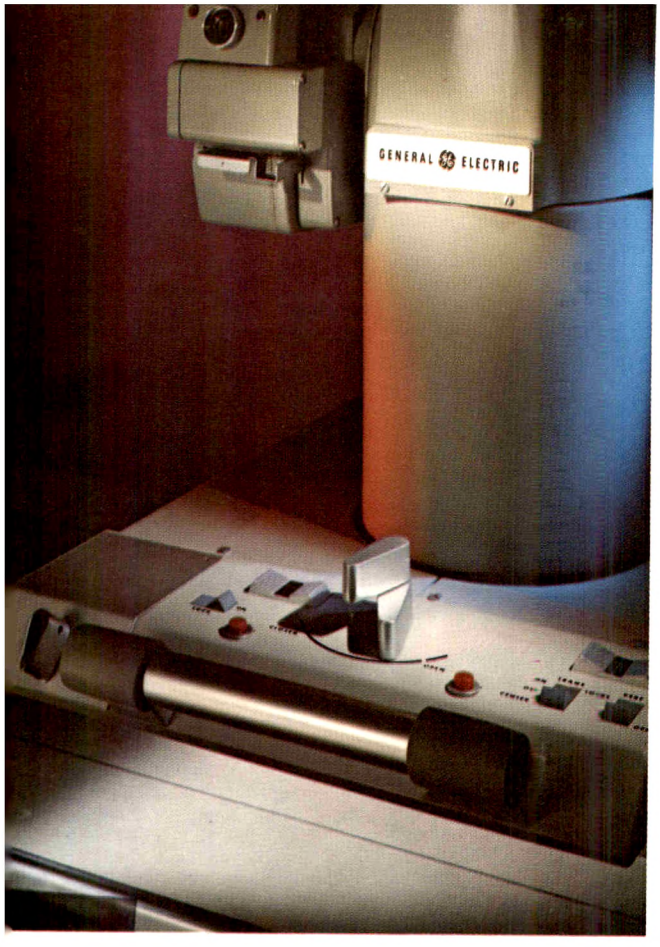
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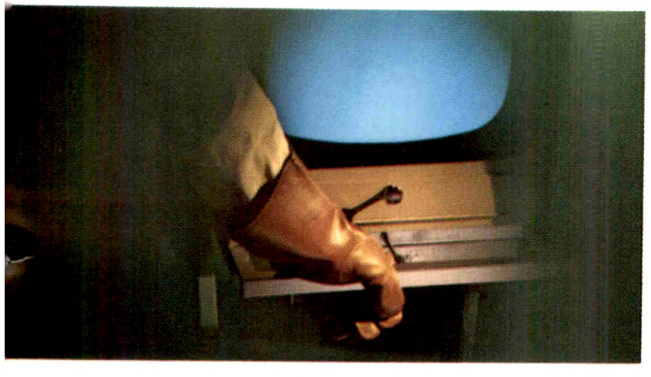





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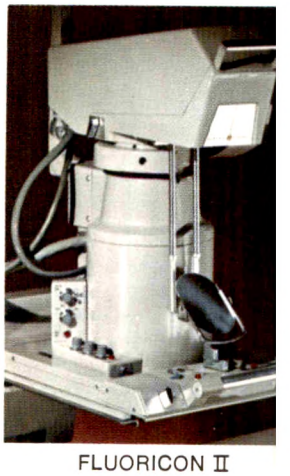
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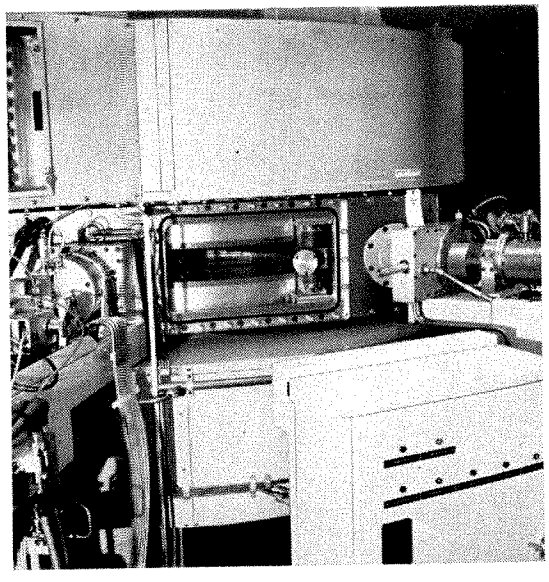
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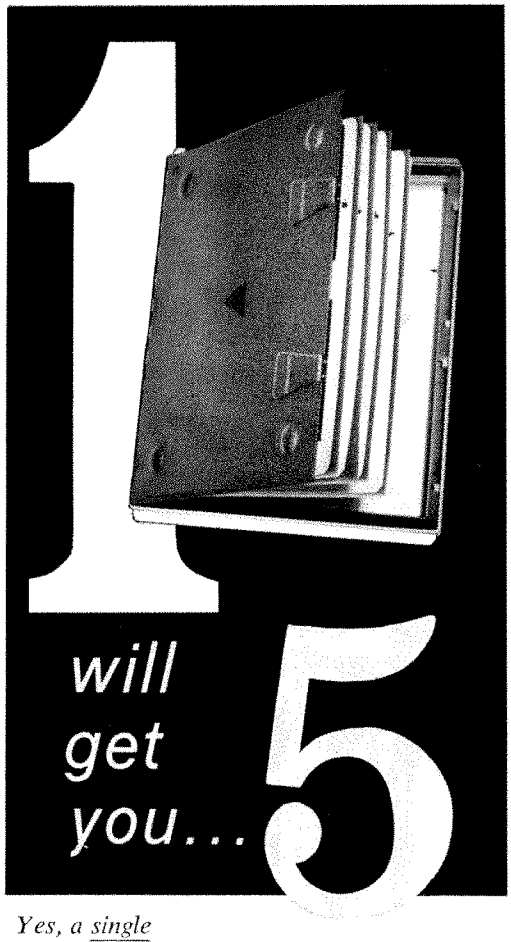
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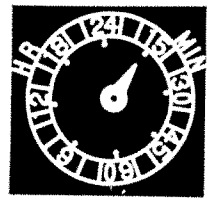
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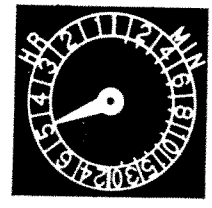



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a research concept in contrast visualization

# for better definition of disease: documenting differences between contrast agents

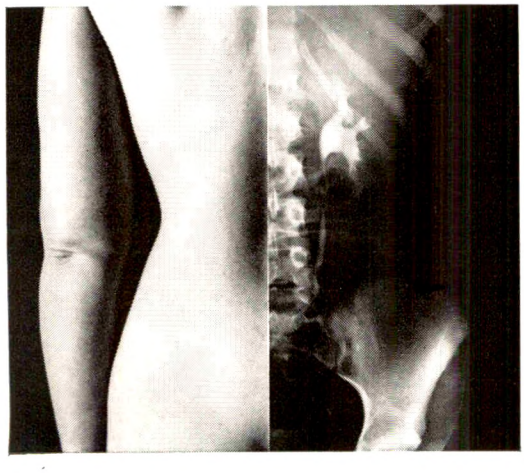
**No. 1 in a series:**

Renografin-60 (meglumine diatrizoate injection) preferred in a study of pyelographies of 2,234 patients.

In a new large scale study,[1] "...to determine which medium would produce adequate visualization of the urinary tract with the fewest toxic effects on the patient," Macht *et al.* have compared Renografin-60 with 2 other contrast agents.

Data were analyzed in 2,234 unselected (consecutive) patients, according to age, sex, and general disease group for the study population as a whole. The first 683 patients received 50% diatrizoate sodium solution, the next 921 patients received Renografin-60, and the final 630 patients received 66.8% sodium iothalamate solution.[1]

**criteria for quality and comparative safety** The contrast agents were evaluated for quality of diagnostic films as follows: films showing a dense concentration of contrast medium with filling and visualization of all major and minor calyces, infundibula, pelves and almost all of each ureter were listed as "good"; films showing less concentration with incomplete visualization of all portions of urinary tract but sufficient to produce diagnostically adequate films were listed as "fair"; films showing unsatisfactory visualization of urinary tract, or films which could not be interpreted, as "poor." The media were also evaluated as to incidence of the following side effects: nausea, vomiting, fainting, shock or severe reaction, hiccups, hives, pain in arm, sneezing, hot flushes, stuffiness of nose or ears.[1]



The following chart* shows comparative results of the 3 media according to age category:

*quality of pyelograms and side effects expected in standard population of 1,000 patients by age, according to type of medium used*

| medium used and type of patient | standard population | quality of pyelograms good | fair | poor | with side effects |
|---|---|---|---|---|---|
| meglumine diatrizoate | | | | | |
| all ages, | 1,000 | 827 | 123 | 50 | 41 |
| 0-19† | 126 | 122 | 3 | 1 | 3 |
| 20-49 | 415 | 374 | 35 | 6 | 27 |
| 50-69 | 315 | 240 | 51 | 24 | 9 |
| 70 or older | 144 | 91 | 34 | 19 | 2 |
| diatrizoate sodium | | | | | |
| all ages, | 1,000 | 782 | 134 | 84 | 72 |
| 0-19 | 126 | 115 | 6 | 5 | 7 |
| 20-49 | 415 | 378 | 26 | 11 | 40 |
| 50-69 | 315 | 227 | 64 | 24 | 19 |
| 70 or older | 144 | 62 | 38 | 44 | 6 |
| sodium iothalamate | | | | | |
| all ages, | 1,000 | 883 | 81 | 36 | 54 |
| 0-19 | 126 | 122 | 4 | 0 | 7 |
| 20-49 | 415 | 393 | 20 | 2 | 29 |
| 50-69 | 315 | 269 | 32 | 14 | 13 |
| 70 or older | 144 | 99 | 25 | 20 | 5 |

*Adapted from Macht[1] †In some patients in this age group, higher than recommended doses were used.

**In theory:** "The choice of contrast agent should ideally be individualized according to the age, sex, and disease group of the patient in order to obtain a high probability of complete visualization of the urinary tract with low probability of adverse side effects."[1]

**In practice:** "The choice is to be made...on the basis of the agent which gives the best concentration and the fewest side effects in the greatest number of patients regardless of age, sex, or disease category."[1]

**In order of preference:** Although Renografin-60 was not rated highest in all categories, the authors feel that their preference of the contrast media for intravenous pyelography would be: first, Renografin-60; second, sodium iothalamate; third, diatrizoate sodium.[1]

For brief summary of prescribing information, please refer to the end of this advertisement.

# definition of safety...

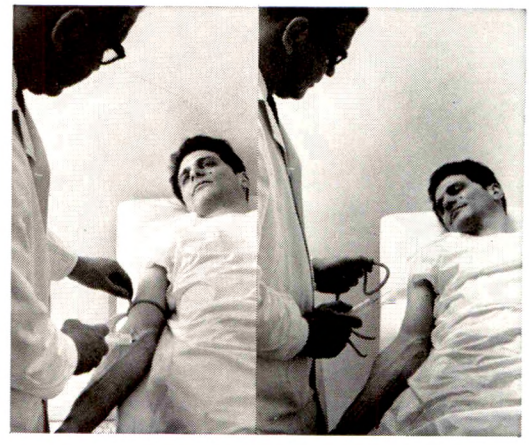
**demonstrated in animals...** Since meglumine diatrizoate is also used for cerebral angiography, toxicity studies of administration via the carotid artery are therefore of interest. Fischer and Eckstein[2] designed angiographic studies in animals in which procedures were very similar to clinical angiography, and which yielded data that was quantitative, graphic and nonsubjective.[2] According to the authors: "We selected the alterations in arterial blood pressure, venous pressure, heart rate and rhythm, the electrocardiogram and end-expiratory $CO_2$ concentration resulting from experimental cerebral angiography as refined, sensitive indications of the toxicity of a particular contrast material."[2] Their results of measured cardiovascular functions in dogs indicated that meglumine diatrizoate was far less toxic than four other contrast media.[2] As Fischer and Cornell reported in a later study: "Despite the testing of more highly concentrated solutions and larger doses, the cardiovascular responses [in dogs] from methylglucamine [meglumine] salts were much less than from sodium salts, an observation consistent with previous experiments."[3]

Other investigators have documented the comparative safety of meglumine salts in experimental studies. In order to determine reaction and tolerance of the intestines, Cooley[4] injected meglumine diatrizoate into mesenteric arteries of dogs and found no damage. Gensini *et al.*[5] reported that cardiovascular responses with it were almost identical to blood transfusions.

**theory of lower toxicity with meglumine...** Gensini and DiGiorgi have offered a hypothesis to explain their findings of lesser toxicity with experimental intravascular injections of methylglucamine (meglumine) salts. "When a relatively undiluted amount of sodium salts of a contrast agent is injected in an artery and carried by the blood stream toward the capillary bed, its molecules rapidly dissociate and readily diffuse through the capillary membrane and into the tissue. There, both the toxic effect of the iodine-containing organic radical and the increased concentration of sodium will readily manifest themselves. At equal concentrations of sodium, the end results will closely reflect the intrinsic toxicity of the iodine-containing organic radical on the tissues...."[5]

"In the case of the methylglucamine compounds, the same dissociation takes place. However the larger methylglucamine molecule, rich in hydrogen bonds, apparently either limits the migration of the organic radicals outside the vessel or at least minimizes their effects on the cellular metabolism."[5]

**proved in practice...** Paralleling similar findings in animals, clinicians have reported a generally lower



incidence of untoward reactions with Renografin-60 in urologic and cerebrovascular use. However, as with all intravascularly injected contrast agents, the possibility of severe reactions should be kept in mind (see Contraindications, Precautions and Side Effects below). In one study of over 600 urologic patients, the investigators reported that Renografin-60 produced urograms of diagnostic quality with a 6% incidence of side effects. The authors concluded: "It is hard to believe that any drug introduced intravenously could be so well borne by so many patients...."[6]

In a 74-patient study,[7] comparing Renografin-60 with diatrizoate sodium in carotid arteriography, Shealy commented: "With confused patients who are to have arteriography under local anesthesia, it is particularly desirable to have an agent that causes little pain." In this study, since "...60 per cent Renografin has resulted in considerably less pain than 50 per cent or 45 per cent [diatrizoate sodium]...we have converted to the routine use of 60 per cent Renografin for carotid arteriography; an additional 1,500 arteriograms done with 60 per cent Renografin have been quite satisfactory."[7]

**better tolerated even in pediatrics...** Citing some difficulties in administering contrast agents intravenously to children, Strasser *et al.*[8] selected Renografin-60 for intramuscular use in excretion urography in 16 pediatric patients because of the mild and relatively few reactions consistently associated with its use. The authors concluded: "The almost complete absence of any kind of local effect from its injection into the gluteal muscle and the absence of any serious reactions, local or systemic, indicate the safety of the medium."[8]

For brief summary of prescribing information please refer to the end of this advertisement.

# definition of efficacy...

**a thoroughly investigated meglumine salt** Extensively evaluated for over a decade, Renografin has been consistently shown to yield a high percentage of diagnostic quality films in many phases of contrast visualization.

Upon intravenous injection, Renografin is rapidly carried to the kidneys and is so well concentrated that renal passages—including renal pelvis, ureters and bladder—may be clearly visualized. This medium also provides high contrast vasography in visualization of the cerebral vessels and the peripheral arteries and veins.

**proved diagnostic excellence...** In a comparative study[9] of 3 contrast agents used in cerebral angiography by Doehner (comprising a cross section of an average neurosurgical practice), Renografin-60 was equal in the arterial phase and slightly superior in the venous phase of the examination.

Findings of a previously cited study by Orr *et al.*,[6] in intravenous pyelography, also attest to the diagnostic excellence of Renografin-60. "Satisfactory roentgenograms of the kidneys were obtained in 636 (97%) of the cases, demonstrating the great diagnostic value of this procedure." And, as noted previously, Shealy found the contrast agent to be "quite satisfactory" in 1,500 carotid arteriograms.[7]

**References: 1.** Macht, S. H.; Williams, R. H., and Lawrence, P. S.: Amer. J. Roentgen. 98:79 (Sept.) 1966. **2.** Fischer, H. W., and Eckstein, J. W.: Amer. J. Roentgen. 86:166 (July) 1961. **3.** Fischer, H. W., and Cornell, S. H.: Radiology 85:1013 (Dec.) 1965. **4.** Cooley, R. N., *et al.*: Angiology 15:107 (Mar.) 1964. **5.** Gensini, G. G., and DiGiorgi, S.: Radiology 82:24 (Jan.) 1964. **6.** Orr, L. M.; Campbell, J. L., and Thomley, M. W.: J.A.M.A. 169:1156 (Mar.) 1959. **7.** Shealy, C. N.: J. Neurosurg. 20:137 (Feb.) 1963. **8.** Strasser, N. F., *et al.*: Radiology 79:408 (Sept.) 1962. **9.** Doehner, G. A., and Brugger, G. E.: New York J. Med. 60:4022 (Dec.) 1960.

**Contraindication**

A history of sensitivity to iodine per se or to other contrast media is not an absolute contraindication to the use of meglumine diatrizoate.

**Precautions and Side Effects**

Severe, life-threatening reactions are rare; when they occur they suggest hypersensitivity. A personal or family history of asthma or allergy warrants special attention and may predict, more accurately than pretesting, the likelihood of a reaction, although not the type nor severity of the reaction in the individual.

The value of any pretest is questionable. The pretest most performed is the slow injection of 0.5-1.0 cc. of the preparation into a peripheral vein. An impending reaction is often indicated by transient burning and flushing, pain, "jump-like" reactions, respiratory difficulty, faintness, sneezing, itching, nausea, vomiting or urticaria. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. Antiallergic drugs may be used to advantage. In a few cases, the reactions to the test dose have been extremely severe.

The more serious anaphylactoid reaction requires immediate treatment and may occur despite a negative sensitivity test. An emergency tray consisting of vasopressor drugs, epinephrine hydrochloride 1:1000, methoxamine (Vasoxyl) or metaraminol bitartrate (Aramine), and glucose and saline is recommended. Oxygen and instruments to guarantee a clear airway must be readily available.

Caution must be exercised, especially in cerebral angiography in extreme age, in severely debilitated patients and in those with marked or severe hypertension, advanced arteriosclerosis, cardiac decompensation, recent cerebral embolism, or thrombosis, chronic pulmonary emphysema and in cyanotic infants.

For full details the Package Insert should be read.

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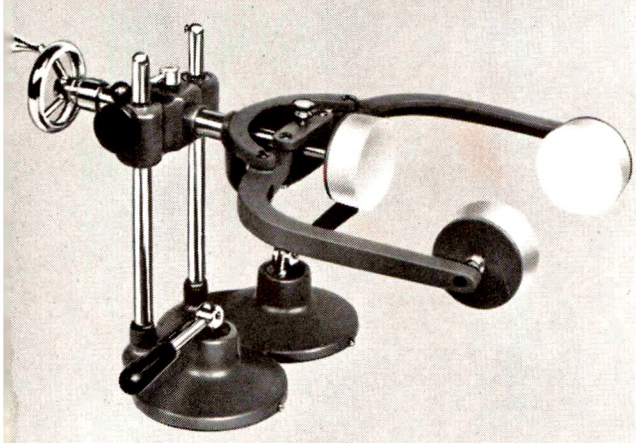
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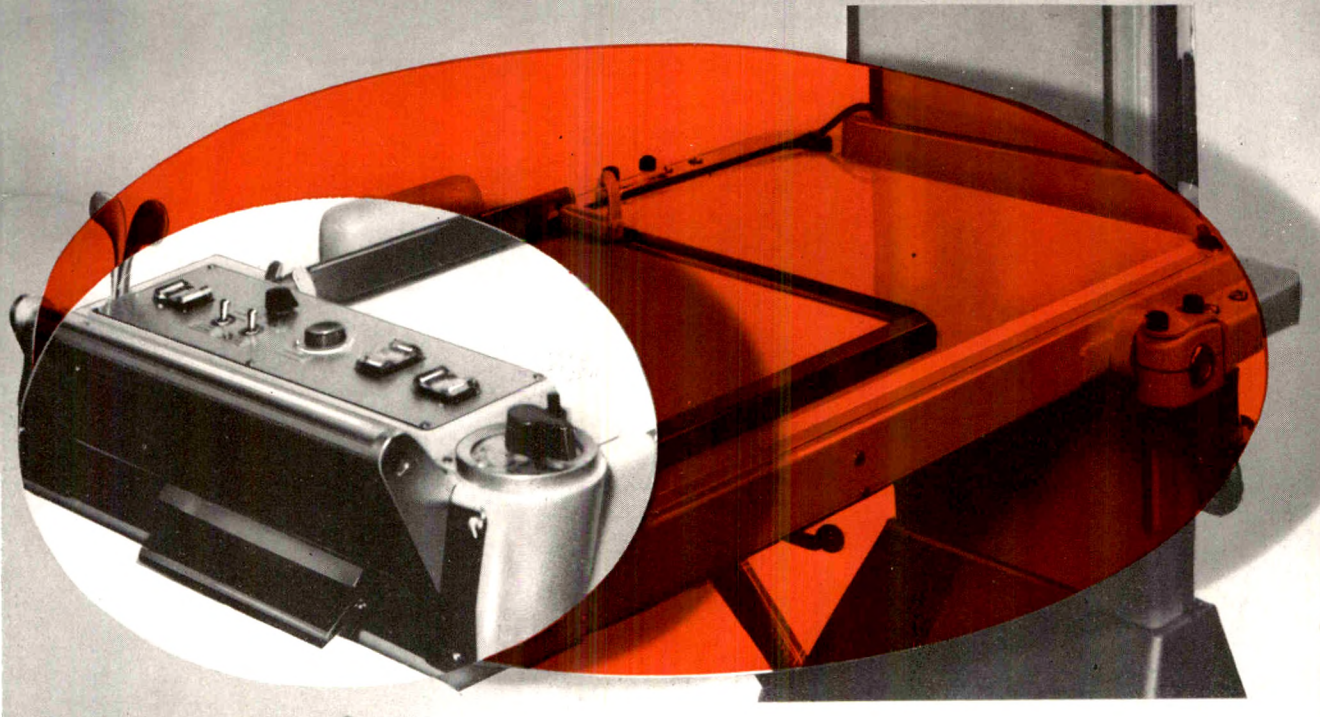
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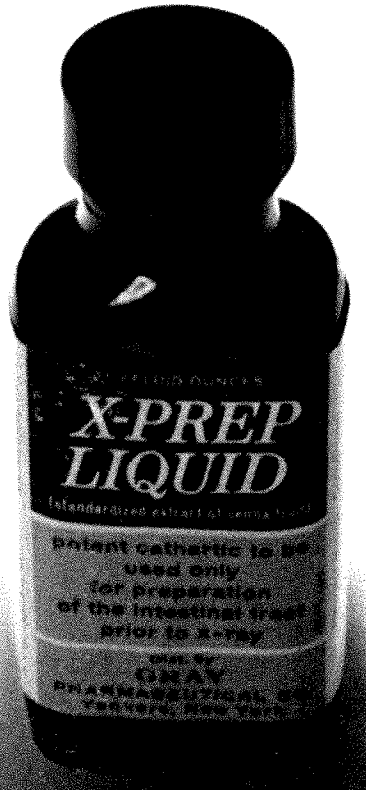
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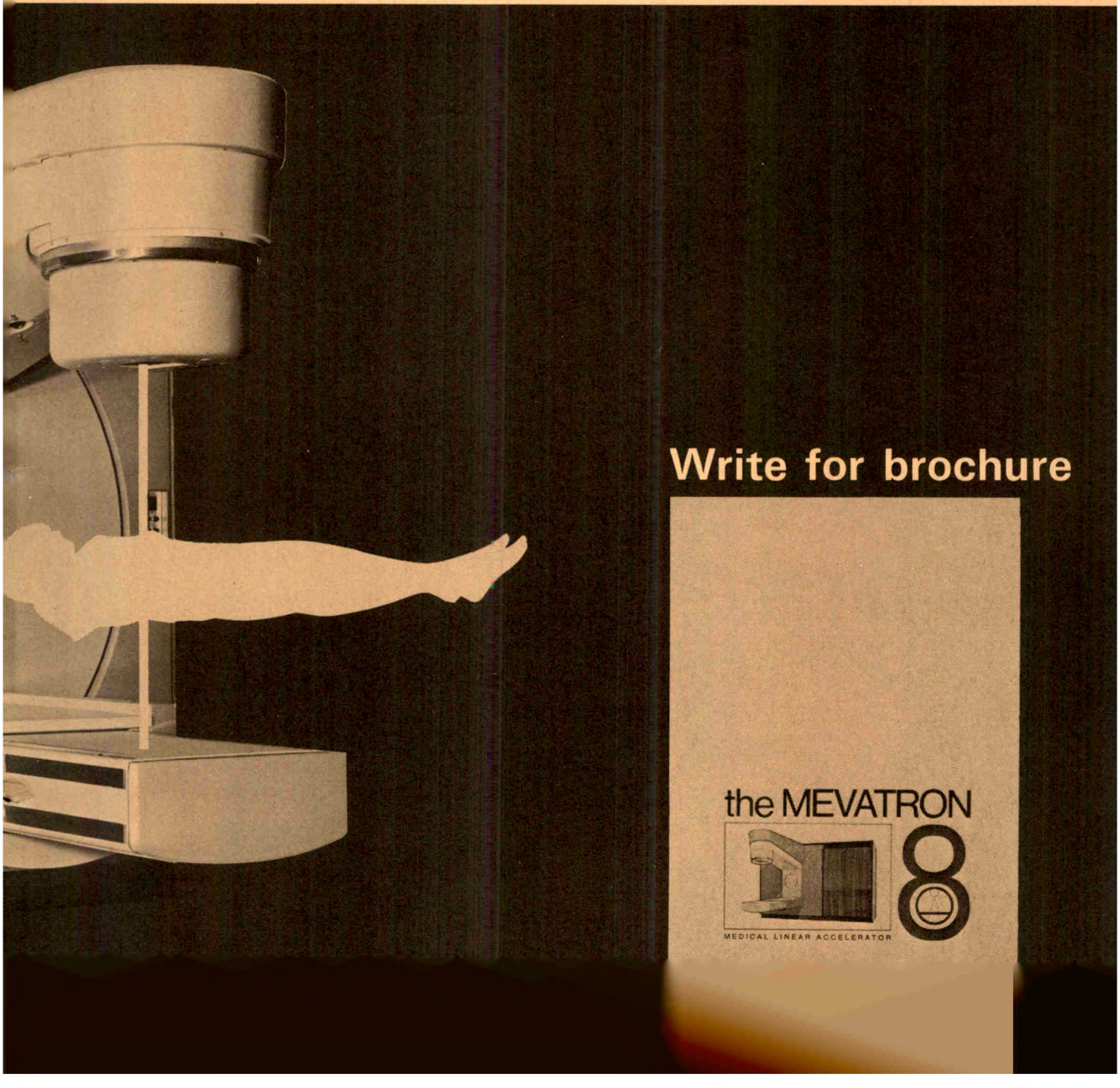
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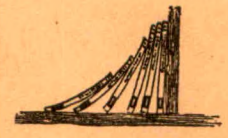
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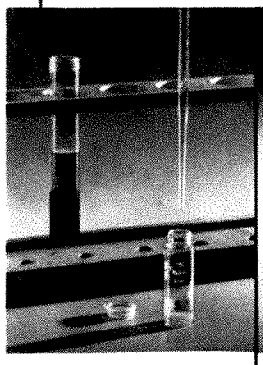
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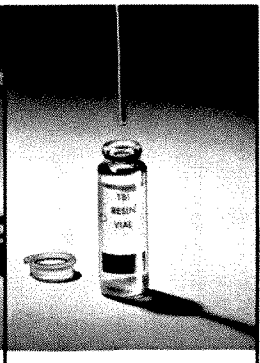
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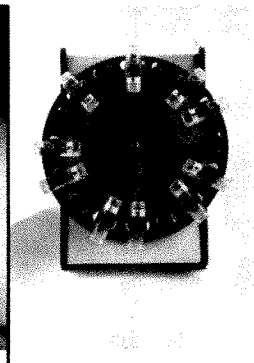
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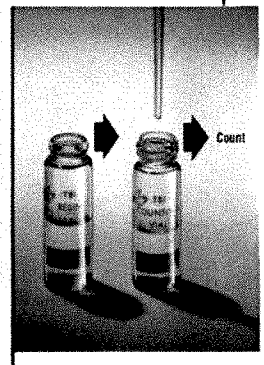
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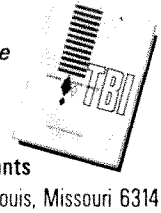
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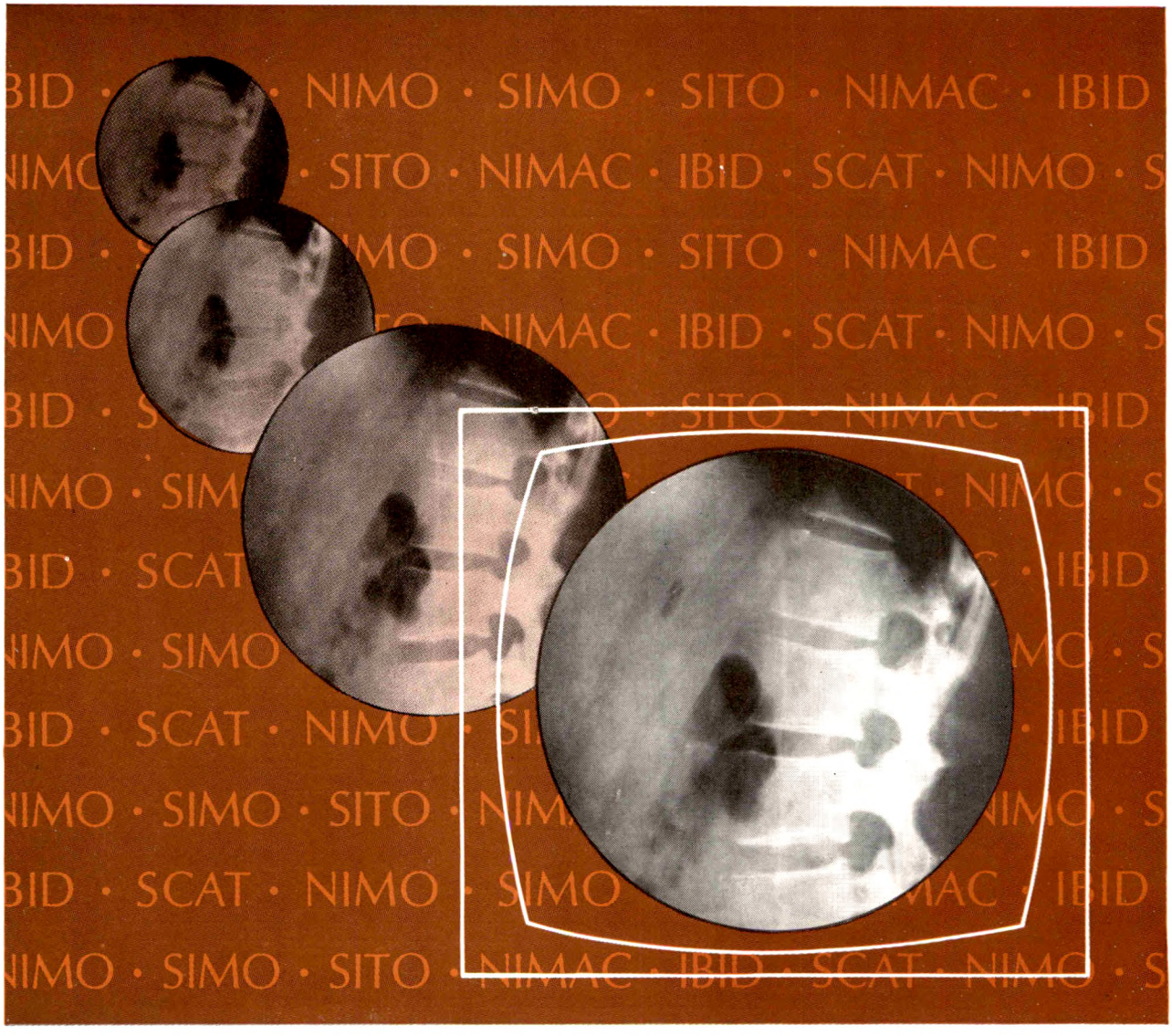
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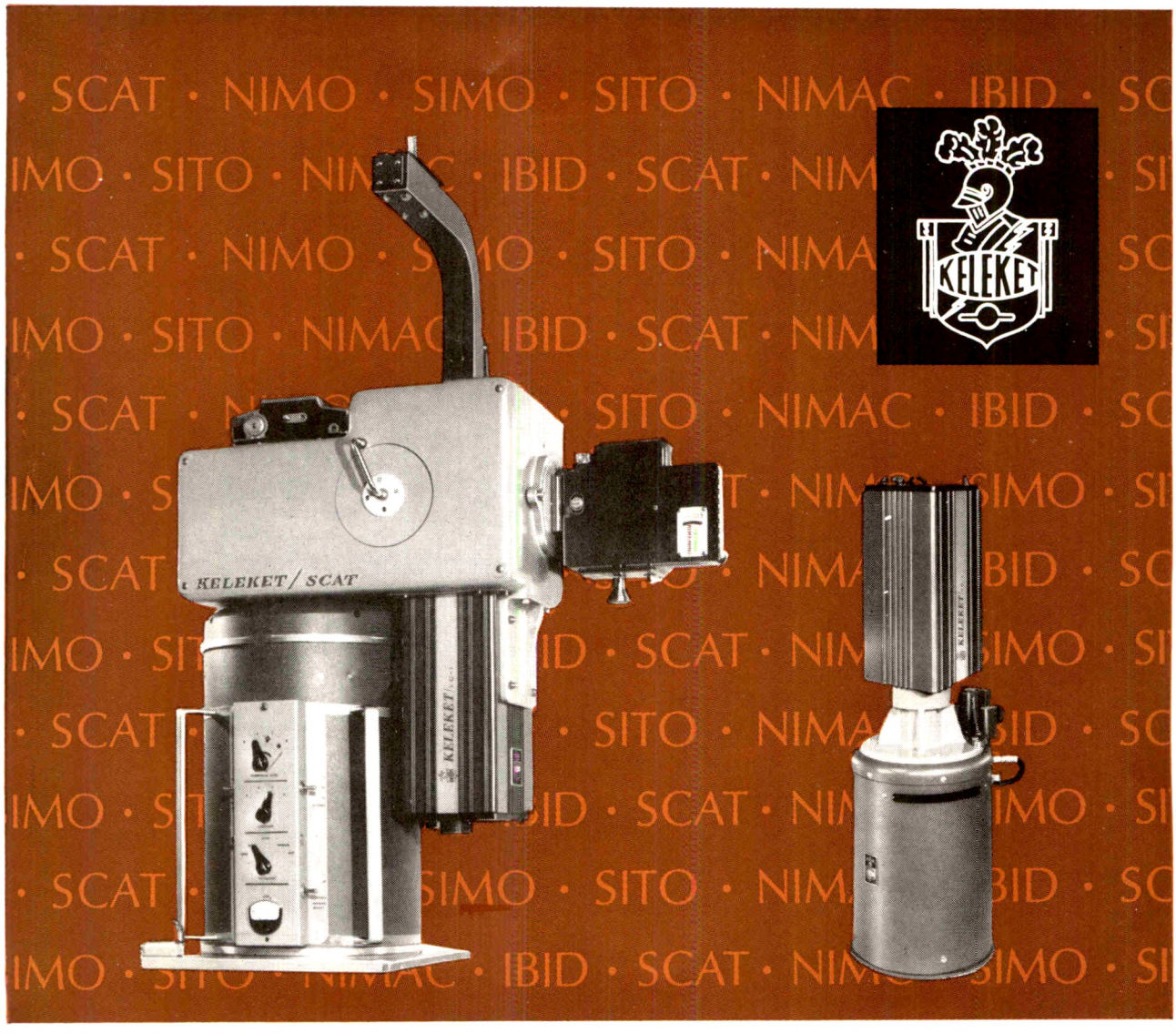
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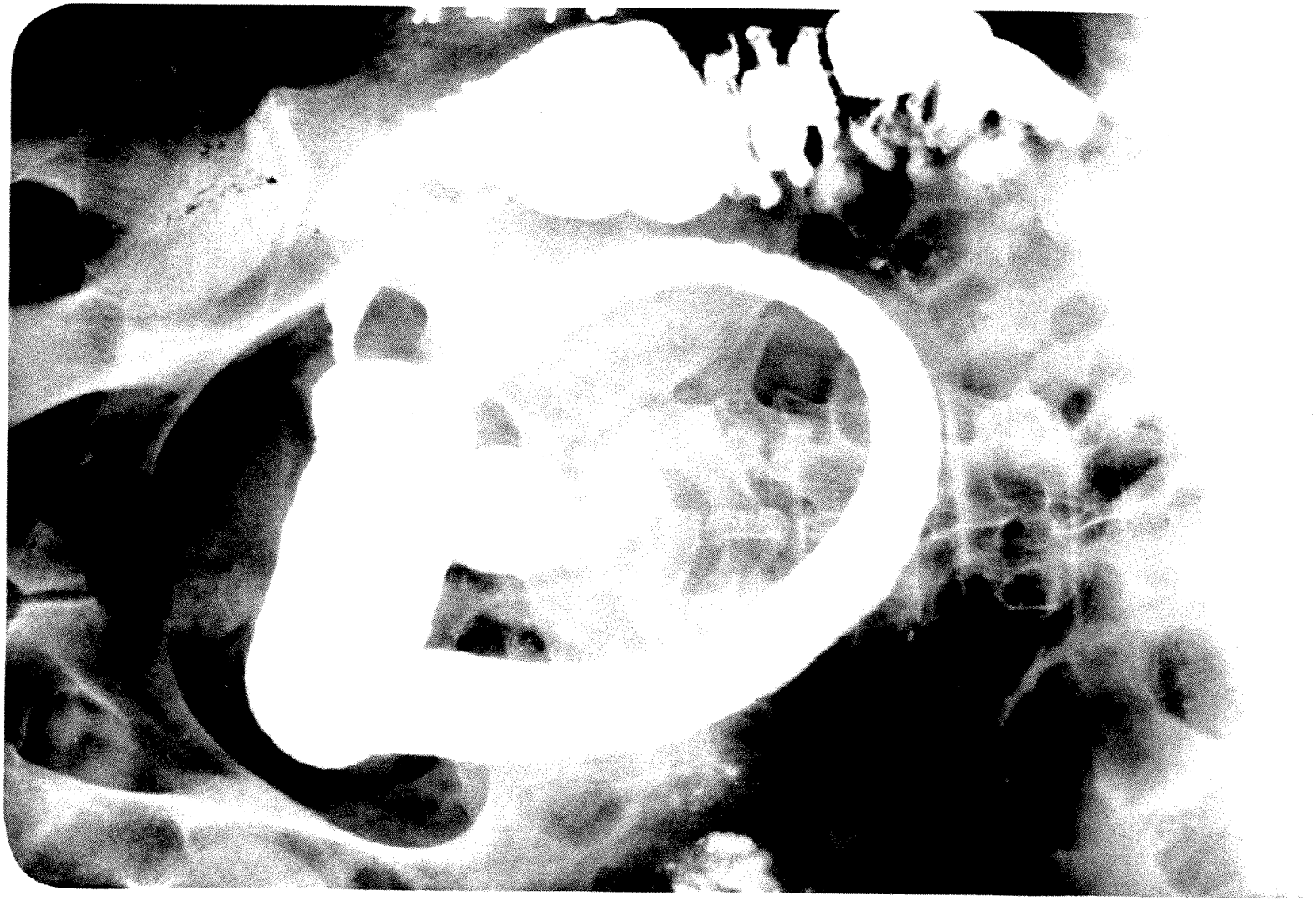
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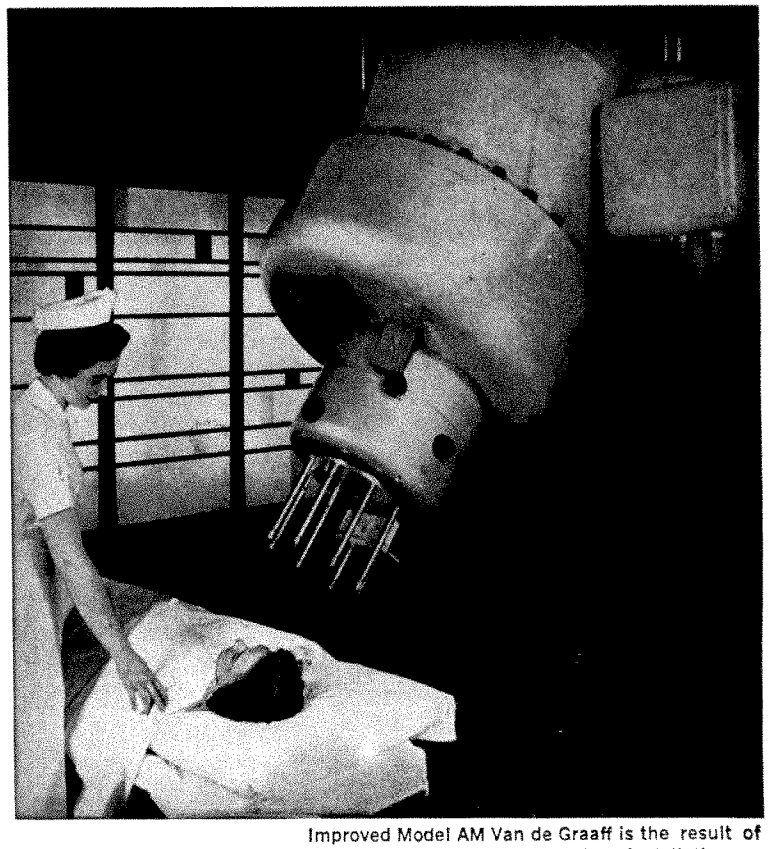
Upon admission, the patient's abdomen was soft with mild umbilical tenderness; bowel sounds were hyperactive. Stool was normal in color and consistency, however the patient developed diarrhea on the second day.

Radiography of the esophagus, stomach and colon disclosed no abnormality. However, a radiographic Small Bowel Study revealed rigidity of a large segment of the ileum. There was gross alteration of the mucosal pattern and the borders were serrated. The terminal ileum was markedly narrowed, giving rise to the "string sign." The findings were those of regional ileitis with multiple involvement.

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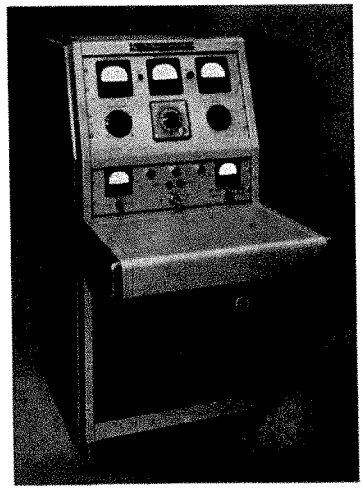


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ALEXANDER R. MARGULIS
Hickey Lecturer, 1967

THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 101 OCTOBER, 1967 NO. 2

# SOME NEW APPROACHES TO THE EXAMINATION OF THE GASTROINTESTINAL TRACT*

HICKEY LECTURE, 1967

By ALEXANDER R. MARGULIS, M.D.

*It is a great honor and privilege to be invited to give this oration commemorating Professor Preston M. Hickey. I did not have the honor of knowing Dr. Hickey personally, but his portrait and memory dominated the Department of Radiology at the University of Michigan during my training. That outstanding department was conceived and designed by this great man. From the first year of my residency I have been privileged to join the yearly pilgrimage to Detroit to attend the Hickey Lecture and listen to distinguished speakers giving this oration.*

*Preston M. Hickey was one of the pioneers of American Radiology who helped establish it as a medical specialty and as a full-fledged academic discipline. Dr. Augustus W. Crane,[17] another pioneer of American Radiology, summarized Dr. Hickey's impact in an obituary: "He took up the roentgen ray when it was a scientific curiosity and laid it down as one of the foundations of modern medicine."*

*Dr. Hickey exemplified the true spirit of the renaissance and his various talents, wide interests, and knowledge in many fields produced the qualities for a great teacher, a prolific writer, and a leader of other physicians. As Professor of Radiology, first at the Detroit College of Medicine and Surgery, the present Wayne State University College of Medicine from 1909 to 1922, and then as Head of the Department of Radiology at the University of Michigan from 1922 until his death in 1930, he laid down the functions and responsibilities of a modern radiology department. He organized clinical conferences and convinced those in other fields of medicine of the important place of radiology. He inspired his fellow radiologists to consider themselves members of a clinical team and to regard radiology as a diagnostic specialty intimately related to other clinical disciplines. He taught them to interpret their studies only in the light of clinical data and to correlate them to surgical and necropsy observations.*

*As the Founder and first Editor of the* AMERICAN QUARTERLY OF ROENTGENOLOGY, *the predecessor of the* AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM

* Presented at the Meeting of the Wayne County Medical Society, The Wayne State University College of Medicine, and the Detroit Roentgen Ray Society, Detroit, Michigan, March 2, 1967.

From the Department of Radiology, University of California, San Francisco Medical Center, San Francisco, California.

THERAPY AND NUCLEAR MEDICINE, *he laid the foundation for an internationally respected publication.*

*Dr. Hickey was the recipient of almost every honor that American Radiology could bestow upon an individual. His investigative interests, as of most other leaders of radiology of that date, were general. He wrote of the chest, the skeletal system, and the gastrointestinal tract, continuously striving for new approaches.*

*To honor the memory of this visionary pioneer, this oration will be devoted to some new approaches to the radiologic examination, particularly those of the gastrointestinal tract.*

THE examination of the gastrointestinal tract has never been stagnant since Professor Walter B. Cannon at Harvard Medical School fed bismuth to a cat. New developments and new approaches have followed in rapid succession. A few milestones that have denoted progress over the years were: the introduction of barium sulfate to replace other media for examination of the intraluminal gut; the barium enema examination; cholecystography; intravenous cholangiography; splenoportography; technical developments such as the tilting fluoroscopic table, the spot-film device, the image intensifier, cineradiography, television, videotaping, and many others. This procession succeeded in adding fresh approaches and widening the scope and direction in which the advances were and are occurring.

Available to the modern radiologist is a vast armamentarium for the examination of the gastrointestinal tract. The field is still in flux, with new advances along many lines occurring practically every day. Because of limitation of time, this lecture cannot encompass all the new approaches in the field of gastrointestinal radiology. I have tried to limit it to the approaches that are examples of entire areas of endeavors. Some that are as important and imaginative are not included.[26]

## I. PHARMACOLOGIC AGENTS IN THE EXAMINATION OF THE GASTROINTESTINAL TRACT

Alteration of the physiology of the gastrointestinal tract by drugs to emphasize normal and abnormal patterns is a promising field in roentgenology. The use of mecholyl in the diagnosis of achalasia and of cholecystokinin in the study of the gallbladder are examples of efforts along this path. Bradykinin and adrenalin in the study of intestinal arteries[8,12] and atropine and probanthine for atonic duodenography are others.[31]

As an example of this type of approach, the use of neostigmine in the examination of the small bowel will be discussed here. This drug is a reversible inhibitor of acetyl cholinesterase. By prolonging the effect of endogenous acetylcholin, the normal small bowel contracts, motility increases, and propulsion of the barium meal is speeded. The mucosal pattern of the ileum then resembles that of the jejunum (Fig. 1, *A* and *B*).

The drug is effective when neuromotor function of the small bowel is normal.[24,42] When the neuromotor mechanism of peristalsis is disturbed, as in sprue, it is ineffective[29,30] (Fig. 2*B*). Neostigmine thus effectively potentiates differences between normal and abnormal patterns of the small bowel. It increases the tone of the small bowel, resulting in narrowing of the lumen.[21] Thus, overlapping of multiple loops is reduced and a better study of individual loops is possible (Fig. 2*A*). Details of the valvulae conniventes and their orientation become more apparent, and fistulae, sinuses, and sharp kinks are seen more easily. Neostigmine also permits incorporation of the examination of the small bowel as an integral part of that of the upper gastrointestinal tract. This examination becomes feasible because the drug speeds filling of the small bowel. More frequent examinations of the small bowel will often reveal unsuspected pathologic conditions that involve this bowel. The dose of neostigmine




Fig. 1. Normal, small bowel pattern obtained in 2 patients after injection of neostigmine. (*A*) Fifteen-minute roentgenogram shows an excellent small bowel pattern. (*B*) Fifteen-minute roentgenogram obtained in another patient, also after injection of neostigmine. (Reproduced with permission of *Radiology*.[42])

recommended is 0.50 mg. for adults and the same for children 2 years or older. For children younger than 2 years, it is 0.25 mg. and for adults weighing more than 190 pounds, 0.75 mg.[22]

Remarkably few conditions contraindicate the use of neostigmine in this dose. Some of these are intussusception, volvulus, complete intestinal obstruction, perforation, peritonitis, and recent myocardial infarction.[24,42] Patients with low blood pressure of long duration should lie down while waiting so as to avoid syncope. Many thousands of small bowel examinations in adults and children have been performed without difficulty after neostigmine had been given.[22,44] Other methods are being applied to speed action of the small bowel but these do not significantly alter the pattern of the small bowel. These include the administration of ice saline with barium[62] and cleansing of the large bowel with the patient in the right decubitus position.[48]

### II. Addition of Substrates to Barium Sulfate to Obtain Abnormal Diagnostic Patterns on Roentgen Examination of the Gastrointestinal Tract

Food has been added to the barium meal for the study of functional abnormalities of gastrojejunostomies.[45] Similarly, abnormal patterns may be produced by adding allergens to barium.[23]

An example of this approach is the production of abnormal patterns in the small bowel and colon in lactase insufficiency. To produce these patterns the small bowel is deliberately challenged by the addition of lactose to barium sulfate administered orally.

Disaccharidase insufficiency, producing diarrhea, has been described in children

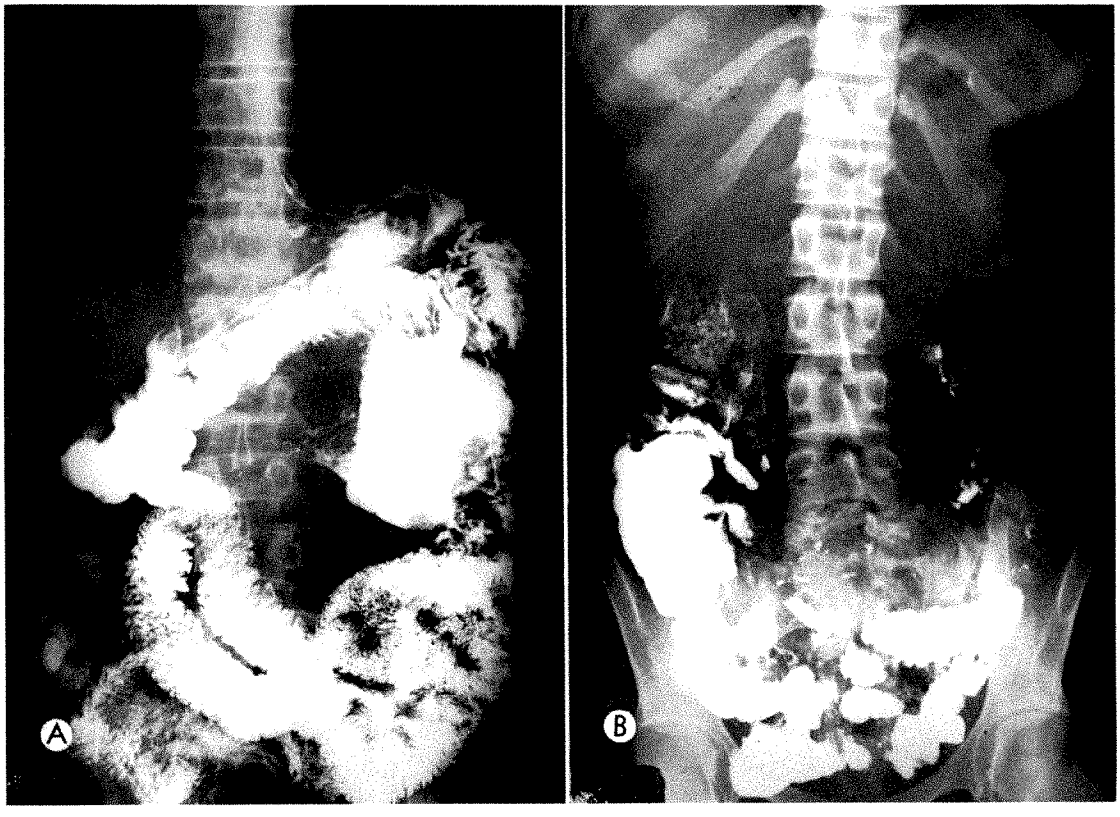



Fig. 2. (*A*) Demonstration of lymphosarcoma of the jejunum after administration of neostigmine. A large mass with a large central cavity displaces the small bowel. (*B*) Two hour roentgenogram in a patient afflicted with nontropical sprue. Note that the small bowel pattern is not affected by neostigmine. (Reproduced with permission of *Radiology*.[42])

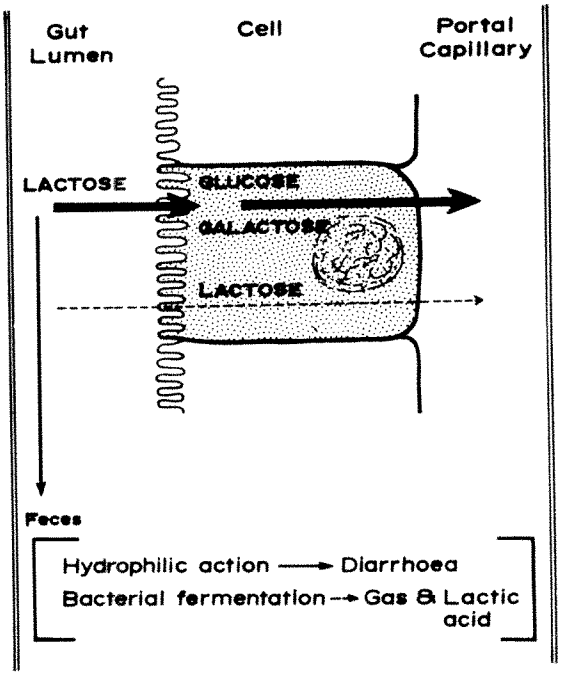
and adults.[18,33,56,60] In the past, this abnormality was diagnosed by producing symptoms with food to which lactose had been added. The glucose blood levels after ingestion of lactose and the assay of enzymes of the intestinal mucosa after biopsy also contributed to the diagnosis. Laws and Neale,[37] Haemmerli and Kistler,[25] and more recently Preger and Amberg,[54] have described roentgenologic methods for the diagnosis of this enzyme insufficiency.

Lactase insufficiency is probably the most common abnormality of the small bowel in man, the only known mammal normally retaining lactase in the small bowel after weaning.[56] The enzyme, which is in the brush border of the intestinal epithelial cell, hydrolizes the disaccharide into glucose and galactose, which are then passed across the cell into the portal circulation (Fig. 3).

The small-bowel symptoms are produced by the osmotic effect of the undigested lactose, which acts as a hypertonic hydrophylic substance.[33] Gas and lactic acid produced by coliform bacteria also cause such symptoms. The increased fluid and gas in the small bowel are believed to be the reasons for the cramps, diarrhea, accelerated transit, and flatulence.

A characteristic roentgen appearance is obtained in patients with lactase insufficiency when lactose has been added to the barium sulfate meal. It shows distention of the lower small bowel and of the large bowel, dilution of barium, and accelerated transit through the bowel (Fig. 4, *A* and *B*; and 5, *A* and *B*). Preger and Amberg[54] correlated these roentgen changes with a flat glucose absorption curve. When a flat lactose curve, rapid transit, dilution and dilatation were present, these patients in-

FIG. 3. Diagram showing mechanism of disaccharide hydrolysis and absorption.




variably experienced cramps, diarrhea, and flatulence during the examination. Preger and Amberg were able to show that in some of their controls, challenged with 100 gm. lactose, the symptoms and roentgen appearance were those of lactase insufficiency. They further showed flat absorption curves and the roentgen appearance of this condition in many other patients. In these, diarrhea or primary gastrointestinal disorders, such as regional ileitis, had been present, or a previous, subtotal gastric resection had been done.

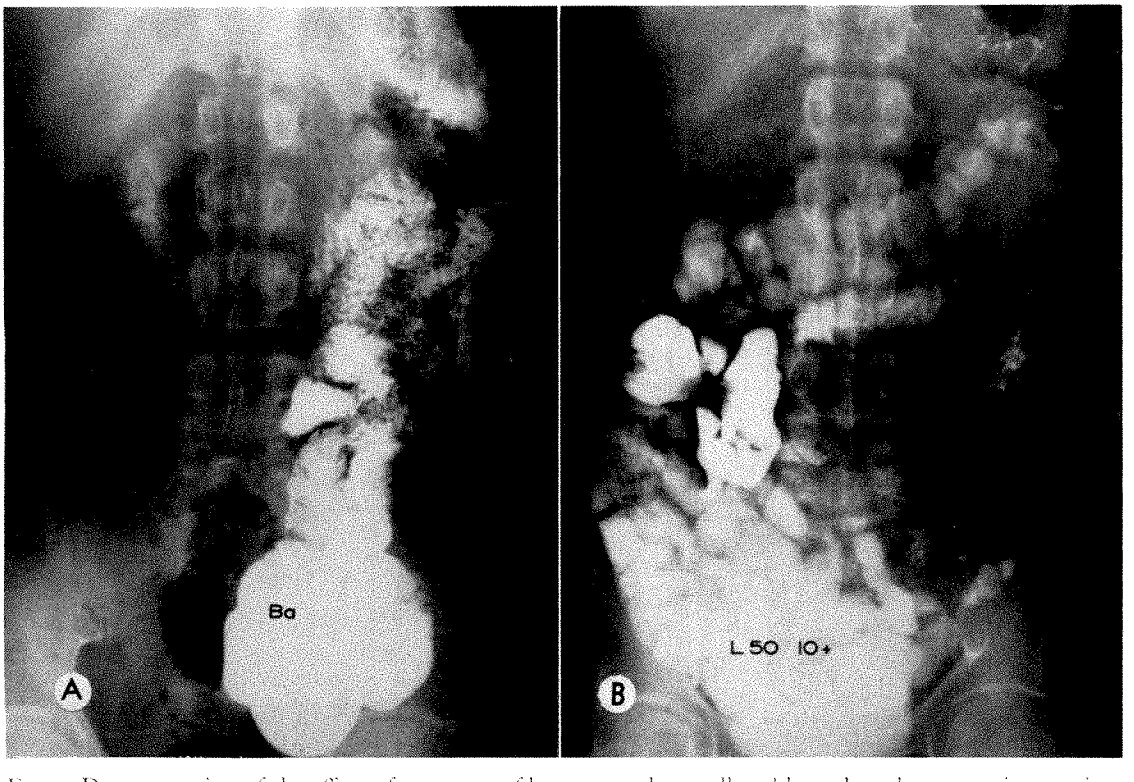



FIG. 4. Demonstration of the effect of 50 grams of lactose on the small and large bowel patterns in a patient with lactase insufficiency. Patient had had a partial gastrectomy (Bilroth II type). (*A*) No abnormalities are seen when lactose was not added to the barium sulfate mixture. (*B*) After addition of 50 grams of lactose to barium the blood glucose level rose 10 mg. per cent. The barium in the ileum is diluted and a dilatation of the distal small bowel was accompanied by cramps and gas. Notice the diluted barium in the colon.

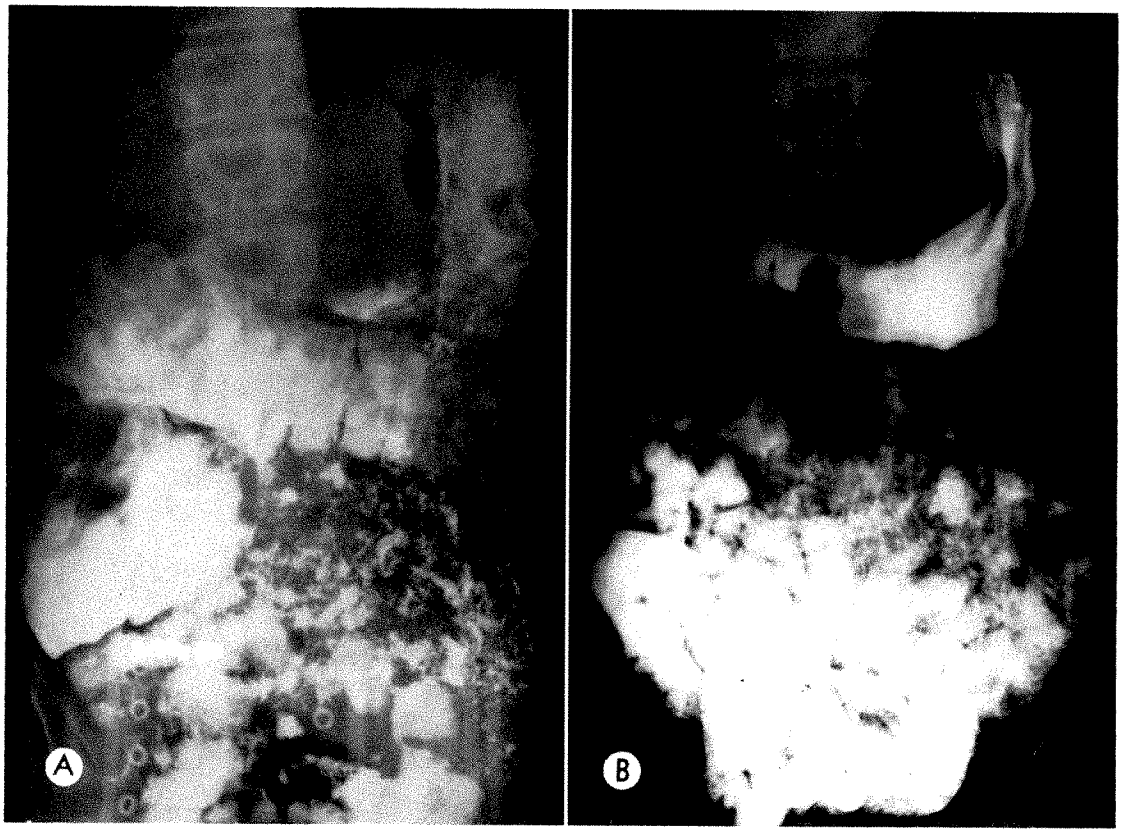



FIG. 5. Effect on the small bowel pattern in a patient with lactase insufficiency when lactose was added to barium. The patient was afflicted with nontropical sprue. Omission of gluten from the diet relieved the symptoms of sprue. The addition of lactose to food, however, reproduced the symptoms. (*A*) After addition of 50 grams of lactose to barium, the blood glucose level did not rise. The small bowel pattern is abnormal. Note the dilution of barium in the terminal ileum and colon. (*B*) After the addition of 50 gm. of sucrose to the barium the blood glucose level rose 34 mg. per cent. Normal roentgen appearance and no diarrhea. (Courtesy of Preger and Amberg.[54])

### III. INDUCTION OF CHANGES IN BLOOD VOLUME TO FACILITATE EXAMINATION OF THE GASTROINTESTINAL TRACT

A new method for facilitating the visualization of esophageal varices has been developed by Preger.[53] Boyer *et al.*[10] reported that in patients with cirrhosis of the liver, the rapid infusion of dextran resulted in a temporary elevation of portal venous pressure. At the same time, the rise of systemic venous pressure was much lower. Preger stated that this temporary gradient between the two circulations is helpful in demonstrating varices in barium esophagrams. He selected patients with a history of gastrointestinal bleeding and known hepatic cirrhosis, but without ascites. Patients with ascites were excluded from Preger's series because in such patients the varices are prone to rupture when the blood volume is rapidly expanded.[19,63] Esophagrams performed in the conventional manner gave negative results. Successful demonstration of varices by roentgenographic methods in the past has been reported to range from 20 per cent[11] to 70 per cent.[34] Infusions of 1,000 ml. of 6 per cent dextran in saline were administered to Preger's patients in 30 to 40 minutes. Roentgenograms following this infusion revealed esophageal varices in half the patients with previously negative results. Two blind readings by each of 8 radiologists confirmed their presence (Fig. 6, *A* and *B*).

### IV. PHYSIOLOGIC DATA RECORDED SIMULTANEOUSLY WITH CINERADIOGRAPHY

Cineradiography continues to be important in the study of the gastrointestinal tract. It permits the study of motility, especially of the pharynx, the esophagus, the duodenum, and small bowel, as well as the study of colon evacuation. Cineradiography records events that are also observed on fluoroscopy, such as of gastrointestinal peristalsis and the detailed flexibility of the gut wall.

The simultaneous presentation of cineradiographic and physiologic data has recently been perfected by E. R. Miller and his team.[20,61] Two television cameras are used, one viewing the roentgenographic image from the intensifier output phosphor and the other viewing the intraluminal pressure recordings. A motion picture camera equipped for sound, records the simultaneous roentgenographic and pressure recordings from the television screen on which all data are displayed (Fig. 7). Thus, sound films, containing the entire physiologic and cineradiographic information may be obtained. The sound is of great importance in timing the occurrences and recording commands and patient response, resulting in better understanding of the film. Particular sounds, such as those of straining and coughing, and bubbling in a suction apparatus calibrated for a particular pressure, further emphasize the event.

This method is of great value and offers vast opportunities in the study of hiatus hernia, varices, defecation, and abnormalities of the colon, such as in Hirschsprung's disease.

Although this method of recording contributes greatly to the study of the gastrointestinal tract, it is also being applied in the genitourinary tract.[61]

### V. NEW APPROACHES TO THE DIAGNOSIS OF DISEASE OF THE COLON

The diagnosis of the colon has depended primarily on the conventional and double contrast barium enema examination. By and large, these methods still remain the main avenue of investigation, but newer approaches have been developed.[39] The following are some examples.

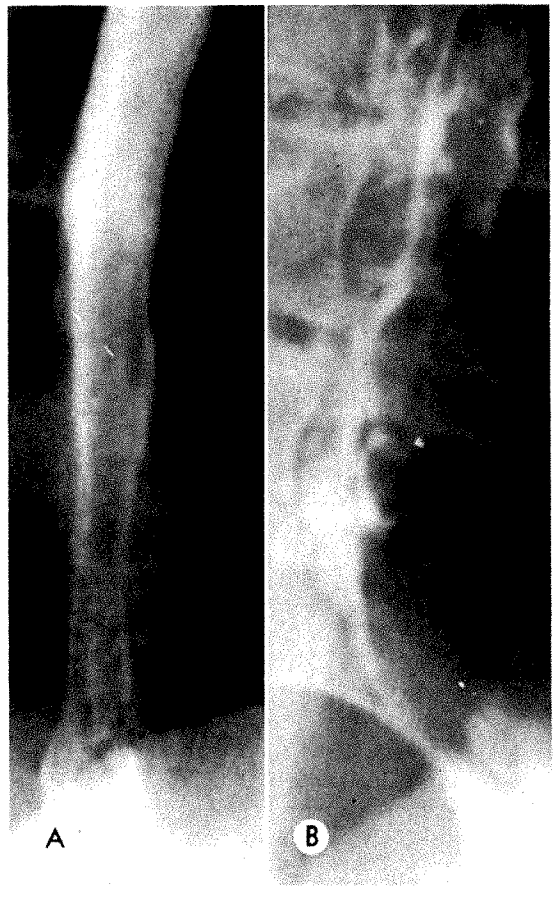



FIG. 6. Effect of dextran on varices in a cirrhotic patient. (*A*) Equivocal evidence of varices before infusion of dextran. (*B*) The same patient, 30 minutes later. After dextran infusion, very prominent varices are seen.

#### A. THE SILICONE FOAM ENEMA

Cook and Margulis[15,16] have reported a method by which the sigmoid and lower portion of the left colon may be examined by the introduction of a prepolymeric mixture of silicone liquid made radiopaque. A catalyst (stannous octoate), which is part of the mixture, promotes polymerization at body temperature within the bowel. The mold that is formed is soft and compressible, allowing an imprint of the colonic

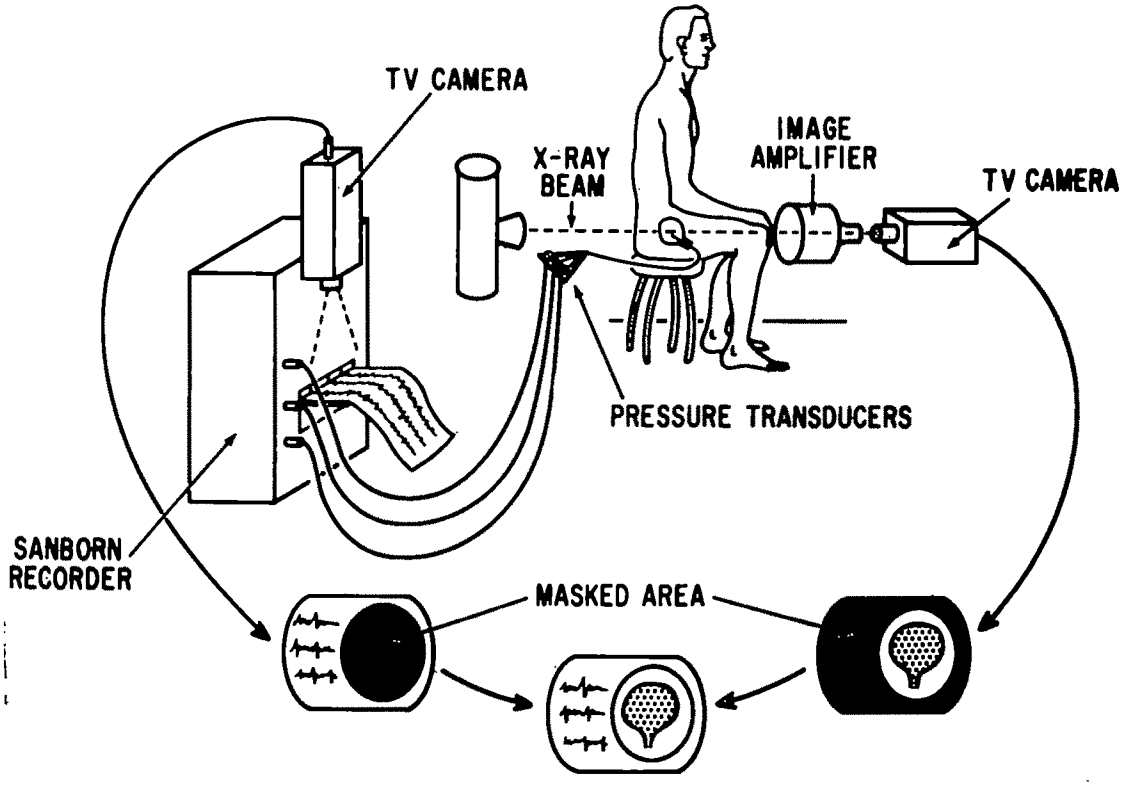



FIG. 7. The multiple recording system used by Dr. Earl R. Miller, which incorporates cineradiographic and physiologic data. The x-ray beam penetrates the patient and enters the image amplifier. The television orthocon camera presents this image on the right side of the monitor. In this illustration, the catheter is in the bladder. The output of the transducers is recorded on a Sanborn 8-channel recorder. A vidicon television camera views the data from the graph and presents it on the left side of the television screen. The television signals are then combined and presented as physiologic data to the left and the x-ray data to the right. A kinescopic and video tape recording is made with a movie sound camera and video recorder simultaneously. The sound track is always included as part of the recording. (Reproduced with permission of *Surg., Gynec. & Obst.*[20])

mucosa with which it is in contact. Cells, especially from lesions, adhere to the surface of the mold that is sufficiently soft to be expelled. A balloon of the Bardex type is placed in the rectum to facilitate expulsion and reduce the amount of silicone required in the area readily accessible to the examining finger and the proctoscope (Fig. 8).

This type of examination has proved to be especially promising in the diagnosis of neoplasms (Fig. 9, *A* and *B*), as well as in the diagnosis of polyps. The cytologic examination of washings obtained from the molds, allows selectivity and is of relatively high accuracy.[58] The radiopacity of the compound helps to control its administration under fluoroscopic vision. The compound is not allowed to pass beyond any obstructive lesions. After the mold is partly expelled, a roentgenographic record is obtained to see the amount of residue. This type of examination is most indicated as a supplementary investigation after barium enema examination reveals a suspicious lesion beyond the reach of the sigmoidoscope (Fig. 10, *A* and *B*). The patient must remain under observation until evacuation of the mold is certain and no complications are expected.

Amberg[1] recently reported a fatality associated with the use of the silicone foam diagnostic enema. An obstruction resulted from the passage of the mold proximally to

a postirradiation stenosis. The patient did not return to the physician until after perforation.

Habitual users of laxatives or of enemas frequently experience difficulty in evacuating the mold. This difficulty does not present a serious problem, however, as the rectoampullary portion of the mold can be broken with a gloved finger. The expelled mold is then reassembled along its broken edges.

### B. ROENTGENOGRAPHIC DENSITY IN THE DIAGNOSIS OF COLONIC LIPOMAS

On the usual barium examination the density of a lipoma and of a soft tissue tumor cannot be relied upon in the differential diagnosis of these lesions. The reasons are twofold: (1) in conventional barium enema examinations high kilovoltage techniques are used; (2) the coating of lesions by barium prevents differentiation of densities.

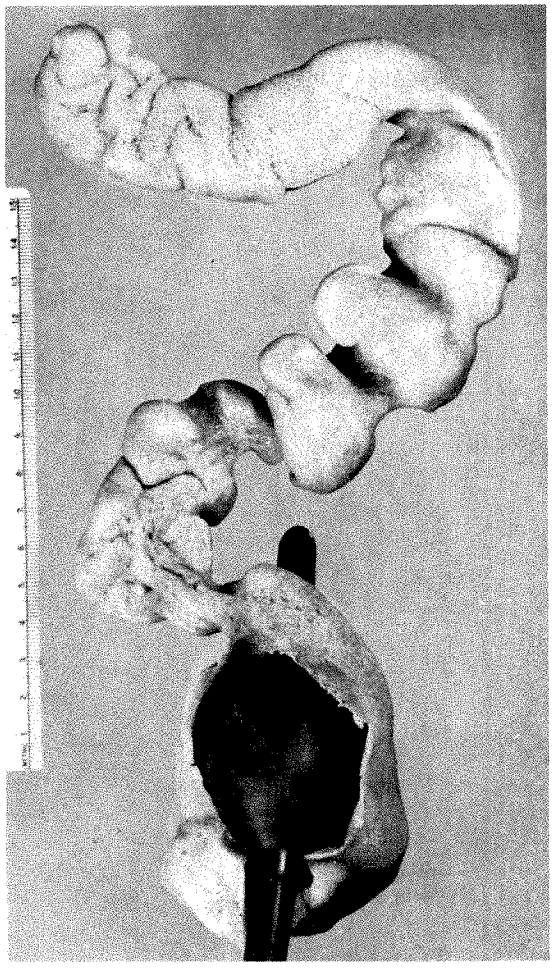


FIG. 8. A silicone-foam mold in a normal individual shows the rectal ampulla with a Bardex balloon in place and the rectum and sigmoid.

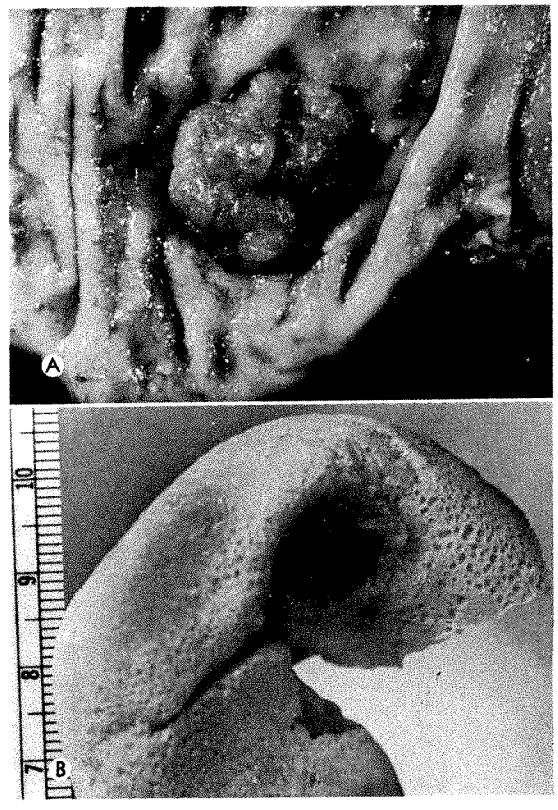
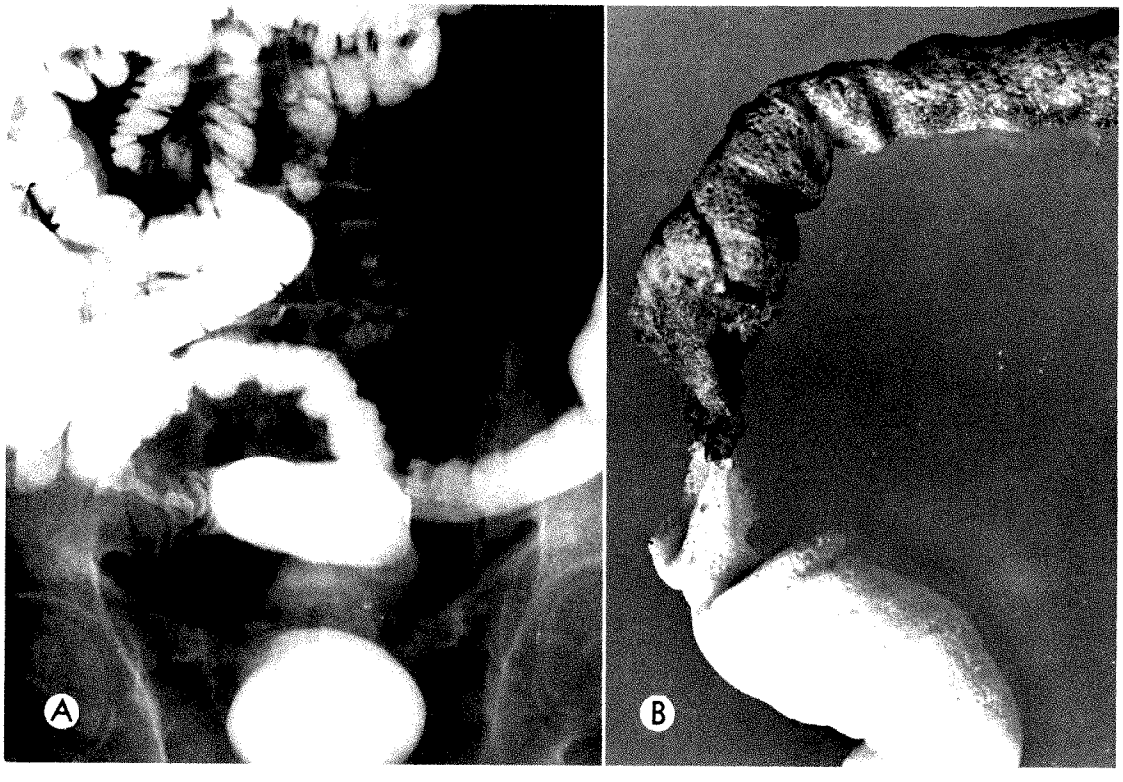



FIG. 9. Silicone-foam mold with imprint of a fungating polypoid carcinoma of the distal sigmoid in an elderly man. (*A*) The polyp after resection of the bowel. (*B*) Detailed imprint in the silicone mold. This lesion was seen on sigmoidoscopy but could not be seen on the conventional roentgenogram of a barium enema examination. (Cook and Margulis.[16])

With low kilovoltage,[57] however, if barium is not used and the colon is filled with water, a fatty tumor may appear as a radiolucent filling defect. Tumors of tissue density will not be distinguishable from the surrounding water (Fig. 11, *A-F*). To avoid confusion with air bubbles, a barium enema examination before such an examination is recommended (Fig. 12, *A* and *B*). The roentgen projection to establish location should be identical to the one that


A
B

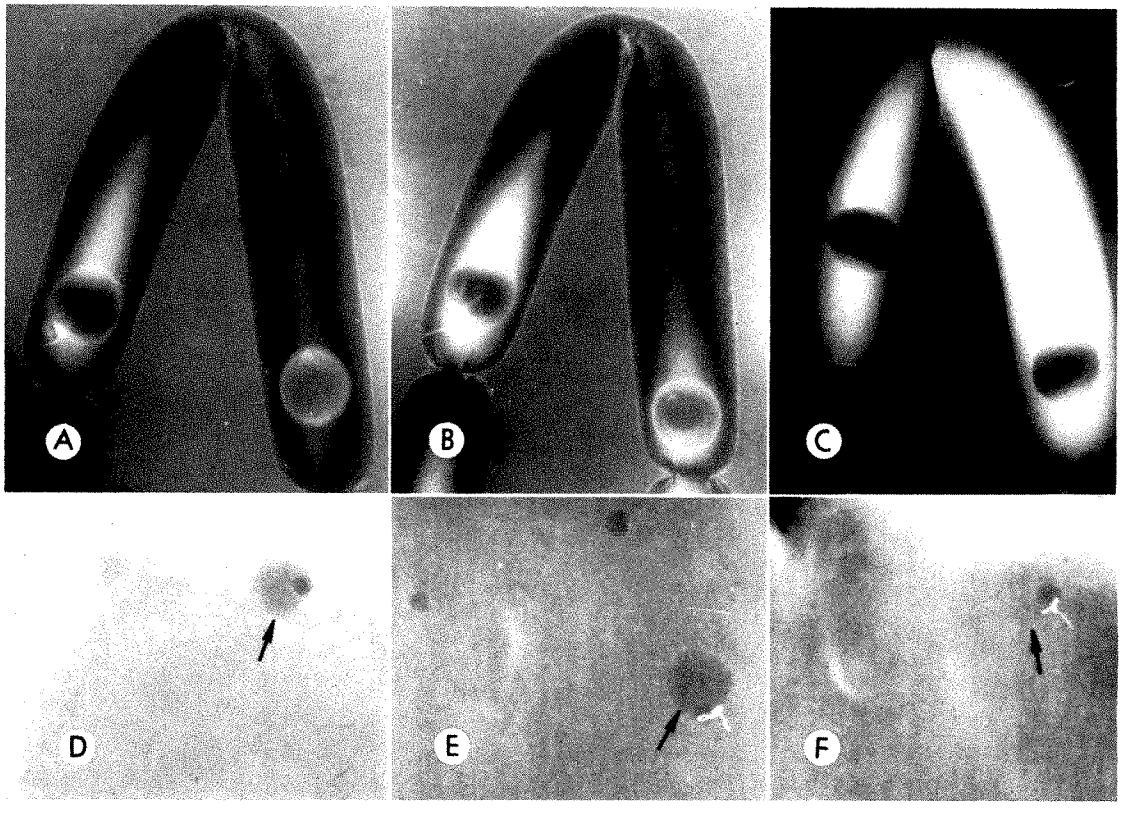

A
B
C
D
E
F

FIG. 10. (*A*) Pre-evacuation roentgenogram of a conventional barium enema examination shows a carcinoma of the sigmoid. (*B*) Silicone-foam mold shows the carcinoma in an elderly male. The distal part of the mold was evacuated by the patient 15 minutes after its administration. Malignant cells were washed off the distal part of the mold. The proximal mold, which was evacuated 16 hours later, is stained by fecal pigment. The 2 fragments of the mold could be fitted together easily. (Cook and Margulis.[16])

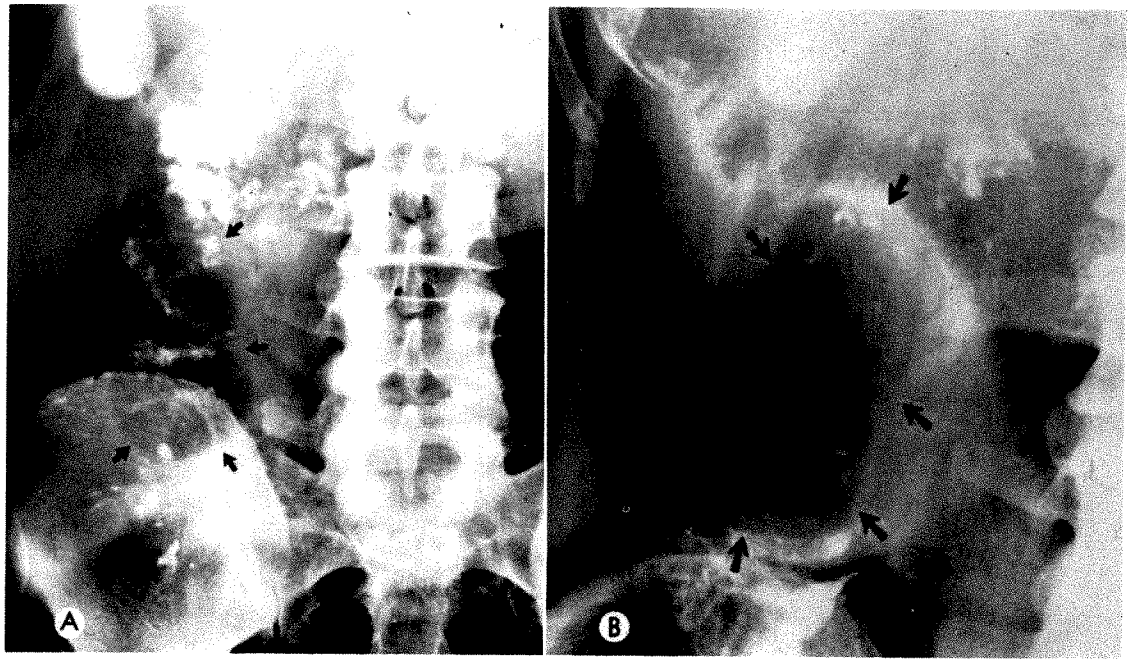



FIG. 12. Demonstration of a large lipoma of the right colon by means of a water enema. Small amounts of barium retained from a previous barium enema aided in the study. Roentgenograms were obtained at 60 kv. (*A*) Roentgenogram of the water-filled colon. (*B*) Detailed view of lipoma (arrows). (Courtesy of Stevens.[59])

FIG. 11. The model demonstrates the value of using low kilovoltage to enhance the difference in roentgenographic density between lipomas and tumors of water density. A plastic finger cot filled with fat is marked with a metallic tie and a water-filled finger cot is in the opposite arm of the plastic tubing. The tubing is tied at regular intervals to simulate haustral folds. It is suspended in a phantom consisting of a plastic container filled with 14 inches of water. The plastic cots and the tubing are coated with barium, then filled with water to simulate a double-contrast examination of the colon. Roentgenograms obtained at (*A*) 30 kv., (*B*) 60 kv., and (*C*) 90 kv. No appreciable difference is seen between radiodensity of the 2 models. When the tubing is filled with water and no barium is present, the lipoma model is seen clearly (arrows) at (*D*) 30 kv. and (*E*) 60 kv. exposures, respectively. (*F*) At 90 kv. the lipoma is barely seen (arrow). The water density model is not visible. (Margulis and Jovanovich.[41])

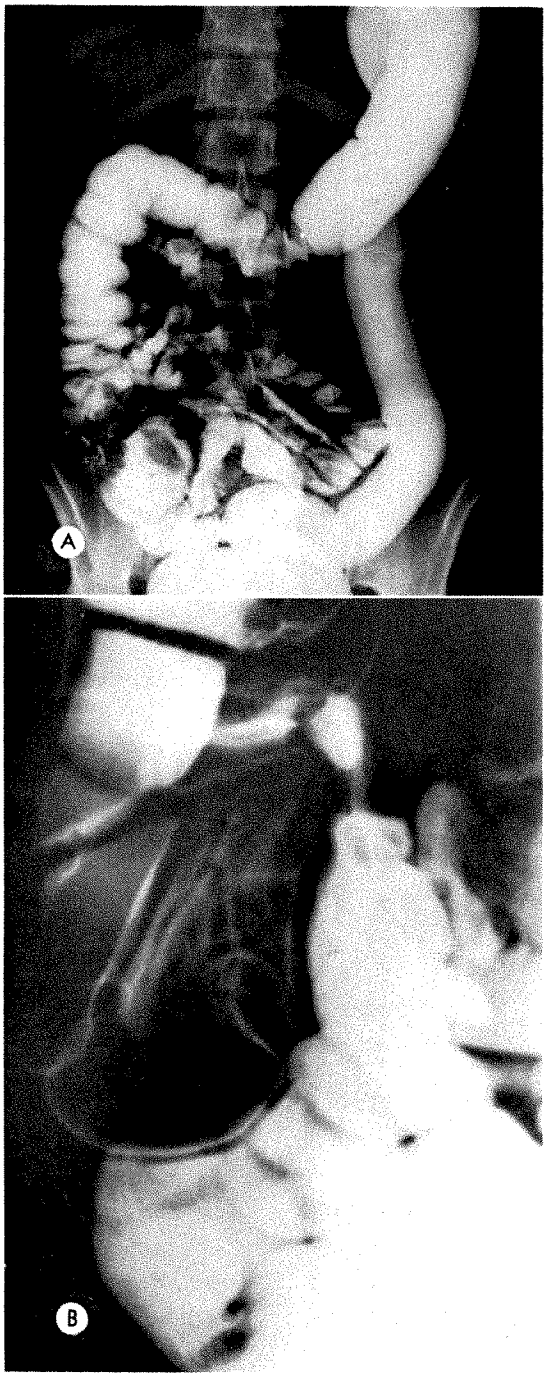



FIG. 13. The value of use of the peroral barium examination of the right colon and terminal ileum. (*A*) Pre-evacuation roentgenogram of a barium enema examination shows a large defect in the cecum, which was demonstrated in two previous examinations. (*B*) Peroral right pneumocolon shows the cecum. Mucosal detail is excellent. The cecum is normal.

best showed the defect in the barium enema examination.[41,59]

### C. PERORAL DOUBLE CONTRAST EXAMINATION OF THE RIGHT COLON

The right colon, the ileocecal valve, and the terminal ileum are difficult to examine by barium enema. Fecal material and difficulty in coating the right colon and the terminal ileum by the rectal route frequently account for inconclusive examinations. The peroral examination of the right colon and ileum is a method that lends itself well for overcoming these difficulties.[27,64]

Cleansing of the colon is accomplished in the same manner as for the conventional barium enema examination of the colon. The preparation consists of cathartics, a clear liquid diet for 24 hours, and cleansing enemas. Barium is then given by mouth. After the head of the barium column has reached the transverse colon or the splenic flexure, air is introduced into the rectum. The right colon may then be examined. This method offers several advantages. The examination is possible in conjunction with the examination of the upper gastrointestinal tract and small bowel, thus saving a day of hospitalization. The examination also permits excellent visualization of the ileocecal valve and the terminal ileum, without superimposition by the tortuous sigmoid. This method is of particular value in the simultaneous examination of the cecum, the ileocecal valve, and the terminal ileum (Fig. 13, *A* and *B*).

## VI. NEW APPROACHES TO THE EXAMINATION OF THE LIVER

### A. CONTROLLED POSITION IN TRANSHEPATIC CHOLANGIOGRAPHY

Percutaneous transhepatic cholangiography was first described by Huard and Do-Xuan-Hop in 1937.[28] It was introduced into the practice of medicine as an accepted procedure by Carter and Saypol in 1952.[14] The procedure has been described extensively and recently the value of the pro-

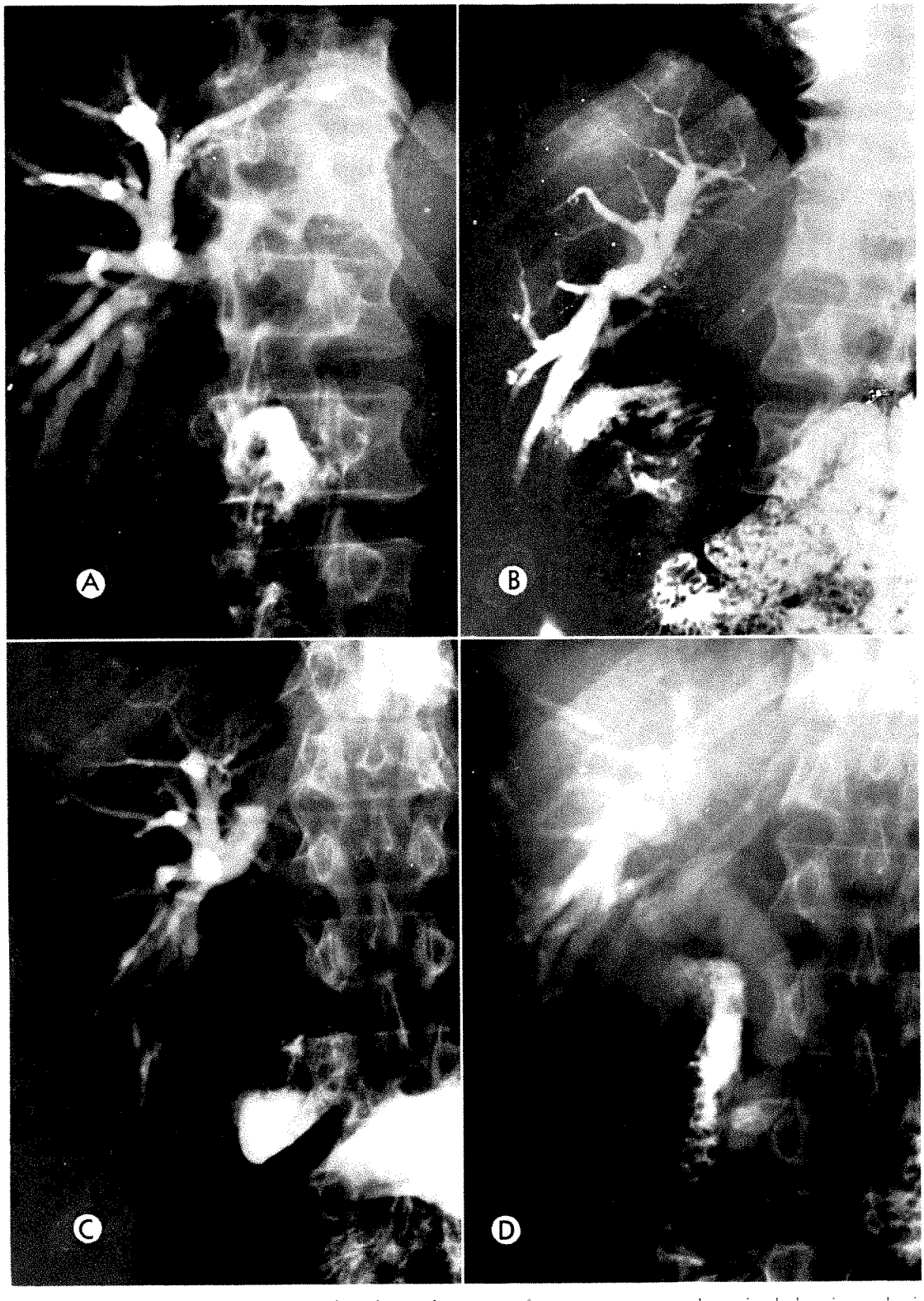



FIG. 14. Advantages of postural control in the performance of percutaneous transhepatic cholangiography in an elderly patient with progressively increasing pain, jaundice, and weight loss. (*A* and *B*) Cholangiograms in opposite oblique views show an indistinct and variable end-point of visualization of a distended hepatic duct. (*C*) An upright roentgenogram shows a fluid level in a distended hepatic duct, demonstrating a trapping effect in a dilated, tortuous hepatic duct. (*D*) Delayed prone roentgenogram of the transhepatic cholangiography shows the common duct obstructed close to the papilla. The site of obstruction is rather sharp, produced by a carcinoma of the head of the pancreas. (Courtesy, Drs. Robert Koch and William Bieber.)

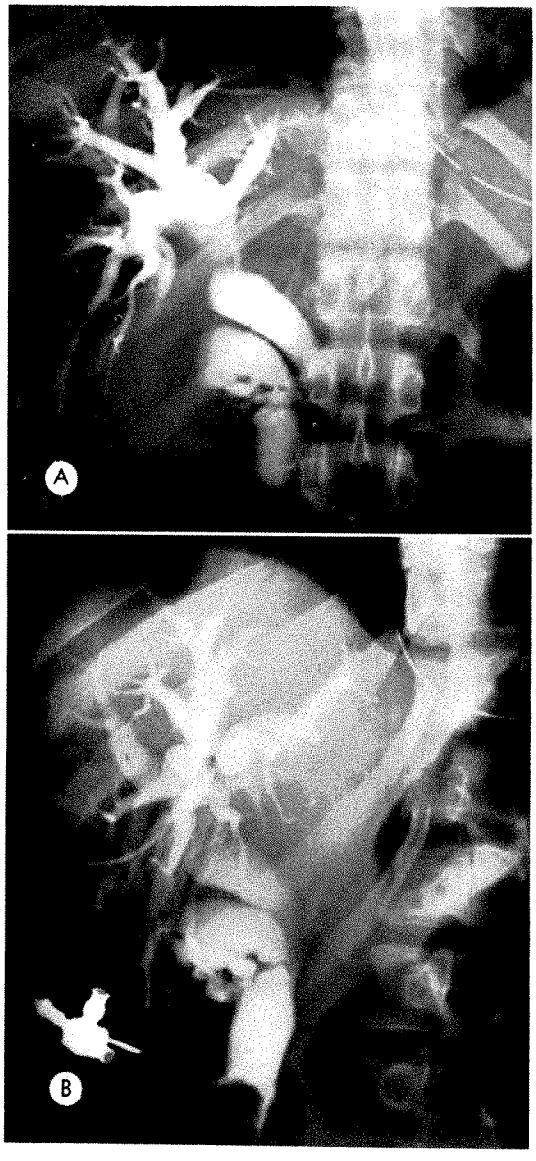



FIG. 15. Advantage of postural control in the performance of percutaneous transhepatic cholangiography. (*A*) A supine roentgenogram shows confusing shadows of the gallbladder and the ducts with an indistinct and abrupt margin of contrast material in the common duct. (*B*) Somewhat delayed, prone roentgenogram shows a cholecystocholedochal fistula with a large filling defect in the common duct, produced by a stone. (Courtesy, Drs. Robert Koch and William Bieber.)

cedure and its present state have been reviewed.[55]

Koch *et al.*[35] have developed a slightly different concept of the approach to the traditional procedure by means of image intensifiers and television for fluoroscopy. Aseptic technique is thus maintained during fluoroscopy and the procedure is performed under constant fluoroscopic control, using test injections and changes in position from oblique to prone and to supine, as well as the erect.

Seldinger[55] emphasized that stasis of bile in a greatly dilated, obstructed common duct may lead to erroneous diagnosis. The obstruction may prevent diffusion of contrast material from the injected peripheral radicle to the site of obstruction in the common duct. By positioning the patient in such a way that contrast material diffuses and mixes with bile, the site of ob-

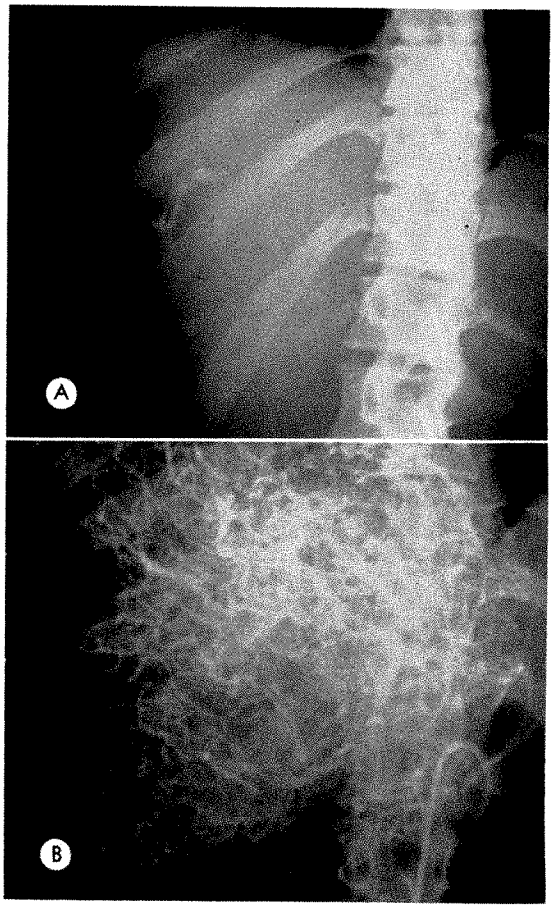



FIG. 16. Slowly progressing hepatoma in a young woman. (*A*) Plain roentgenogram of the abdomen shows a calcification in a greatly enlarged liver. (*B*) Selective hepatic arteriogram shows the massive involvement of the entire liver by neoplastic vessels.

struction and its nature may be identified (Fig. 14, *A-D*; and 15, *A* and *B*).

Careful control of the position will ensure diagnostic roentgenograms that show sharp borders at the site of obstruction. Faint, indistinct borders of contrast material in the most distally visualized part of the ductal system lead to pitfalls.

### B. SELECTIVE HEPATIC ARTERIOGRAPHY

Since the introduction of the Ödman catheter and its modification,[50] percutaneous catheterization of the branches of the aorta has been possible. With increasing skill, the procedure of visceral arteriography becomes more and more selective. Selective arteriography of the hepatic artery is another approach to the diagnosis of primary carcinoma of the liver[6,7,9,46,52] (Fig. 16, *A* and *B*), adenomas[51] (Fig. 17), and liver abscesses and cysts. Although some metastatic lesions are not supplied from the arterial side, in most instances arteriography is diagnostic. The detail obtained by these selective studies is usually superb. Selective hepatic arteriography offers the possibility of studying specific arterial patterns of various metastatic lesions and permits correlation of these patterns with the histologic changes of the tumor.

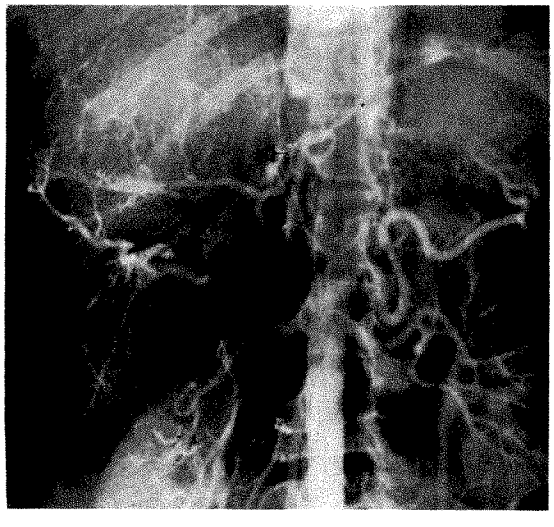


Fig. 17. Multiple adenomas of liver in a young patient. The arteriogram shows 3 adenomas in the right lobe of the liver (arrows).

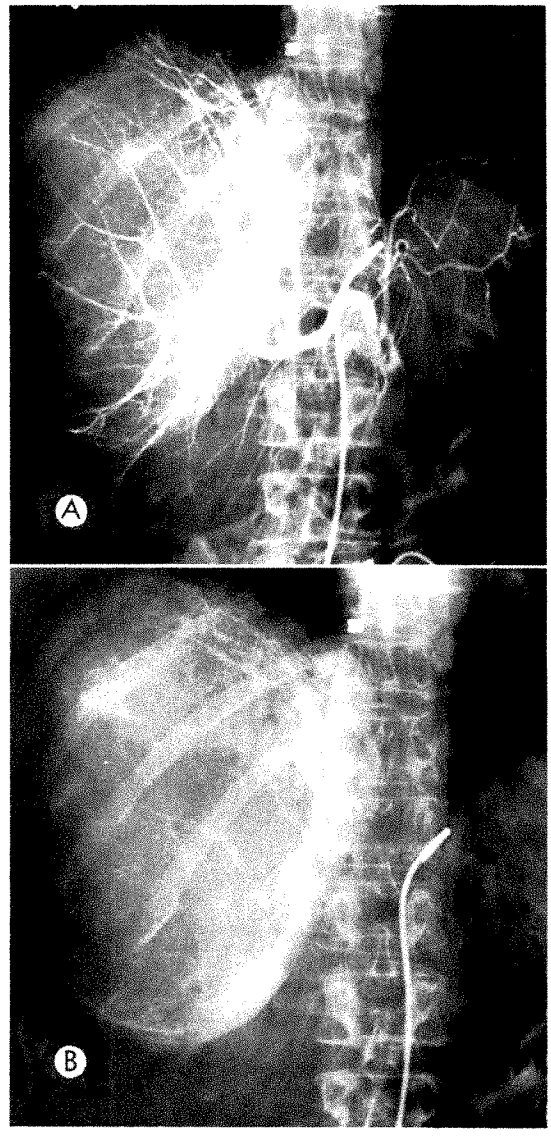



Fig. 18. (*A* and *B*) Excellent arteriographic demonstration of a large liver abscess. The liver vessels are compressed by the large mass which they surround. The mass itself is avascular.

The differential diagnosis of cysts and abscesses is possible when arteriographic changes are correlated with the clinical history (Fig. 18, *A* and *B*).

### C. PORTAL VENOGRAPHY BY SELECTIVE SPLENIC ARTERIOGRAPHY

Percutaneous splenoportography is a procedure that offers considerable diagnostic value, but also involves risks, especially in patients who tend to bleed. Rup-

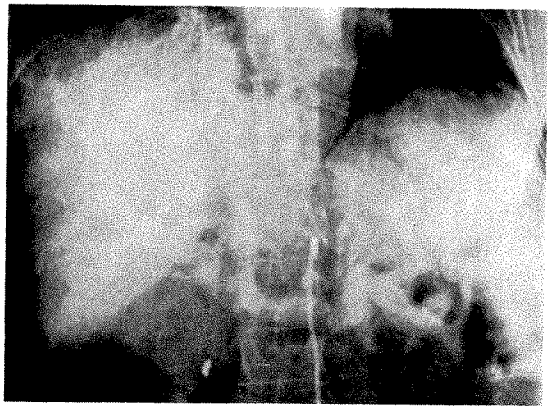


FIG. 19. Selective splenic arteriogram in a patient with liver cirrhosis, obtained with 76 per cent renografin injection. The 25 second roentgenogram shows the sluggishness of circulation. Note the large size of the portal intrahepatic radicles.

ture of the capsule of the spleen is a known complication of the procedure. Splenoportography, furthermore, is not possible after removal of the spleen. It presents technical difficulty if the spleen is very small or in an abnormal position.

Nebesar and Pollard[47] have employed selective splenic arteriography with a large amount of highly concentrated contrast material to study the venous phase and demonstrate the portal circulation. When performed in this manner, the procedure is highly successful in many patients. It allows the gathering of physiologic information about the splenic and portal circulation (Fig. 19). By this method the amount of ascitic fluid may be roughly estimated and the extent of the liver evaluated (Fig. 20, *A* and *B*). By combining selective hepatic arteriography and selective splenic arteriography, information about circulation of both sides of the liver is obtainable.

### D. PERCUTANEOUS UMBILICAL VENOGRAPHY

Studies have shown that the umbilical vein collapses after birth rather than becomes obliterated. In most instances, identification by dissection and the introduction of a catheter into the collapsed vessel is possible.[4,5,13,36] This procedure is a surgical one that may involve entry of the peritoneal cavity. It is only a local dissection, however, and may be done under local anesthesia. After introduction through the umbilical vein, the catheter enters the

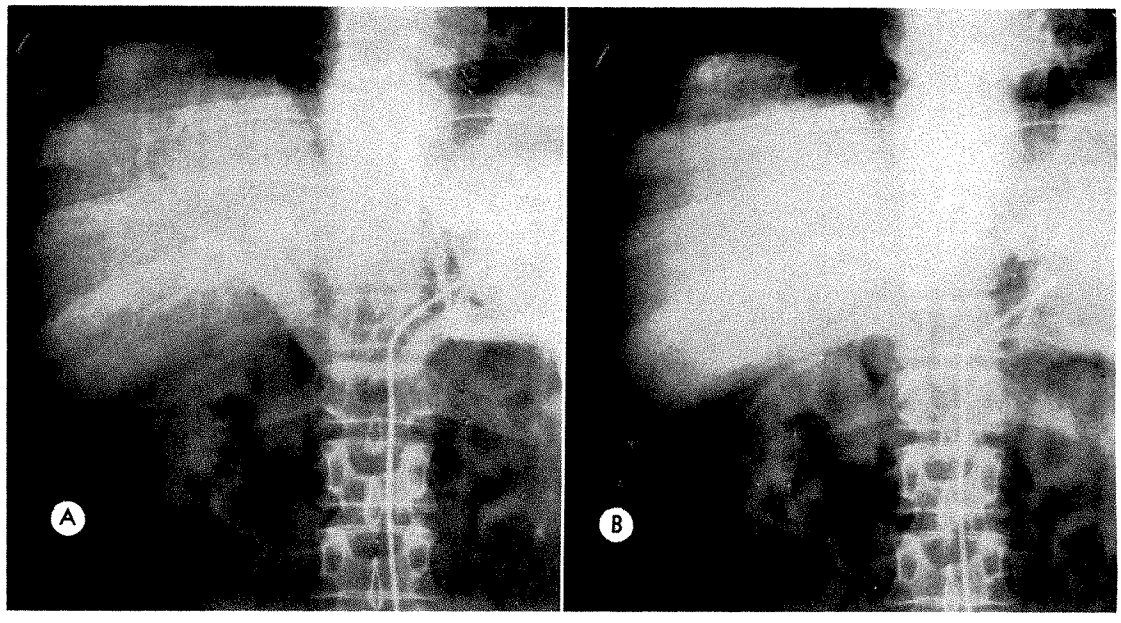



FIG. 20. Demonstration of ascites by means of selective splenic artery injection and portal venogram. (*A*) The venous phase of the selective splenic arteriogram. (*B*) During the phase of liver opacification the distance between the opacified liver and the rib cage is increased. (Courtesy, Dr. Robert Koch.)

portal system and if combined with directional control, study of the portal bed is possible. The method is simpler than percutaneous splenoportography. It offers the choice, with only minimal surgical risk, of a catheter in the portal circulation within the liver or in the extrahepatic portal bed. This ingenious method permits the study of varices, portal collaterals (Fig. 21, *A* and *B*), and even space-occupying lesions in the liver, such as metastases, hepatoma, cysts, or abscesses.

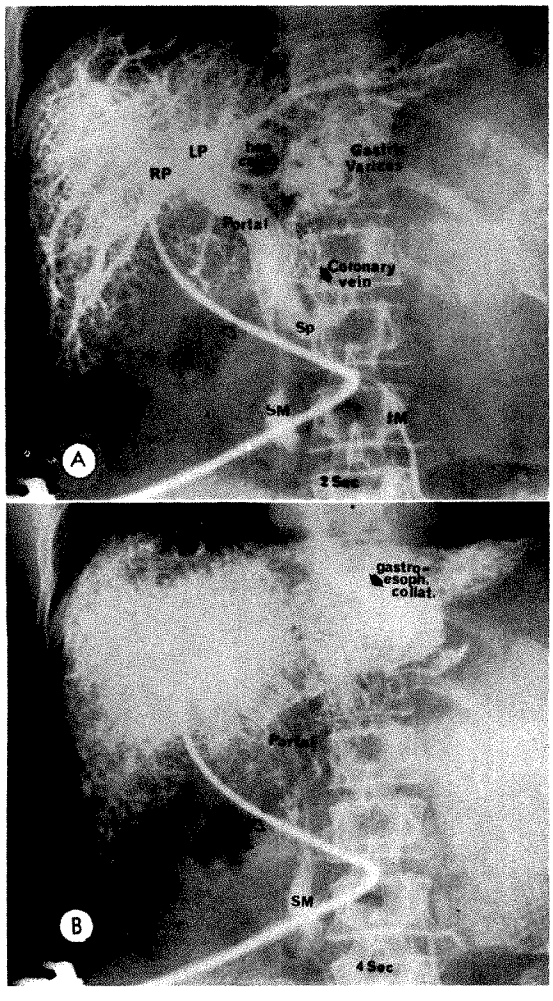



FIG. 21. (*A* and *B*) Percutaneous, transumbilical venous portogram in a patient with cirrhosis shows excellent visualization of the portal vein and of the liver radicles. These are irregular and of a broken-branch appearance. Gastric varices and collaterals are well demonstrated. (Courtesy, Dr. G. B. Skinner, Royal Victoria Hospital, McGill University, Montreal, Canada.)

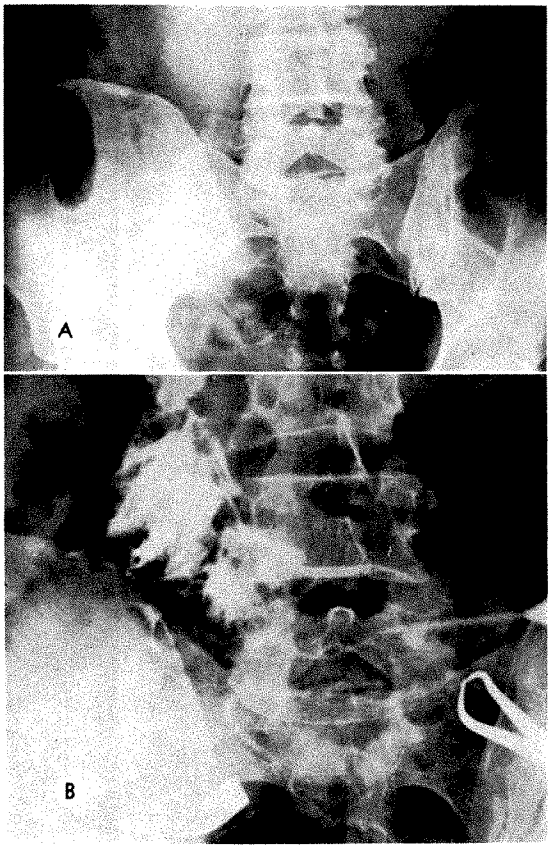



FIG. 22. Operative arteriogram in a patient with carcinoma bleeding into the cecum. (*A*) Pre-injection roentgenogram shows no contrast medium in the bowel. (*B*) After injection of the ileocecal artery, contrast medium has extravasated into the cecum. (Reproduced with permission of *Surgery*, and C. V. Mosby Co., Publisher.[40])

### E. ARTERIOGRAPHIC DEMONSTRATION OF INTRALUMINAL, GASTROINTESTINAL BLEEDING

Massive bleeding into the gastrointestinal tract may be detected arteriographically.[2,3,40,49] This approach is of particular merit when careful search by more conventional means has failed to disclose a site of bleeding. Vascular malformations or very shallow superficial ulcers in some patients have been studied repeatedly, even explored surgically, and no site of bleeding was found. In these patients an arteriogram is indicated. The more selective the approach the greater the likelihood of success. Studies by Jaffee *et al.*[32] revealed that to show the site of bleeding by aortography in dogs, a bleeding rate of 4 to 5

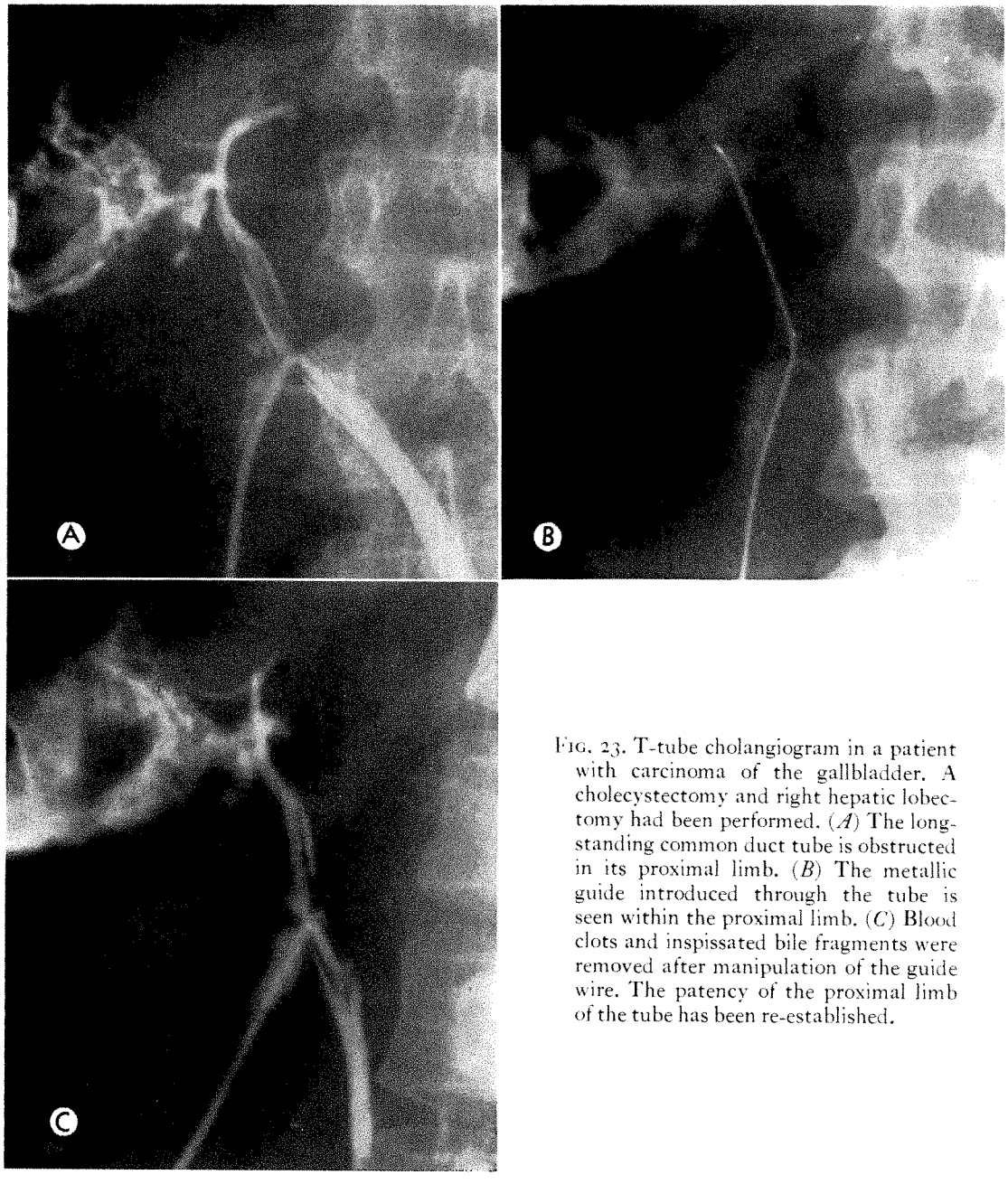



Fig. 23. T-tube cholangiogram in a patient with carcinoma of the gallbladder. A cholecystectomy and right hepatic lobectomy had been performed. (*A*) The long-standing common duct tube is obstructed in its proximal limb. (*B*) The metallic guide introduced through the tube is seen within the proximal limb. (*C*) Blood clots and inspissated bile fragments were removed after manipulation of the guide wire. The patency of the proximal limb of the tube has been re-established.

ml. per minute must be present. With a much more selective approach, Nusbaum and Baum[49] showed that 0.5 ml. per minute was a sufficient rate for demonstrating the site of bleeding.

Performance of selective arteriography in the radiologic department may be contraindicated in a patient with massive bleeding. Selective catheterization in the operating room while the patient is under anesthesia and receiving transfusions may frequently be the safer method (Fig. 22, *A* and *B*).

### F. Interventional Roentgenography in the Gastrointestinal Tract

In the interventional approach to diagnostic roentgenology, the radiologist per-

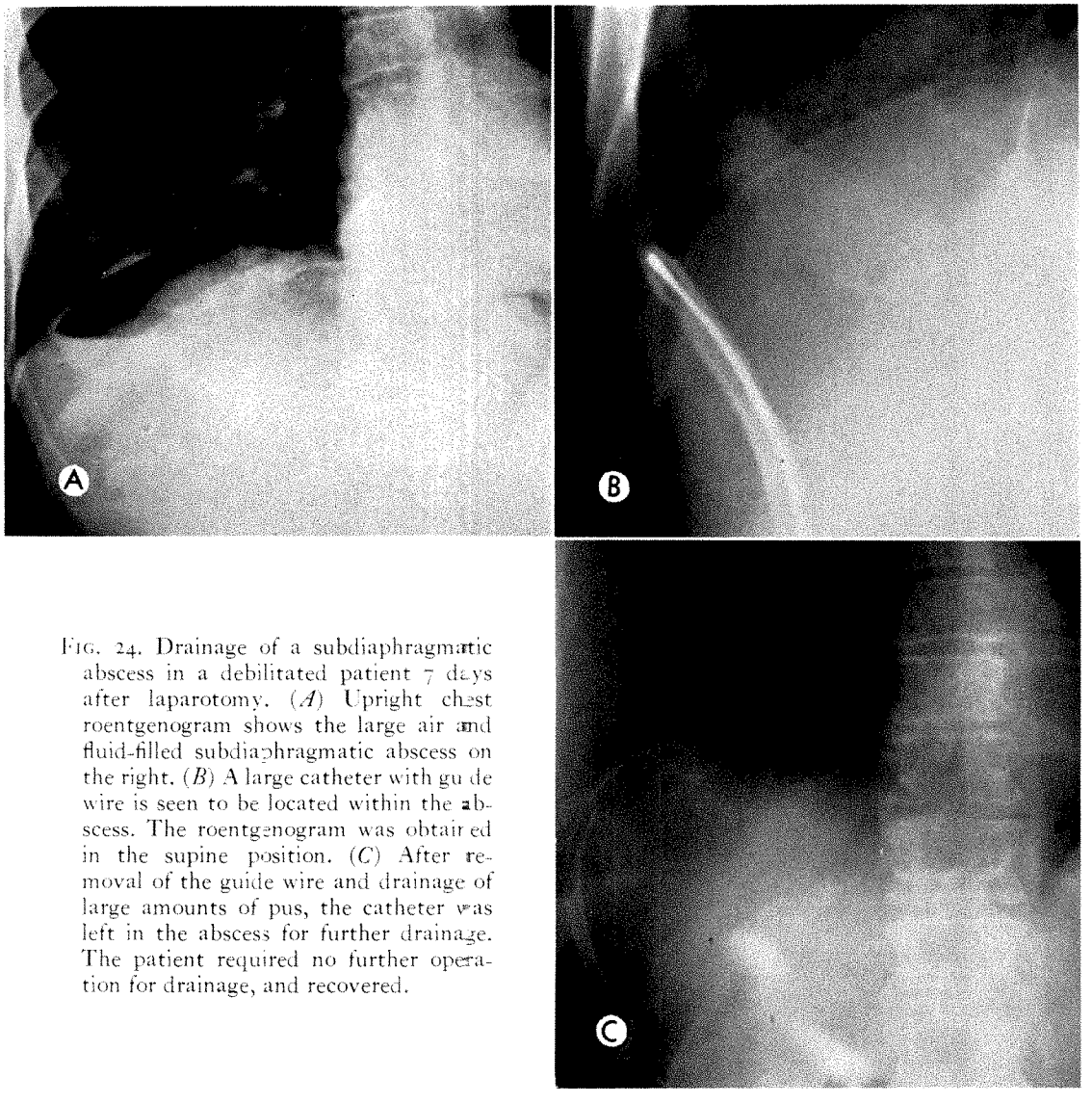



FIG. 24. Drainage of a subdiaphragmatic abscess in a debilitated patient 7 days after laparotomy. (*A*) Upright chest roentgenogram shows the large air and fluid-filled subdiaphragmatic abscess on the right. (*B*) A large catheter with guide wire is seen to be located within the abscess. The roentgenogram was obtained in the supine position. (*C*) After removal of the guide wire and drainage of large amounts of pus, the catheter was left in the abscess for further drainage. The patient required no further operation for drainage, and recovered.

forms a fluoroscopically controlled interventional procedure that may be of therapeutic value. With image intensification and television, performance of these procedures is much facilitated and their scope is widening.[38]

An example of a long accepted and widely practiced interventional procedure is the reduction of intussusception of the colon. Percutaneous transhepatic cholangiography with the catheter left in the duct for drainage is another example.

A brief description of 2 examples of this new approach to gastrointestinal roentgenology follows. One is the reaming and aspiration of sludge and clot from an obstructed T-tube (Fig. 23, *A-C*). This method obviates the necessity of surgical intervention and reexploration of the duct as well as of introduction of irritating chemicals.[43] With further development of catheter-controlled clamps, the crushing of small residual stones will also be possible. In patients with unresectable, obstructing carcinoma, catheters that re-establish the patency of the duct through the tumor may be introduced through a cholecystostomy or T-tube.

In selected instances of extremely ill postoperative patients the radiologist, in conjunction with the surgeon, may also perform a percutaneous, television-controlled drainage of subphrenic abscesses (Fig. 24, *A-C*). Thus, a second laparotomy with its increased morbidity and mortality is avoided.

After introduction of the tube, flushing of the cavity and injection of antibiotics may follow the drainage.

As in all these procedures, close cooperation with physicians and surgeons accomplishes the best results. Radiologists performing these procedures must have the skill, training, and clinical knowledge of complications and know how to remedy them. Observation of the patients must continue and the responsibility for the follow-up must extend beyond the usual period in the radiology department.

Although roentgenograms are obtained during the performance of interventional roentgenology, they are directed primarily at controlling the procedure, as well as at evaluating its results.

## CONCLUSIONS

Because of the limitation of time, many subjects have been discussed somewhat superficially. These represent but a few advances of the last few years. The intention of dealing with so many different approaches is to convey to you a feeling of the vitality, the speed of development, and the multiple directions in which gastrointestinal roentgenology is advancing. Most of the approaches I have discussed were selected because of my own or my associates' experience with them, or because I was impressed by their results during my travels.

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VOL. 101, NO. 2

# SWEET DIARRHEA*

## ROENTGEN DIAGNOSIS OF DISACCHARIDASE DEFICIENCY

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DIARRHEA due to intestinal disaccharidase deficiency is seen in children and adults.[5,11–14] Diagnosis in the past has relied on three methods: (1) Intestinal symptoms after giving or withdrawing the appropriate disaccharide from the diet; (2) blood analysis for glucose, an end product of disaccharide hydrolysis; and (3) intestinal mucosal enzyme assay.

More recently, Laws and Neale,[8] and Haemmerli and Kistler[6] have introduced a roentgenologic method to identify patients with disaccharidase deficiency. When disaccharidase deficient patients are given barium sulfate mixed with a disaccharide, the unhydrolyzed disaccharide remains unabsorbed in the bowel lumen. Within the small bowel it acts as a hydrophilic substance, becomes diluted, distends the small bowel and produces diarrhea. The dilution of the barium produces a characteristic roentgen appearance in the small bowel and colon. Normally disaccharides are absorbed at the brush borders of the intestinal cells. Here hydrolysis by the appropriate enzyme splits the disaccharide into its constituent monosaccharides. Normally only the monosaccharides are absorbed into the blood stream.

A simplified classification of the clinically important disaccharidases and their end products is seen in Table 1. For clinical purposes the lactase group is quite specific, hydrolyzing only lactose to produce glucose and galactose. The sucrase group hydrolyzes sucrose to glucose and fructose but also has some maltase-like activity. The maltase group consists of 5 different enzymes, 2 of which exert a sucrase-like activity. Maltase hydrolyzes a molecule of maltose into 2 molecules of glucose. Thus all of these enzymes can be assessed by measuring blood glucose levels following ingestion of the disaccharide.

TABLE I

PRINCIPAL SMALL BOWEL DISACCHARIDASES

| | | |
|---|---|---|
| Lactose | Lactase Group → | Glucose+Galactose |
| Sucrose | Sucrase Group → | Glucose+Fructose |
| Maltose | Maltase Group → | Glucose+Glucose |

Because of the nonspecificity and multiplicity of the maltase group and since the normal diet contains little maltose, symptomatic maltase deficiency is either rare or nonexistent. Sucrose and lactose are common dietary disaccharides and are hydrolyzed by enzymes which are clinically substrate specific and cannot be adequately replaced by other enzymes if absent. Sucrase deficiency is quite rare and this is fortunate as sucrose has an ubiquity in the ordinary diet that would make its elimination and replacement difficult. Lactase deficiency occupies our main interest as it is quite common and symptom producing. Its detection by roentgenologic methods is the subject of this paper.

### PATIENT MATERIAL

Three groups of patients were examined:

(1) Seven volunteer physicians who acted as control subjects
(2) Fifteen patients with diarrhea or primary bowel disorders
Regional (granulomatous) enteritis ——————————2

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Ulcerative colitis——1
Mucous colitis——1
Pancreatitis——1
Nontropical sprue——1
Scleroderma——1
Peptic ulcer——3
Unexplained diarrhea——5

(3) Eleven patients with previous gastric surgery
Partial gastrectomy with gastroduodenotomy (Bilroth I)——3
Partial gastrectomy with gastrojejunostomy (Bilroth II)——7
Pyloroplasty and vagotomy——1

## METHODS

Following overnight fasting, venous samples were obtained for glucose analysis. Patients then ingested 300–360 cc. of a nonflocculating barium sulfate suspension* to which 25, 50 or 100 grams of lactose had been added. Fifteen subjects received 100 grams, 17 subjects received 50 grams and 1 received 25 grams. Posteroanterior roentgenograms of the abdomen were made and venous samples for glucose analysis were drawn at ½ hour intervals for 2 hours. If the contrast material had not reached the transverse colon in 2 hours, a roentgenogram at 4 hours was obtained. Seven of the 26 patients had a similar examination with 50 or 100 grams of sucrose added to the barium sulfate suspension. Three of the 26 patients had an examination done with 100 grams of maltose added. All 26 patients had small bowel examinations after the ingestion of 300–360 cc. of barium sulfate suspension without the addition of a disaccharide. Roentgenograms were taken in an identical manner at ½ hour intervals.

Fifty grams of lactose were added to 1,000 cc. of barium sulfate suspension and this was used in 2 patients for the examination of their colon.

## RESULTS

### MAXIMAL GLUCOSE ELEVATION

A rise in blood glucose over the fasting level of greater than 20 mg. per cent was considered normal. A rise of less than 20 mg. per cent was considered "flat."

1. *Control subjects.* In 7 healthy subjects the maximal rise in blood glucose over fasting level was 7–37 mg. per cent. Two subjects experiencing rises of 7 and 14 mg. per cent had diarrhea and cramping induced by the 100 gram lactose dose. The remaining subjects had rises of 20–37 mg. per cent without any intestinal symptoms.

2. *Patients presenting with diarrhea or primary gastrointestinal tract disorder.* Six out of the 15 patients had a maximum blood glucose rise above fasting level of less than 20 mg. per cent. The remaining 9 patients had rises of 28–127 mg. per cent. The results are summarized in Table II.

3. *Patients with previous gastric surgery.* Four out of the 10 patients with subtotal gastrectomies had a rise in blood glucose of less than 20 mg. per cent after 50 grams of lactose. The results are summarized in Table III.

### ROENTGEN FINDINGS

In the 16 patients with a normal lactose tolerance curve, the small bowel roentgenograms with and without lactose were similar in appearance.

In the 10 patients who had flat lactose tolerance curves, the roentgen appearance of the small and large bowel was abnormal. The small and large bowel were distended and the barium diluted in the distal ileum and colon with the barium-lactose mixture. When barium alone, barium-maltose or barium-sucrose was used, the barium became more concentrated in the nondistended distal small bowel owing to the normal absorption of water. A more rapid transit time of the barium column into the large bowel with the barium-lactose mix- was also noted in this group.

The 3 roentgen findings of dilution, dilatation and intestinal hurry were always accompanied by diarrhea and cramps and were always seen when there was a flat lactose curve. This quintuple response (flat lactose curve, intestinal hurry, dilution, dilatation and diarrhea) was also found in 2 out of 7 healthy control subjects

* Barotrast, Barnes-Hind Co., Sunnyvale, California. Barotrast does not contain added lactose for dispersal purposes. It does contain other hydrophilic substances.

TABLE II

RESULTS IN PATIENTS WITH GASTROINTESTINAL DISEASE WHEN GIVEN A BARIUM-DISACCHARIDE MIXTURE

| Case | Race | Diagnosis | Disaccharide Administered (gm.) | Maximal Blood Glucose Rise (mg. %) | Diarrhea Induced/ Exacerbated | Roentgen Observations |
|---|---|---|---|---|---|---|
| 1 | Cauc. | Nontropical sprue | Lactose—50 | 0 | Yes | Dilution, dilatation, intestinal hurry |
| | | | Sucrose—50 | 34 | No | Normal |
| 2 | Cauc. | Chronic diarrhea | Lactose—25 | 4 | Yes | Dilution, dilatation, intestinal hurry |
| 3 | Negro | Duodenal ulcer | Lactose—100 | 6 | Yes | Dilution, dilatation, intestinal hurry |
| | | | Maltose—100 | 72 | No | Normal |
| | | | Sucrose—100 | 50 | No | Normal |
| 4 | Negro | Pancreatitis | Lactose—100 | 0 | Yes | Dilution, dilatation, intestinal hurry |
| 5 | Cauc. | Chronic amebiasis | Lactose—50 | 6 | Yes | Intestinal hurry only |
| 6 | Cauc. | Granulomatous colitis | Lactose—100 | 12 | Yes | Dilution, dilatation, intestinal hurry |
| | | | Sucrose—100 | 30 | No | Normal |
| | | | Maltose—100 | 48 | No | Normal |
| 7 | Cauc. | Scleroderma | Lactose—50 | 30 | No | Intestinal hurry |
| 8 | Cauc. | Granulomatous enteritis | Lactose—50 | 44 | No | Normal |
| 9 | Cauc. | Mucous colitis | Lactose—50 | 46 | 0 | Intestinal hurry |
| 10 | Cauc. | Ulcerative colitis | Lactose—50 | 30 | 0 | Normal |
| | | | Sucrose—50 | 55 | 0 | Intestinal hurry |
| 11 | Cauc. | Duodenal ulcer | Lactose—50 | 43 | 0 | Normal |
| 12 | Cauc. | Chronic diarrhea | Lactose—100 | 43 | 0 | Normal |
| 13 | Cauc. | Chronic diarrhea | Lactose—100 | 28 | 0 | Normal |
| | | | Sucrose—100 | 49 | 0 | Normal |
| 14 | Cauc. | Amebiasis, diabetes | Lactose—100 | 127 | 0 | Normal |
| 15 | Cauc. | Diabetes, gastric ulcer, renal carcinoma | Lactose—100 | 85 | 0 | Normal |

when challenged with 100 grams of lactose. In the 2 patients who had colon examinations performed using a barium-lactose mixture, no untoward symptoms occurred during or after the study.

The following are presented as examples of the results obtained:

A healthy radiologist responded normally to a 100 gram dose of lactose by a 34 mg. per cent rise in blood glucose without intestinal symptoms or roentgen abnormalities (Fig. 1*A*). In contrast, a healthy gastroenterologist responded with only a 14 mg. per cent rise, intestinal hurry, as evidenced by contrast material in the rectum at 90 minutes, dilution of the barium column in the transverse colon, and dilatation of the terminal small bowel and colon (Fig. 1*B*).

A patient with nontropical sprue showed a quadruple response of flat lactose tolerance curve (0 mg. per cent rise), intestinal hurry (barium sulfate suspension at splenic flexure at 90 minutes), dilution of barium and dilatation of bowel after 50 grams of lactose; in addition there was flocculation (Fig. 2*A*). In contrast, after 50 grams of sucrose the appearance was normal (Fig. 2*B*). Examination with barium sulfate suspension alone was also normal.

A patient with a total colectomy, terminal ileal resection and ileoproctostomy for granulomatous colitis was given 100 grams of lactose with a quadruple response (Fig. 3*A*). This patient had 18 bowel movements in the first few hours after the lactose dose. This, plus a similar incident with one of the control subjects, prompted the reduction of the lactose dose to 50 grams. The roentgen appearance, when the patient was given 100 grams of sucrose mixed with the barium sulfate suspension, did not re-

TABLE III

RESULTS IN PATIENTS WITH PREVIOUS GASTRIC SURGERY

| Case | Race | Operation | Disaccharide Administered (gm.) | Maximal Blood Glucose Rise (mg. %) | Diarrhea Induced/ Exacerbated | Roentgen Observations |
|---|---|---|---|---|---|---|
| 16 | Negro | BII | Lactose—50 | 8 | Yes | Dilution, dilatation, intestinal hurry |
| 17 | Negro | BII | Lactose—50 | 10 | Yes | Dilution, dilatation, intestinal hurry |
| | | | Sucrose—50 | 86 | No | Dilatation, intestinal hurry |
| | | Converted to BI | Lactose—50 | 8 | Yes | Dilution, dilatation, intestinal hurry |
| 18 | Negro | BII | Lactose—100 | 9 | Yes | Dilution, dilatation, intestinal hurry |
| | | | Maltose—100 | 104 | No | Dilatation, intestinal hurry |
| | | | Sucrose—100 | 122 | No | Intestinal hurry |
| 19 | Negro | BII | Lactose—50 | 6 | Yes | Dilution, dilatation, intestinal hurry |
| 20 | Cauc. | BII | Lactose—50 | 54 | No | Dilatation, intestinal hurry |
| 21 | Cauc. | BII | Lactose—50 | 66 | No | Dilatation, intestinal hurry |
| 22 | Cauc. | BII | Lactose—50 | 54 | No | Dilatation |
| 23 | Cauc. | BI, Diabetic | Lactose—50 | 156 | No | Dilatation |
| 24 | Cauc. | BI | Lactose—50 | 72 | No | Dilatation |
| 25 | Cauc. | BI, Diabetic | Lactose—50 | 99 | No | Dilatation |
| 26 | Cauc. | Pyloroplasty | Lactose—50 | 20 | Yes | Dilution, dilatation, intestinal hurry |

Note: Only those patients with impaired lactose hydrolysis had roentgenographic evidence of dilution of barium.

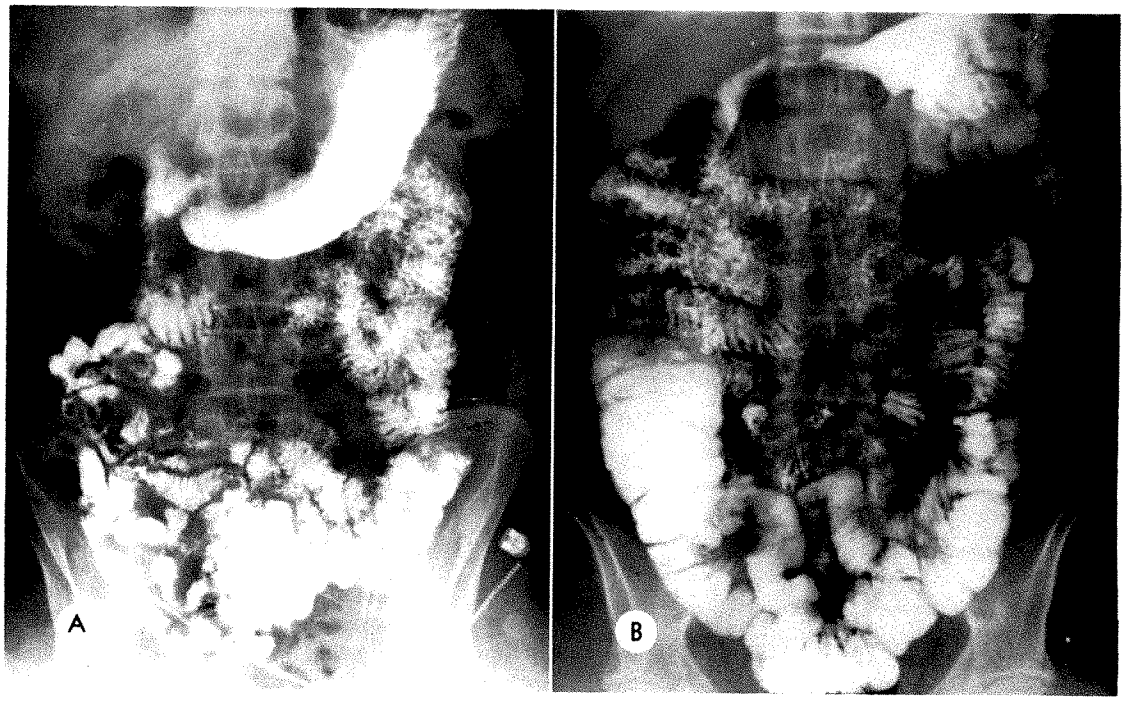



FIG. 1. Asymptomatic subjects. (*A*) Normal rise in blood glucose of 34 mg. per cent following ingestion of 100 gm. lactose. Normal roentgen appearance. (*B*) Maximum elevation of 14 mg. per cent following ingestion of 100 gm. lactose. Dilution of barium in transverse colon and rapid transit were accompanied by diarrhea.

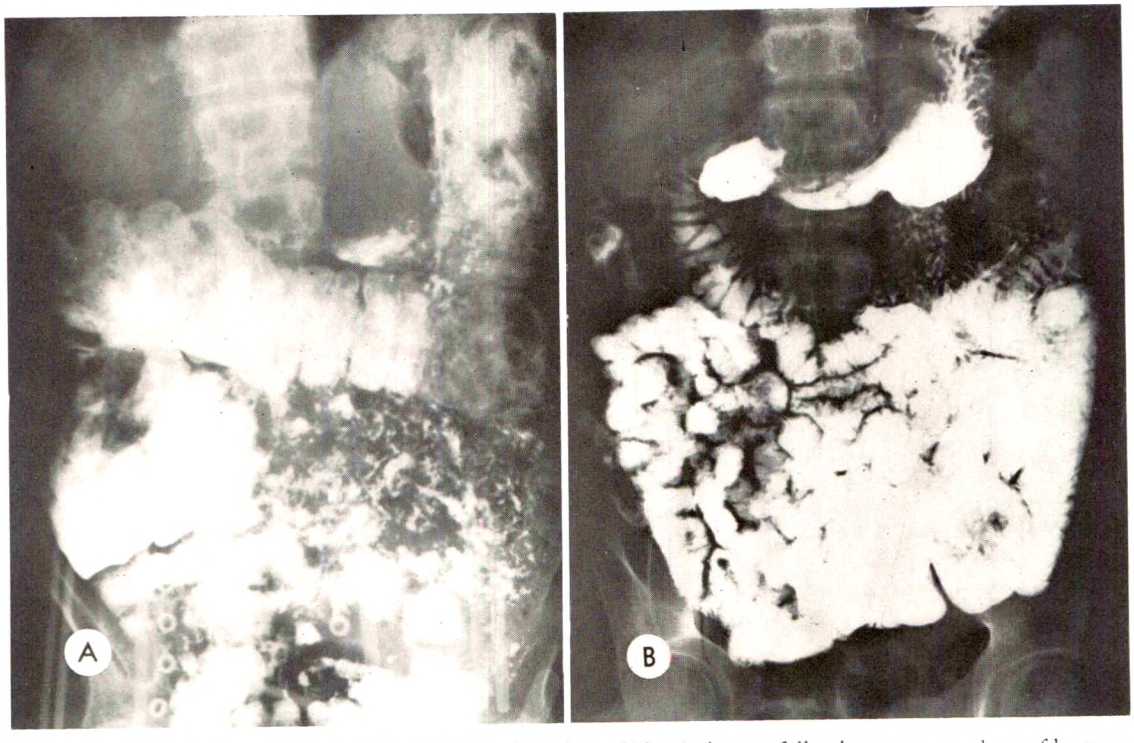



FIG. 2. Patient with nontropical sprue. (*A*) No elevation of blood glucose following a 50 gm. dose of lactose. Dilution of barium suspension in terminal ileum and colon. Bowel dilatation is present. (*B*) A 34 mg. per cent rise in blood glucose following a 50 gm. dose of sucrose. Normal roentgen appearance and no diarrhea.

sult in dilution of the contrast material or intestinal hurry. The patient experienced no cramps or diarrhea (Fig. 3*B*).

In a patient with partial gastrectomy and gastrojejunostomy (Bilroth II operation) rapid motility, dilution and dilatation accompanied the clinical symptoms of diarrhea when 50 grams of lactose were given (Fig. 4*A*). This patient had a flat lactose tolerance curve. The appearance with barium sulfate suspension alone was normal (Fig. 4*B*).

In a patient suffering from chronic diarrhea following pyloroplasty and vagotomy for chronic duodenal ulcer, the signs of intestinal hurry, dilution and dilatation accompanied by diarrhea were present with the lactose examination (Fig. 5*A*). The lactose tolerance curve was borderline (20 mg. per cent). When the examination was repeated with barium sulfate alone, no abnormality was noted (Fig. 5*B*).

## DISCUSSION

Our aim was to assess a simple clinical method for the identification of disaccharidase deficiency.

Man is the only mammal to drink milk throughout adult life and the only mammal in whom lactase activity is maintained after weaning.[11] Despite this, intestinal lactase deficiency has been shown to be common in a variety of intestinal disorders and in a proportion of the normal population. From what is known today, it must be considered the most common small bowel disorder.

Symptoms of this deficiency are diarrhea, flatulence and abdominal cramping. These symptoms will only be induced when there is a disparity between the disaccharidase activity in the intestinal epithelium and the amount of disaccharide ingested. The disparity may be due to excessive amounts of ingested disaccharide, although slow gastric emptying will minimize this. It may be due to reduced length of small bowel from surgical resections or to diseased mucosa containing a reduced amount of enzyme. The diarrhea is attributed to an osmotic effect of undigested lactose in

the small bowel acting as a hydrophilic substance.[7] The roentgenologic studies of Laws and Neale[8] provide confirmation of this. They found that magnesium sulfate added to the barium in normal patients produced a similar small bowel appearance as when lactose was given to lactase deficient patients. Our 2 patients who had lactose instilled in the large bowel without symptoms also support the evidence that the main reason for the diarrhea is the osmotic effect of unabsorbed lactose in the small bowel. The abdominal cramping and increase in flatulence in these disaccharide deficient individuals is most probably related to an increase in intestinal motility. The increased gas and lactic acid production by coliform bacteria acting on unhydrolyzed disaccharide undoubtedly also contributes to increased intestinal motility.

There are a number of screening methods for disaccharidase deficiency. The simplest of these involves the addition or withdrawal of lactose from the diet. While this is of great value, the information obtained is entirely subjective. Lactose tolerance tests are also of great value in determining lactase deficiency. Unfortunately this is beset with certain problems such as delayed gastric emptying. If the disaccharide does not leave the stomach, erroneously low values will be obtained. In patients with diabetes, normal values may be obtained despite lactase deficiency because

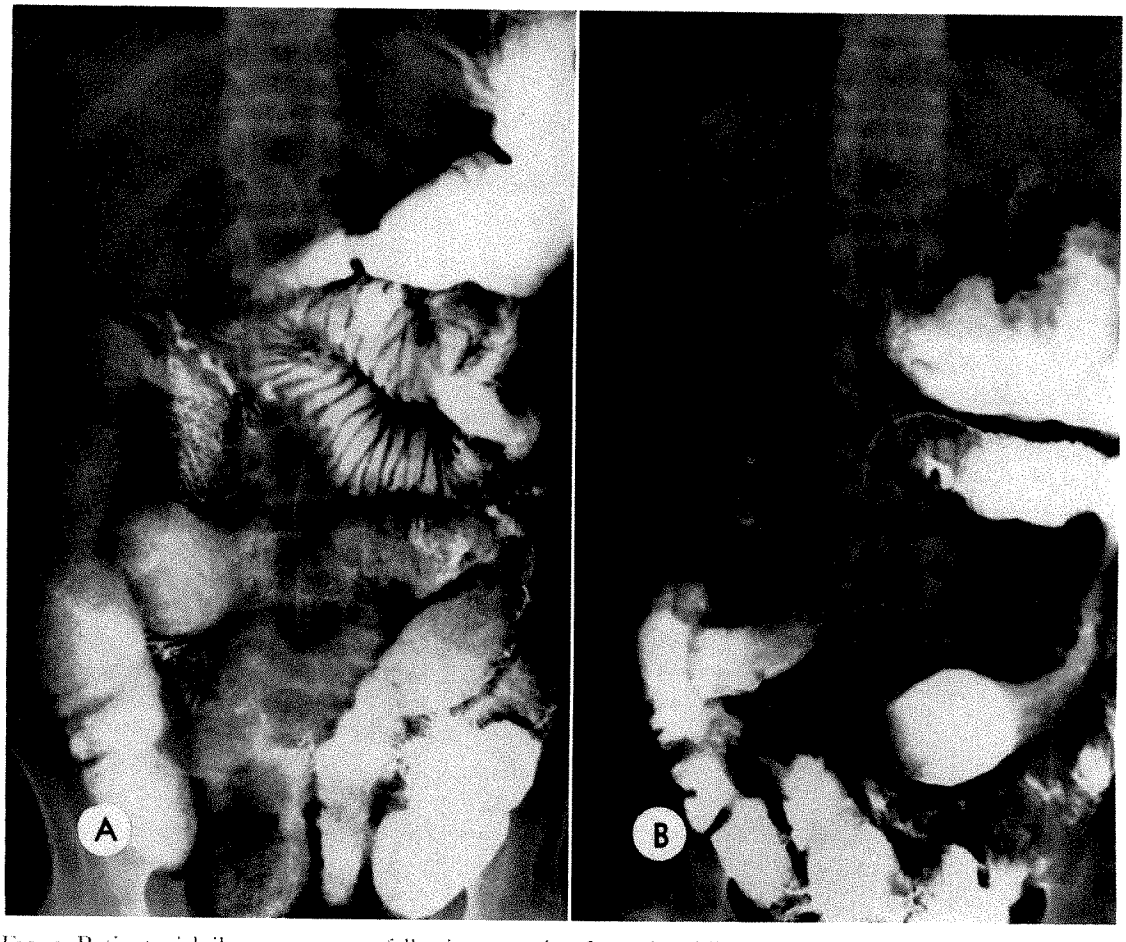



FIG. 3. Patient with ileoproctostomy following resection for regional ileocolitis. (*A*) A 12 mg. per cent rise in blood glucose followed ingestion of 100 gm. lactose. Dilution of barium, dilatation of small bowel and violent diarrhea occurred. (*B*) Normal roentgen appearance and no symptoms following 100 gm. of sucrose added to barium suspension.

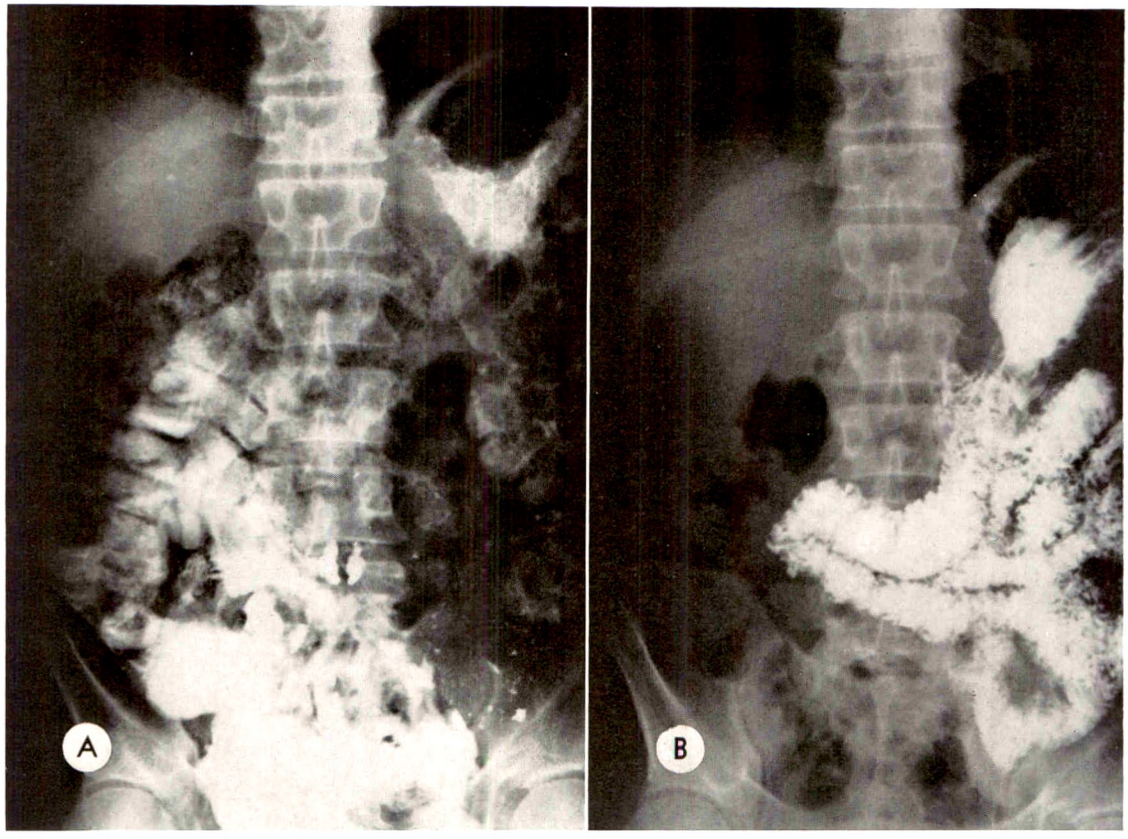



Fig. 4. Patient with Bilroth II type partial gastrectomy. (*A*) A rise in blood glucose of 8 mg. per cent followed a dose of 50 gm. lactose. Dilution of barium and dilatation of distal bowel were the roentgen findings that were accompanied by cramps and diarrhea. (*B*) When studied with barium suspension alone, no abnormalities could be seen.

of slow glucose metabolism. A flat glucose curve which may be associated with diarrhea may be seen in otherwise normal subjects after lactose ingestion. Such subjects cannot be considered truly normal since enzyme assays have shown low lactase activity. Enzyme assay of small bowel mucosa collected by per oral biopsy is reliable but is impractical as a screening procedure.[1,2] Our impression coincides with that of other investigators[6,8] who have suggested that the roentgenologic study may be the most valuable screening aid. It is also convenient as most of these patients have gastrointestinal roentgenologic studies in the normal course of events.

The problem that needs further evaluation is the dose of disaccharide to be used. Although 25 grams of disaccharide is sufficient to induce diarrhea and produce typical roentgen changes in many patients lacking in appropriate enzymes, we are now using a 50 gram dose. This larger dose diminishes the danger of missing patients with slow gastric emptying. A 100 gram dose seems an unrealistic challenge to the small bowel. Fifty grams of lactose represents the amount of disaccharide present in about one quart of milk. This would be about the maximal normal intake expected at any one time.

For clinical purposes serial roentgenograms, a fasting and 30 minute glucose determination seemed to be a simple and sufficient method for good screening. As we gained experience, we felt able to make the diagnosis from the roentgenograms alone. It is fortunate that lactose is not particularly sweet so it may be added to the barium sulfate suspension with little

likelihood of detection by the patient. This allows a more objective analysis of symptom response.

The diagnosis of disaccharidase deficiency is based on 3 roentgen findings and their correlation with symptoms. The roentgen finding that seems to be the most reliable is that caused by the dilution of the barium as it passes through the small bowel. The dilution effect is particularly noticeable in the terminal ileum and colon because in normal patients removal of water from the contrast material results in increased opacity in these areas. Patients who have had a partial gastrectomy may show jejunal dilatation and dilution of barium in the proximal small bowel together with intestinal hurry but if they have a normal lactase assay, the barium-lactose mixture will be concentrated normally by the time it reaches the ileocecal region. Dilatation of the small bowel is a natural consequence of the ingress of water into the bowel lumen in response to the osmotic forces of the disaccharide. This dilatation of the bowel is a powerful peristaltic stimulant and as a consequence rapid intestinal motility is the rule. The extreme variations that occur naturally in intestinal motility made this of least value of the triad of roentgen findings.

Since lactase deficiency is the most common small bowel disorder in man and is roentgenologically detectable, it is our feeling that almost all persons who are to have a small bowel examination should have 50 grams of lactose added to the barium sulfate mixture. Our results suggest that this will not interfere with the examination of the small bowel in patients without lactase deficiency. When a lactase deficiency is discovered, a small bowel examination with barium alone is indicated to identify any morphologic abnormality present.

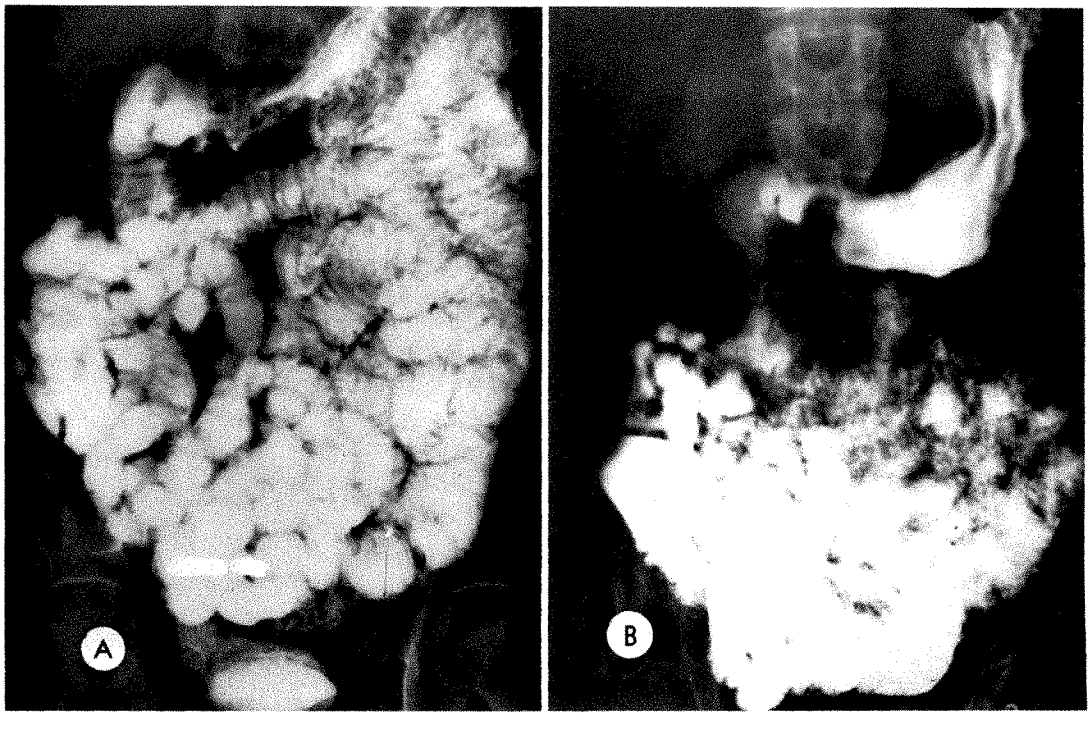



FIG. 5. Patient after vagotomy and pyloroplasty. (*A*) A borderline rise in blood glucose of 20 mg. per cent followed ingestion of 100 gm. lactose. The distal small bowel is dilated and contains diluted barium suspension. Diarrhea accompanied this examination. (*B*) Normal appearance of bowel following ingestion of barium sulfate without lactose.

SUMMARY

Patients with lactase deficiency are unable to hydrolize or absorb lactose. These patients may present flatulence, abdominal cramps and diarrhea, since unabsorbed lactose acts as a hydrophilic purgative. The identification of this deficiency using a barium-lactose mixture is described. Characteristic roentgenographic findings are: barium dilution, bowel dilatation and intestinal hurry. It is suggested that barium-lactose mixture he used routinely in small bowel examinations as lactase deficiency has been shown to be the most common abnormality of the small bowel.

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# FOCAL ULCERATIONS OF THE COLON IN GRANULOMATOUS COLITIS*

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THE segmental distribution, eccentric location, and deep ulcerating nature of granulomatous colitis has been thoroughly described.[5–10,16] Ulcerations localized to a few centimeters in granulomatous colitis have been described,[8–10] but have not been illustrated in the reproduced roentgenograms.[1–16] This report stresses the occurrence of localized eccentric ulcerations of the colon in granulomatous or transmural colitis. Roentgenograms and illustrations of histologic specimens of 2 cases are presented.

## REPORT OF CASES

CASE I. A 53 year old white man complained of weight loss, bloody diarrhea, and cramping abdominal pain of 3 months' duration. His local physician treated him with antibiotics and oral prednisone, which resulted in decrease of diarrhea. When medications were withdrawn, however, the symptoms recurred. The patient had had a perirectal abscess 8 years previously. On admission, barium enema examination disclosed 2 lesions in the left colon (Fig. 1). A left hemicolectomy was performed. The gross and histologic specimens are illustrated in Figures 2, *A* and *B*; and 3, *A* and *B*.

CASE II. A 14 year old white girl was admitted because of arthralgia, fever, skin lesions, rectal bleeding, abdominal pain, and weight loss of 7 months' duration. A rectovulvar fistula was present and several perirectal fistulae. On sigmoidoscopy, the rectum appeared normal. The stools contained no gross blood. An upper gastrointestinal roentgen series showed ulceration and a pseudopolypoid pattern of the terminal ileum. The cecum was also deformed and its mucosal pattern was irregular. The roentgenographic results on barium enema examination and the abnormalities noted on histologic examination of the sections of the colon are illustrated in Figures 4; and 5, *A* and *B*.

## ROENTGEN AND HISTOLOGIC CHANGES

Short segments of the colon were involved in both patients. The abnormal segments were 3 to 6 cm. long. In Case I (Fig. 1–3), both roentgenographic and histologic studies showed 2 areas of disease in the left colon, with normal intervening bowel. Three ulcerating areas in the left colon with normal bowel between were noted in Case II (Fig. 4). The cecum in this patient was also abnormal. The rectum was free of disease in both instances. The short segmental lesions involved only one wall of the colon in both patients. The bowel lumen in these segments was not significantly narrow in caliber. Gross examination disclosed the ulcer margins to be sharp with clearly demarcated overhanging

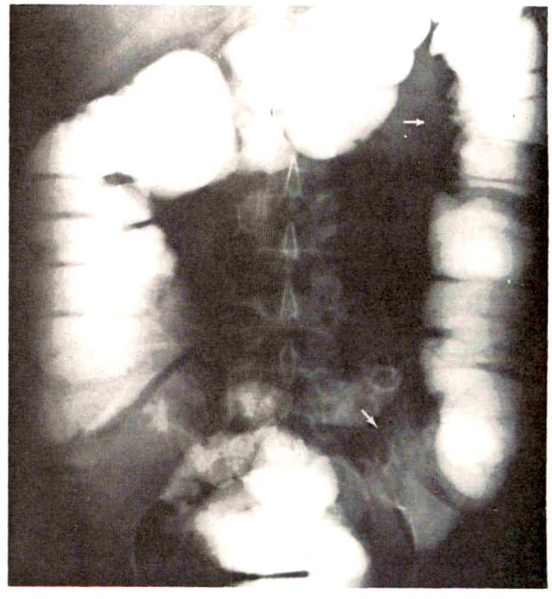


FIG. 1. Case I. In the medial wall of the left colon are 2 lesions, each 5 to 6 cm. long. Sharply defined intraluminal projections with loss of mucosal contour are seen in the upper lesion (upper arrow).

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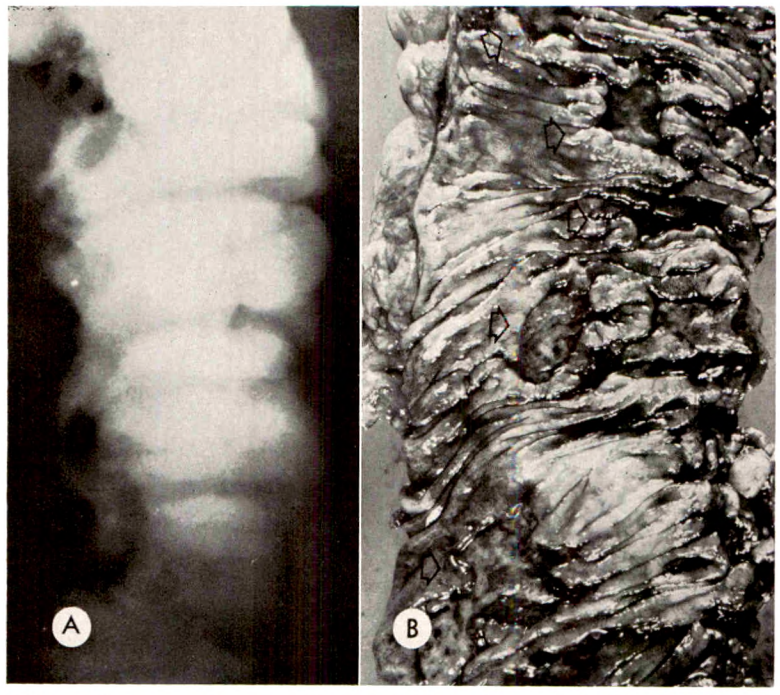



FIG. 2. Case 1. (*A*) Magnified view of the 6 cm. lesion in the proximal descending colon. (*B*) Sharply demarcated ulcerations (arrows) in a segment of the proximal descending colon are in close proximity to one another. The gross mucosal pattern between ulcers is preserved.

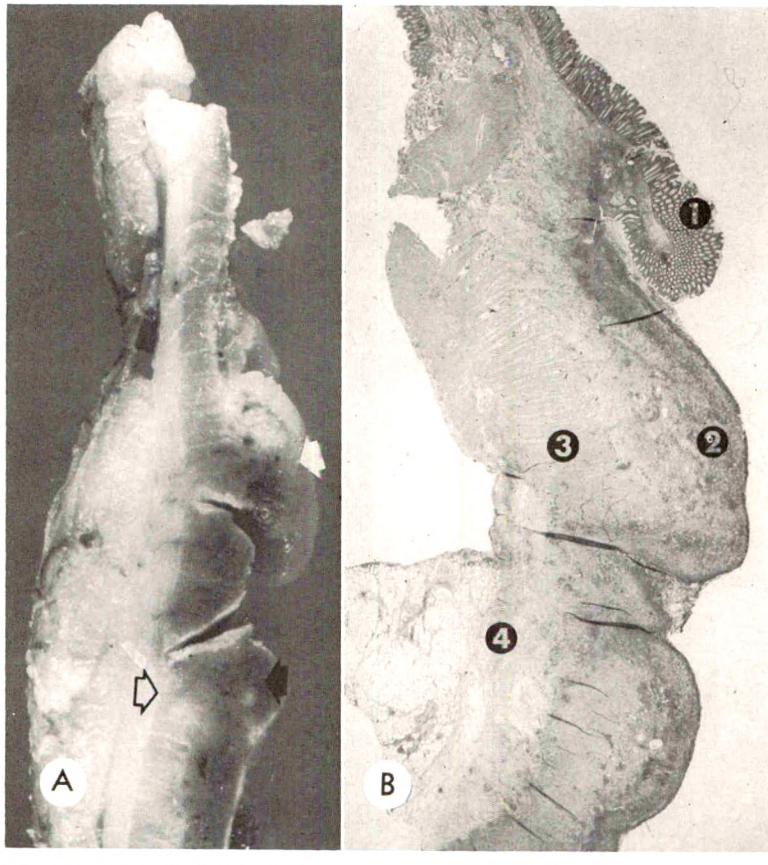



FIG. 3. Case 1. (*A*) Section through the fixed specimen of Figure 2 *B*. The mucosa at the margin of the ulcer is thickened, forming an overhanging edge (solid white arrow). A thickened and discolored submucosa (solid black arrow) and greatly widened muscularis (open arrow) are present. Clefts or fissures penetrate the ulcer base. (*B*) Microscopic section (×10) of specimen from the margin of ulcer shows: (1) hyperplastic mucosa forming an overhanging lip or margin; (2) edematous submucosa with vascular engorgement and cellular infiltrate forming the base of ulcer; (3) hypertrophied, thickened muscularis; and (4) thickened serosa, also infiltrated by inflammatory cells.

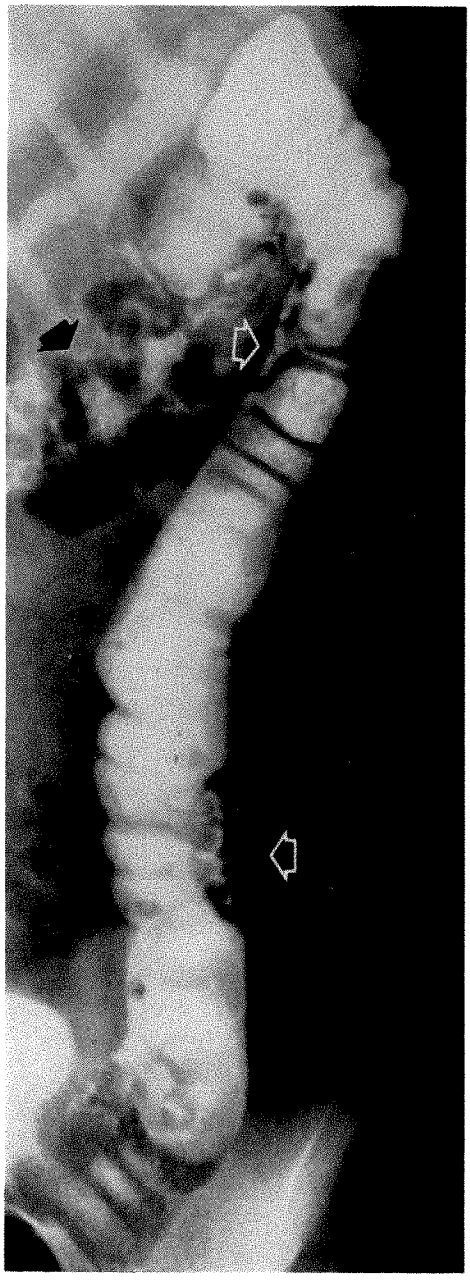


FIG. 4. Case II. The left colon contains 2 distinct areas of involvement, characterized by short, eccentrically located ulcers with sharply defined, overhanging margins (open arrows). A third area of involvement is in the transverse colon (solid arrow).

edges. The ulcers appeared either as single large plaque-like lesions, or as multiple contiguous lesions (Fig. 2*B*). The depth of the ulcers was accentuated by the overhanging margins, composed of intact hyperplastic mucosa infiltrated by inflammatory cells. Normal mucosa extended to within 1 or 2 cm. of the ulcer margin (Fig. 3*A*). The mucosa between ulcerating lesions was normal.

On roentgenograms, the ulcers did not appear to project beyond the luminal contour, even though they were deep (Fig. 1 and 4). Because of inflammation and edema of the entire bowel wall, an indurated tissue mass (mound) had formed, projecting into the lumen (Fig. 3, *A* and *B*; and 5, *A* and *B*). Ulcers in such involved areas may not project beyond the contour of the colon. The area of greatest thickening and induration was in the muscle layers. The transmural nature of the disease was demonstrated on histologic examination. Acute and chronic inflammatory cells were scattered through all layers of bowel, as well as in periserosal tissue. Edema was present throughout. Clefts or fissures penetrated to the level of longitudinal muscle (Fig. 3, *A* and *B*; and 5, *A* and *B*). No granulomata were seen histologically in either case.

## DISCUSSION

Involvement of the colon in granulomatous colitis ranges from universal colon disease to small multiple segmental lesions. Examples of the latter have been presented to stimulate awareness of the truly local, eccentric form of granulomatous colitis. Janowitz *et al.*,[6] Lindner *et al.*,[7] Lockhart-Mummery and Morson,[8,9] Marshak and Lindner,[10] and Wolf and Marshak[16] have aptly characterized the roentgenographic features of granulomatous colitis. Skip lesions, lack of rectal involvement, deep longitudinal ulcers with transverse fissures, and eccentric involvement are the distinguishing roentgenographic characteristics. Strictures of multiple short segments have also been reported.[8,9] Pseudodiverticula may appear along the wall opposite the site of segmental disease,[10] although none was noted in the 2 patients of this report. Despite limitation of the disease process to a 3 to 6 cm. area along

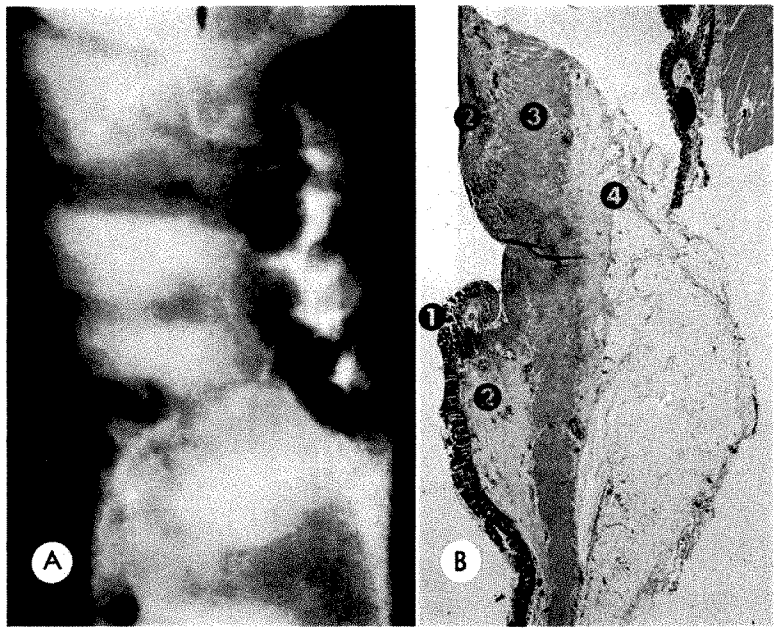



FIG. 5. Case II. (*A*) Magnified view of lower ulcer on lateral wall of descending colon. (*B*) The microscopic section (×10) of specimen of ulcer margin shows: (1) mucosa forming a well demarcated margin of the ulcer crater; (2) edematous submucosa with acute and chronic inflammatory cells; (3) thickened muscularis. The widened submucosa and muscularis form a mound of tissue projecting into the lumen; (4) serosa thickened by cellular infiltrate and edema.

one wall when viewed roentgenographically, on histologic examination all layers of the bowel were seen to be involved. Eccentric ulcerations did not result in significant narrowing of the bowel. Thus, even in extremely short segments in colitis, the transmural nature of the disease was manifest.

The recognition that in granulomatous colitis short segments may be ulcerated is important. These ulcers differ from those of ulcerative colitis. They form on a mound or tissue mass composed of edematous inflamed submucosa and muscularis. Thus, they appear to occur on an intraluminal mass and do not project beyond the lumen. This development is comparable to that of a malignant ulcer in a gastric tumor, in which the crater also does not project beyond the lumen. This type of ulceration has not been illustrated in the literature. More typical of ulceration in granulomatous colitis is the serpiginous longitudinal ulcer, and the deep transverse fissure penetrating all layers of bowel.[10] Cone-shaped ulcers, a few millimeters in diameter, have also been described in early granulomatous colitis.[2]

Diverticulitis and carcinoma are 2 other disease processes that may be confused with eccentric ulcerations of short segments. In diverticulitis, extramural abscesses along one wall of bowel, associated with intramural abscesses, cause smooth filling defects of the wall, which may simulate the deep ulcers of local granulomatous colitis. The presence of diverticula and the absence of ulcerations and of sharp overhanging margins aid in differentiating diverticulitis from the focal eccentric ulcers of granulomatous colitis. Although scirrhous carcinoma may be eccentric, associated ulcers with sharp overhanging margins would be unlikely.

## SUMMARY

Two cases of granulomatous colitis that involved short segments of bowel are presented. These illustrate that focal, eccentric, ulcerating lesions are one part of the

spectrum of colon involvement that also includes universal colon disease. On roentgenograms, sharp overhanging margins of the ulcers did not appear to project beyond the lumen of the colon. Histologic examination showed that the ulcers arose on a mound of tissue composed of edematous submucosa and muscularis infiltrated by inflammatory cells. This mound projected into the lumen of the bowel. Correlative results between roentgenographic and histologic examinations are presented.

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# CALCIFIED ADENOCARCINOMA OF THE COLON*

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TO THE radiologist, calcifications are indicators of various pathologic or physiologic processes within the body. The roentgenographic identification of calcification within the abdomen is frequently a valuable aid in reaching the correct diagnosis. However, calcification rarely suggests a gastrointestinal malignancy.

Because of the importance of recognizing neoplastic lesions of the gastrointestinal system, the following case of a calcified primary adenocarcinoma of the colon is reported.

## REPORT OF A CASE

L.H. 122 49 65, an 18 year old Negro female was admitted to the Johns Hopkins Hospital because of abdominal swelling. She had been in good health prior to admission, except for minor abdominal swelling which previously had occurred and subsided synchronously with menstruation. Three weeks prior to admission, the patient experienced marked generalized abdominal swelling during her menstrual period. This swelling, however, did not recede at the end of menstruation, but progressively increased until one week prior to admission. At that time, she noticed sharp right upper quadrant crampy pain and became constipated. There was no melena or rectal bleeding.

Admission physical examination revealed a blood pressure of 125/80, pulse 80, respiration 20, temperature 37.4° C. Her abdomen was distended and tympanitic. The liver and spleen were not palpable. She had slight right upper quadrant tenderness without rebound. The bowel sounds were normal. Rectal examination was unrevealing but on pelvic examination a mass was felt in the pouch of Douglas. The uterus was of normal size.

Laboratory examination demonstrated a hematocrit of 33 per cent and the white blood cell count was 6,700 with a normal differential. The stools were strongly guaiac positive. Liver function tests, including bilirubin determinations, were normal. Malignant cells compatible with adenocarcinoma were demonstrated on cytologic examination of the vaginal pool.

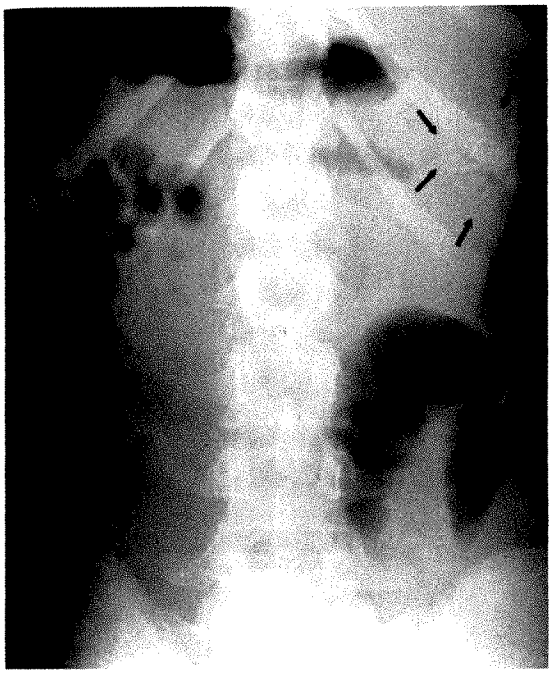


FIG. 1. Abdominal roentgenogram showing calcified mass in left upper quadrant. Gas-filled loop of bowel is seen below the calcification suggesting partial bowel obstruction.

Roentgenographic examination of the abdomen revealed mottled and curvilinear calcific densities within a 4×6 cm. area in the left upper quadrant (Fig. 1 and 2). On the lateral roentgenogram, a large soft tissue mass was seen which occupied the entire anterior abdomen and pelvis. A distended loop of small bowel in the left mid abdomen suggested partial bowel obstruction. Intravenous pyelography showed the calcification to be anterior to the left kidney. A barium enema study demonstrated complete obstruction to the flow of barium at the site of the calcified mass in the transverse colon, just proximal to the splenic flexure (Fig. 3).

At operation, a large, nodular tumor was

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FIG. 2. Detailed view of left upper abdominal quadrant showing calcification.

found in the transverse colon immediately proximal to the splenic flexure. There was extensive peritoneal and omental tumor seeding. The transverse colon, greater omentum, spleen and tail of the pancreas were resected and an end-to-end colonic anastomosis was done.

The resected specimen included 35 cm. of colon with the mesentery, omentum, spleen and tail of the pancreas (Fig. 4). The mesentery and omentum were extensively infiltrated by gelatinous tumor. The tumor extended into the peripancreatic tissues and into the hilus of the spleen. Large subserosal nodules, as seen in the close up photograph (Fig. 5), were present on the colon and attached mesentery at the site of the primary tumor. The roentgenogram of the specimen illustrated the calcification within these nodules (Fig. 6). When the bowel was opened, the primary tumor was found to be approximately 6 cm. in length, extending circumferentially around the lumen of the bowel and through the muscle wall into the surrounding tissues.

Microscopically, the tumor was a diffusely infiltrating mucin-producing adenocarcinoma of the "signet-ring" variety (Fig. 7). Numerous calcified areas within the pools of mucin were evident (Fig. 8). At higher magnification, these calcified foci were found to represent calcified necrotic tumor cells (Fig. 9). Less dense calcium deposits were present in the mucin.

## DISCUSSION

Nine cases of roentgenographically demonstrable calcification in primary adenocarcinoma of the colon have been previously described in the English language medical literature.[3,4,6–8,11,13,14] They are summarized with the present case in Table 1.

In this series, there was an equal sex incidence. Eight of the 10 patients were less than 34 years of age and 3 were under 18 years.

Five of the cases originated in the ascending colon,[3,7,13,14] 2 in the rectum,[6,11] and 1 each involving the descending colon,[8] cecum,[4] and transverse colon (authors). Hermann and Rozin[6] reported calcification



FIG. 3. Barium enema examination showing obstruction of transverse colon immediately proximal to splenic flexure at site of calcification.

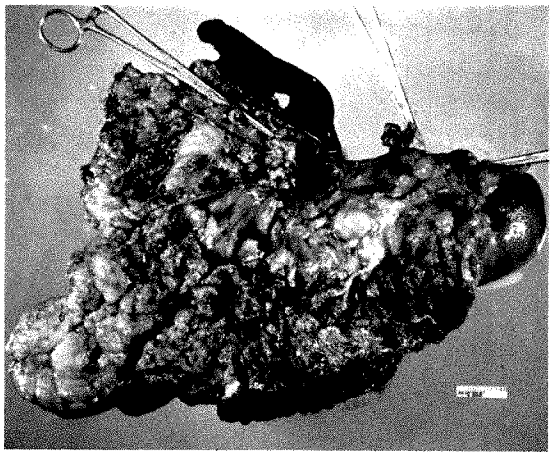


Fig. 4. Photograph of resected specimen with clamps on the colon margins. The attached spleen is on the right. The mesentery is extensively involved by tumor.

in a recurrence of a carcinoma of the transverse colon which was initially treated by surgery and radiation therapy.

The calcifications have generally tended to be finely mottled.[1] The authors' case, however, showed curvilinear calcification combined with mottled calcific densities. All of the cases reported have been mucus-producing adenocarcinomata.

While roentgenographically demonstrated calcification appears to be uncommon, several histologic studies indicate that colonic carcinoma may undergo calcification or ossification which can not be appreciated roentgenographically.[1,2] Calcification occurs in areas of necrosis or mucinous

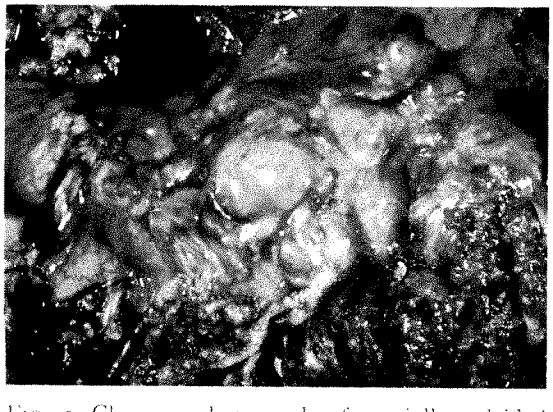


Fig. 5. Close-up photograph of partially calcified subserosal nodules corresponding to roentgenogram of specimen (Fig. 6).

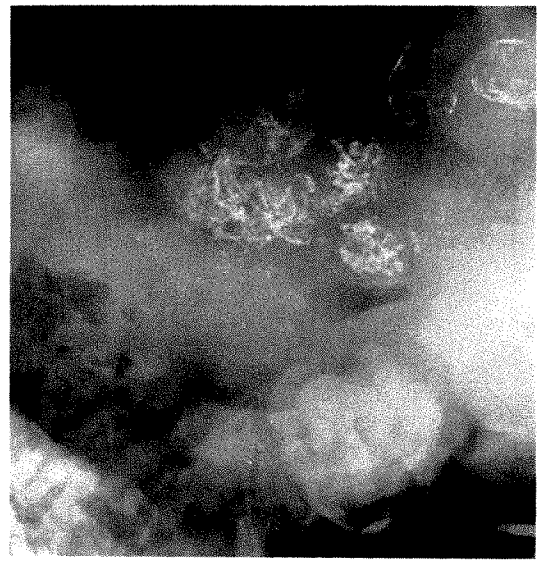


Fig. 6. Roentgenogram of surgical specimen showing shell-like and punctate calcifications.

degeneration. While osseous metaplasia in colonic neoplasms can be readily identified histologically, it cannot be differentiated from calcification roentgenographically.[4]

The mechanism of calcium deposition in these tumors is not understood. Changes in concentration of calcium and phosphate ions combined with alkalinity of the tissue environment may favor the deposition of calcium salts. The glycoproteins produced by mucoid degeneration may also enhance calcium deposition.[4]

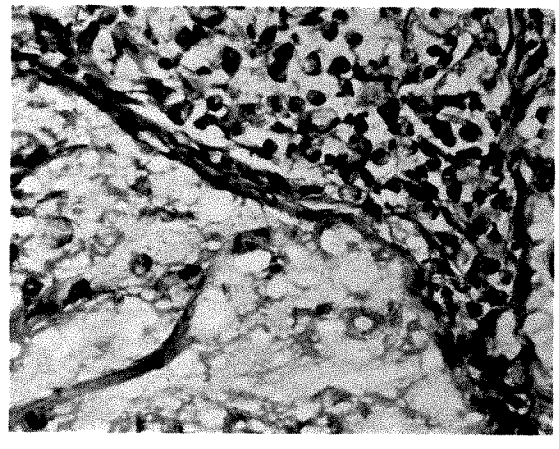


Fig. 7. Photomicrograph of representative area of the viable tumor. Mucin-producing "signet-ring" cells in lower left. Extracellular mucin upper right. H & E (300×).

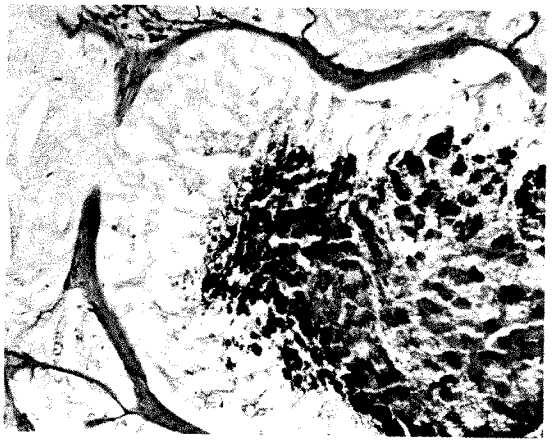


FIG. 8. Low power photomicrograph showing calcified area of the tumor suspended in mucin. H & E (120×).

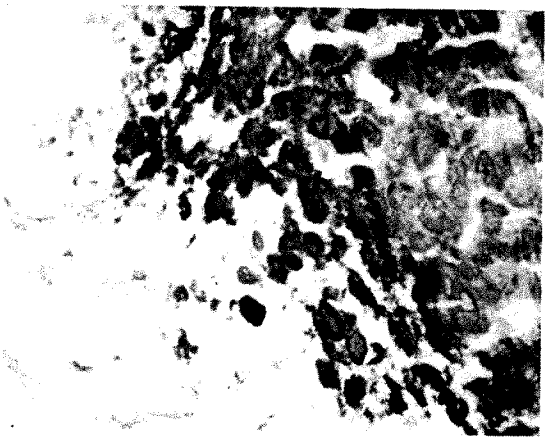


FIG. 9. High power of Figure 8 illustrating calcification of necrotic tumor. Compare to noncalcified viable tumor (Fig. 7). H & E (300 ×).

The differential diagnosis of calcified intra-abdominal lesions is extensive and has been thoroughly discussed by McAfee and Donner.[9] Calcified colonic carcinoma should be considered in all cases in which the distribution of the calcium follows the course of the large bowel. Other lesions related to the large bowel which calcify include: pericecal abscess, mucocele of the appendix, calcification in congenital duplications and the colonic calculi of Hirschsprung's disease.[9] Most of these can be excluded by the appearance of the calcifications.

TABLE I

SUMMARY OF REPORTED CASES

| Author | Sex | Age | Site of Lesion | Roentgenographic Description of Calcification | Histology | Location of Calcification |
|---|---|---|---|---|---|---|
| 1. Sangster[11] 1954 | F | 33 | Rectum | Fine granular calcification | Adenocarcinoma and mucoid carcinoma | Not stated |
| 2. Matthews *et al.*[8] 1958 | M | 25 | Descending colon | Punctate calcification at site of constricting neoplasm | Mucinous adenocarcinoma | In mucinous material |
| 3. Green[3] 1959 | F | 51 | Ascending colon | Irregular lattice-like pattern of calcification | Mucus secreting adenocarcinoma | Not stated |
| 4. MGH CPC 46492[7] 1960 | M | 26 | Ascending colon | Mottled calcifications | Colloid carcinoma | Not stated |
| 5. Zboralske *et al.*[14] 1962 | M | 14 | Ascending colon | Mottled irregular calcification | Mucinous adenocarcinoma | Within mucinous spaces |
| 6. Zboralske *et al.*[14] 1962 | M | 15 | Ascending colon | Mottled irregular calcification | Mucinous adenocarcinoma | Within mucinous spaces |
| 7. Hall[4] 1962 | F | 70 | Cecum | Amorphous rounded and punctate calcifications | Papillary adenocarcinoma | Mucinous degeneration with calcification |
| 8. Shevach[13] 1962 | F | 32 | Ascending colon | Dumbbell shaped calcific mass | Mucinous adenocarcinoma | Calcifications within mucus |
| 9. Hermann and Rozin[6] 1964 | M | 21 | Rectum | Miliary calcifications | Mucous cell adenocarcinoma | Not stated |
| 10. Present authors 1967 | F | 18 | Transverse colon | Mottled and curvilinear calcifications | Mucinous adenocarcinoma | Necrotic tumor cells within mucinous pools |

Calcification in metastases from carcinoma of the colon have also been described.[6,10,12]

Positive cytologic examination of cervical or vaginal pool smears is a rare finding in carcinomas not related to the female genital tract. Of 102 women with positive cytology, Hellwig[5] found 1 patient with malignant cells due to carcinoma of the sigmoid colon.

## CONCLUSION

Calcification in primary carcinoma of the colon is an unusual finding, but one which should be considered in the differential diagnosis of calcified abdominal lesions. A new case of calcification in an adenocarcinoma of the colon which was roentgenographically demonstrated is reported, with a review of 9 similar cases from the literature. The calcifications in these lesions tend to be mottled in appearance, but may have curvilinear components. Histologically, those neoplasms of the large bowel in which calcium is found are mucus-producing adenocarcinomas.

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# PRIMARY SCIRRHOUS CARCINOMA OF COLON*

## ROENTGENOLOGIC DIAGNOSIS

*By* A. J. McQUEENEY, R. W. OLSON, *and* A. MEDWID
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PRIMARY linitis plastica or scirrhous carcinoma of the colon characteristically produces diffuse, segmental, stenosing fibrosis of the bowel wall, with minimal to absent mucosal ulceration; this fact alone tends to obscure its roentgenologic diagnosis.

Two basic types of scirrhous carcinoma involve the colon:

1. Metastatic lesions, secondary to breast or gastric carcinoma. These spread to the colon via the hematogenous route, or, in the case of a primary gastric carcinoma, metastasis is usually to the transverse colon via the gastrocolic ligament.[1,3,5,6]

2. Primary scirrhous carcinoma arising in the colon in the absence of gastric carcinoma.[2,3,4,6,7,8] It is this type which presents the radiologist with challenging problems of differential diagnosis, and is the subject of the present paper.

A relatively uncommon form of primary colonic carcinoma, the scirrhous carcinoma, has a reported incidence of about 1 per 1,000 colorectal carcinomas surgically resected at the Mayo Clinic.[2] Since less than 20 proven cases are recorded in the literature, this form of scirrhous carcinoma of the colon is relatively unfamiliar to the diagnostic radiologist.

### PATHOLOGY

Like its counterpart in the stomach, linitis plastica or scirrhous carcinoma of the colon is a diffusely infiltrating, anaplastic carcinoma which produces marked fibrosis, with symmetric thickening and rigidity of a relatively long segment of colon throughout its entire circumference.

Mucosal ulceration is characteristically minimal, superficial or absent, although the mucosa is fixed and normal mucosal folds are flattened and obliterated, particularly in the stenotic central portion of the lesion.

Individual tumor cells are sparsely scattered throughout the abundant fibrous tissue, and often show the characteristic "signet ring" displacement of the nucleus by a mucin globule, which identifies the lesion as an anaplastic adenocarcinoma.

The histologic appearance may be confused with an inflammatory process, particularly in frozen sections from scirrhous areas where inflammatory cells coexist with tumor cells (Fig. 4).

The lumen of the involved segment of bowel is stenotic, usually measuring about 1.0 cm. in diameter, and tumor extension along mesenteric vascular channels produces a fibrotic, thickened, shortened mesentery which is characteristically rigid. The radiologist generally sees primary carcinoma of this type in the stomach, but rarely encounters such lesions in the colon.

Metastatic spread almost always involves the regional lymph nodes, and angioinvasion or perineural invasion occurs in about half of the cases.[2] In female patients, large Krukenberg type metastases are often present in both ovaries.[3]

About one third of reported cases arise as a complication of long standing ulcerative colitis.

The following case, verified by autopsy, is typical of primary scirrhous carcinoma of the colon, and serves to illustrate many of its clinical and roentgenologic features.

### REPORT OF A CASE

A 24 year old white housewife presented with a 3 month history of increasing constipation unrelieved by laxatives, and associated with

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Fig. 1. Scirrhous carcinoma, primary in colon. Note the high grade obstruction, the long segment of involvement, the tapered ends, and the lack of intraluminal mass or mucosal ulceration.

severe lower abdominal cramps. The stool was soft, occasionally blood-streaked and became progressively smaller in caliber.

Physical examination revealed a very thin young woman, who appeared chronically ill. The breasts were normal. Abdominal, pelvic and rectal examinations were negative.

Barium enema study showed almost complete retrograde obstruction to the flow of barium by a large lesion in the sigmoid colon (Fig. 1 and 2), which appeared to be intramural, constricting the lumen and showing smooth funnel-shaped margins and a flattened mucosal pattern with questionable superficial mucosal ulceration. Chest roentgenograms, intravenous pyelograms and upper gastrointestinal series were negative. Sigmoidoscopic examination showed almost complete obstruction at the 14 cm. level by an "extra-mucosal lesion"; the examiner commented on the absence of bleeding, and stated that there was "no lesion to biopsy."

On September 30, 1964 a large constricting lesion of the upper sigmoid was resected after a frozen section diagnosis of anaplastic fibrocarcinoma, linitis plastica type. Prior to resection, abdominal exploration revealed normal ovaries, with no evidence of gastric carcinoma.

The resected segment of bowel showed an indurated, obstructive, stenosing lesion in its middle third, with marked thickening of the attached sigmoid mesentery (Fig. 3). The tumor measured 6 cm. in length within the bowel wall

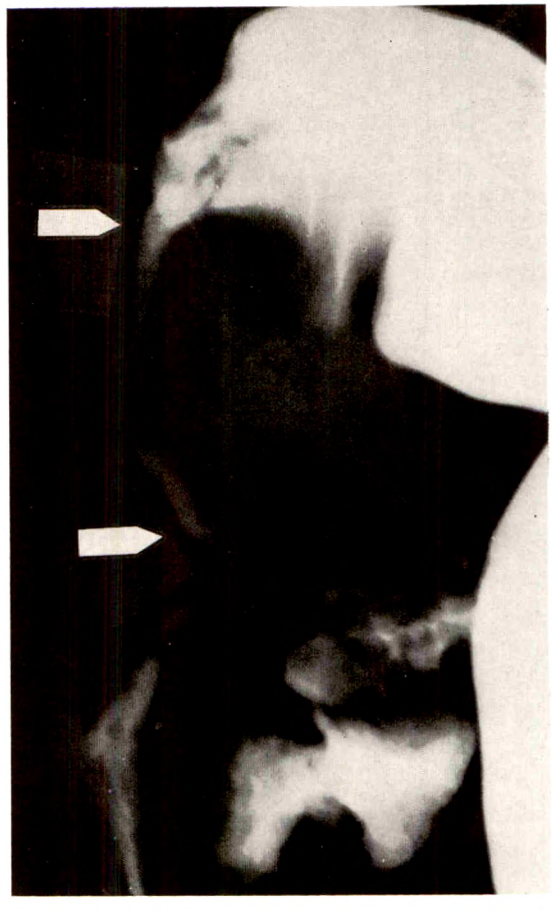


Fig. 2. Detail of stenotic segment, showing funnel-shaped margin and intact mucosa.




Fig. 3. Primary scirrhous carcinoma of colon, showing marked thickening of rigid bowel wall, with apparently intact mucosa.

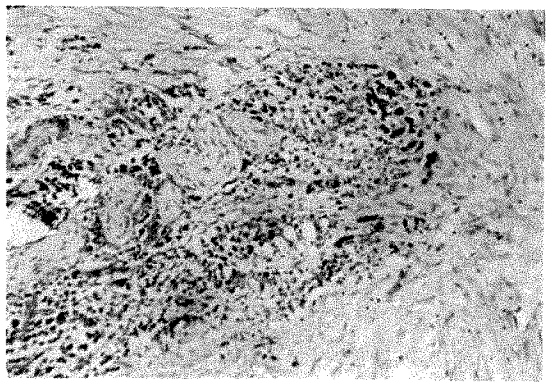


FIG. 4. Primary scirrhous carcinoma of colon, showing sparse tumor cells and marked fibrosis, simulating an inflammatory process.

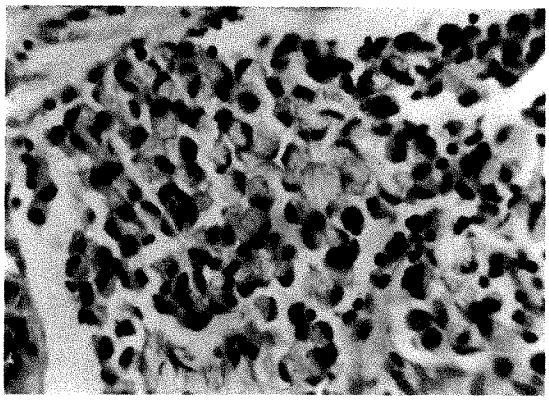


FIG. 5. Characteristic "signet ring" tumor cells, as seen in primary lesion, regional lymph nodes, and in Krukenberg type ovarian metastases.

and 9 cm. in length within the mesentery. The margins of the lesion were ill-defined, and both the bowel and mesentery were rigid and inflexible.

The mucosal surface was markedly edematous and swollen on the proximal side of the lesion. Centrally, the normal mucosal pattern was completely obliterated for a distance of 4.0 cm., where the mucosa was fixed, reddened, granular and superficially ulcerated. The thickened bowel wall and associated mesentery measured 16 mm. in thickness.

Microscopic examination showed primary scirrhous carcinoma of the colon with infiltration of the mesentery and metastases (100 per cent) to regional lymph nodes (Fig. 4 and 5).

Postoperatively, ascites developed, and was unsuccessfully treated with nitrogen mustard.

Autopsy was performed on April 25, 1965, 7 months after surgical resection, and 10 months after the onset of symptoms. The breasts were small, but otherwise normal. The stomach was likewise negative for primary carcinoma. Recurrent carcinoma was present in the sigmoid anastomosis (Fig. 6), presacral retroperitoneum, left ureter, and multiple adherent loops of ileum.

Metastases were present in both lungs as well as in the periaortic, lumbosacral and hilar lymph nodes. The ovaries showed bilateral massive metastatic tumors of Krukenberg type (Fig. 6). Chronic inanition, hypoproteinemia, marked ascites and massive bilateral hydrothorax with compression atelectasis were also recorded at postmortem examination.

## ROENTGENOGRAPHIC FEATURES AND DIFFERENTIAL DIAGNOSIS

The roentgenographic diagnosis, like the proctoscopic diagnosis,[8] of primary scirrhous carcinoma of the colon, is fraught with difficulty as its roentgenographic appearance either simulates the changes expected with inflammatory diseases of the colon, or the changes of a variety of other lesions involving the bowel wall.

The usual roentgen criteria of a primary malignancy of the bowel—intraluminal mass, mucosal ulceration, a short segment of involvement, and sharply demarcated hooked margins—are, in general, all absent in primary scirrhous carcinoma of the colon, making it difficult to suggest this diagnosis on the roentgenographic report.

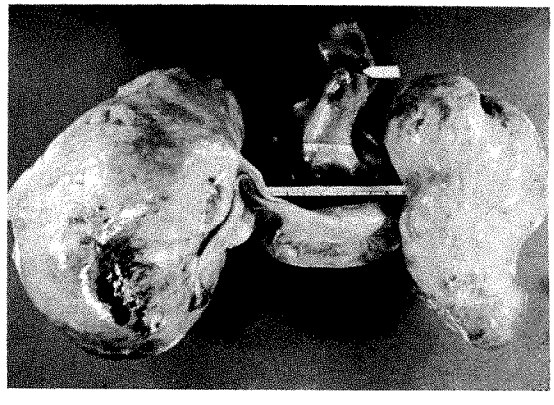


FIG. 6. Scirrhous carcinoma of colon, recurrent at sigmoid anastomosis (arrow), is dwarfed by huge Krukenberg metastases, which replace both ovaries.

For the radiologist to recognize this uncommon lesion when encountered, it behooves him to be acquainted with its existence and its roentgen features. Armed with this knowledge, and with the facts that this lesion often occurs in young individuals,[2] and most commonly in the rectum or sigmoid,[7] it is quite possible for him to include this lesion in the differential diagnosis.

The roentgenographic features include the absence of an intraluminal mass, and, except in a few instances,[7] the absence of mucosal ulceration. The mucous membrane tends either to be effaced by the intramural tumor, or the folds are thickened. The ends or margins of the lesion are tapered, more in the fashion of a benign lesion than a malignant one. Usually, quite a long segment is involved, and this is extremely unusual for a primary carcinoma, although it is not so for a primary lymphoma of the colon. Some retrograde obstruction to the flow of barium usually occurs, but it is rarely complete, as is so often the case with an acute stenosing diverticulitis.[7] Moreover, as Wolf and Marshak[7] have noted, there is less irritability than one might expect with an inflammatory process of comparable size.

Our case presents almost all of the features (Fig. 1 and 2) noted above. The classic criteria for primary carcinoma of the colon were absent, and obstructing intramural lesions were listed in the differential diagnosis.

Primary scirrhous carcinoma of the colon is, of course, closely simulated by metastatic scirrhous lesions secondary to carcinoma of the stomach, or less often, the breast. However, metastatic lesions are usually in the transverse colon, rather than the rectosigmoid, and are often multiple; this emphasizes the importance of an upper gastrointestinal study to exclude a gastric primary, especially since metastatic scirrhous carcinomas of the colon[1] outnumber primary scirrhous lesions by a ratio of about 10:1.

Primary scirrhous carcinoma of the colon must also be differentiated from segmental ulcerative colitis, a differentiation which is further obscured by the fact that scirrhous carcinoma often develops as a complication of long-standing chronic ulcerative colitis.[2,3,6] In addition, diverticulitis, granulomatous colitis, involvement of the bowel wall by endometriosis, and segmental infarction of the colon could all simulate primary scirrhous carcinoma.

Finally, if one encountered a lesion which involves primarily the wall of the colon without intraluminal mass or mucosal ulceration, with tapered edges and extending over a fairly long segment of rectosigmoid especially in a younger person, and if there is evidence of retrograde obstruction to the flow of barium due to narrowing of the lumen, associated with rigid walls resulting in a fixed appearance of the involved segment, then, one should seriously consider primary scirrhous carcinoma in the differential diagnosis, particularly if there is no known primary carcinoma elsewhere.

SUMMARY

Primary scirrhous carcinoma of the colon is discussed as a rare but distinctive lesion, with characteristic roentgenologic features.

An autopsied case is reported in a 24 year old woman, fatal less than 1 year after onset. The preoperative differential diagnosis is challenging, but not impossible.

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For permission to report this case, the authors are indebted to Helen Hart, M.D., Student Health Service, University of California, Santa Barbara.

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# COLONIC PERICARDIAL FISTULA*

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THE use of segments of colon to replace or bypass obstructing esophageal lesions was introduced in 1911 by Vulliet in France and Kelling[6] in Germany. In 1921 Lundblad[8] described the earliest successful use of colon for esophageal substitution in children. It has only been since the mid 1950s that intrathoracic colon for esophageal bypass has gained widespread usage in thoracic surgery. The purpose of the authors is to describe a case of colonic transplant necrosis, with resultant mediastinitis, pericarditis, and colonic pericardial fistula. Progressive enlargement of the cardiac silhouette and a pneumopericardium were the chief roentgenographic features.

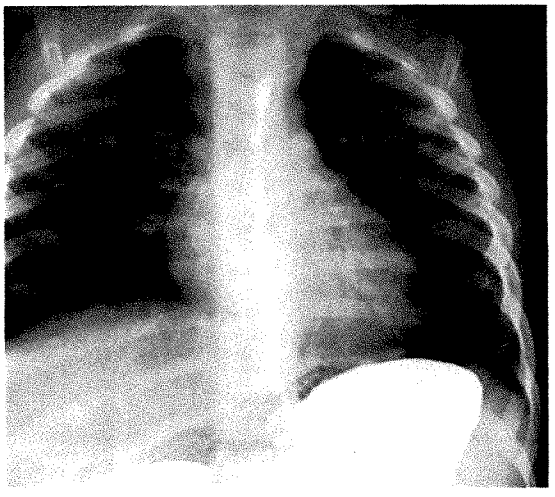


FIG. 1. Esophagogram. There are ulcerations of the esophagus with marked narrowing extending from the aortic arch to just above the cardia.

## ILLUSTRATIVE CASE

A 2 year old male was noted to have burns of the mouth following lye ingestion. For the next 3 weeks he was able to take only oral fluids. The first admission to St. Louis Children's Hospital was 1 month after the lye ingestion, at which time the oral cavity had healed and there were no abnormal physical findings. An esophagogram 1 day after admission revealed marked chronic ulcerative esophagitis, with narrowing extending from the aortic knob to 2 cm. above the gastroesophageal junction (Fig. 1). Esophagoscopy 2 days later showed posterior esophageal wall granulation tissue 13 cm. from the mouth and circumferential granulations at 16 cm., through which No. 10–14 bougies could be passed. The patient continued on a liquid diet and was discharged to be re-evaluated in 6–8 weeks for possible esophageal bypass. During the next month the child experienced progressive dysphagia.

Three days prior to the second admission (2 months post lye ingestion), the patient developed fever and a productive cough. A chest roentgenogram demonstrated a confluent right lower lobe pneumonia. Under direct laryngoscopy a catheter was passed through the esophagus into the stomach. One week later esophagoscopy showed a marked stricture 17 cm. from the mouth. Two weeks after esophagoscopy a small abscess cavity appeared in the center of the right lower lobe infiltrate. The cavity and pneumonia progressively cleared.

A right colopyriformostomy and transverse cologastrostomy were performed. The colon segment received its blood supply from the colic branch of the ileocolic, right colic and middle colic arteries. The segment was placed retrosternally. Intestinal continuity was reestablished by an ileocolostomy. A gastrostomy was performed, and a chest tube inserted into the right pleural space.

The postoperative course was characterized by intermittent low grade fever and some difficulty with fluid maintenance. Six days after surgery a chest roentgenogram showed enlargement of the heart shadow. Pericarditis secondary to mediastinitis was postulated (Fig. 2). The patient developed anasarca with marked facial involvement. At the same time dyspnea, rales and hepatomegaly were noted. An electrocardiogram was consistent with myocardial disease or pericarditis. Previous electrocardiograms had been normal.

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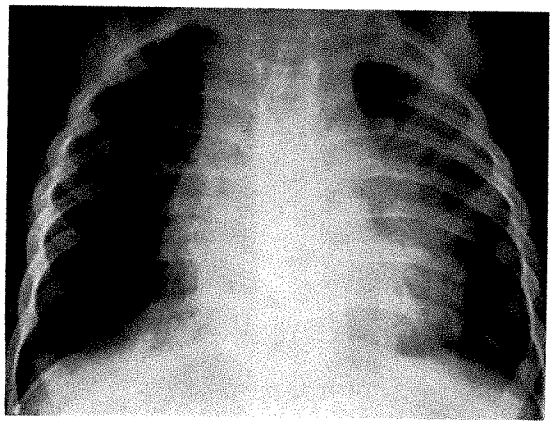


FIG. 2. Posteroanterior chest roentgenogram showing increase in cardiac silhouette, and a diffuse pneumonia involving all lobes.

Chest roentgenograms 4 days later again demonstrated a large cardiac silhouette. Pneumopericardium and bilateral pneumonic infiltrates were evident. Perforation of the colon was postulated and a hypaque swallow study performed. Contrast material passed through the colon transplant which had marked mucosal irregularity down to an area just above the diaphragm, where the contrast medium flowed into the pericardial cavity (Fig. 3 and 4). Roentgen diagnoses were: colonic pericardial fistula (secondary to necrosis of the colon transplant) and pneumonia.

The patient was taken from the Radiology Department directly to the operating room. The entire colon segment from the pyriform sinus to the stomach was observed to be necrotic. There were marked mediastinitis and pericarditis. Fluid was present in the peritoneal cavity. The entire transplanted colon segment was removed and the mediastinum debrided. The opening into the pericardial cavity was enlarged and the pericardium sutured to the anterior chest wall. The patient expired following closure of the incisions.

Autopsy revealed an acute and chronic esophagitis with marked stricture formation, bilateral pleural effusions and pneumonia in all lobes, greatest in the right lower lobe, a 1 cm. abscess cavity in the right lower lobe, and acute, partially organized fibrinous pericarditis.

## DISCUSSION

There has been increased use of the colon for esophageal replacement or bypass in recent years. The indications for transplantation of the colon are: progressive esophageal obstruction, either from malignancy or benign stricture; certain instances of esophageal atresia (with or without tracheo-esophageal fistula); and esophageal varices that have failed to respond to vascular shunting procedures.[14] Preference as to the use of either ascending or descending colon is dependent on the individual surgeon, but the anatomic length of the colon available on either side may be the deciding factor.[16] The advantage of the colon is that it has sufficient length along

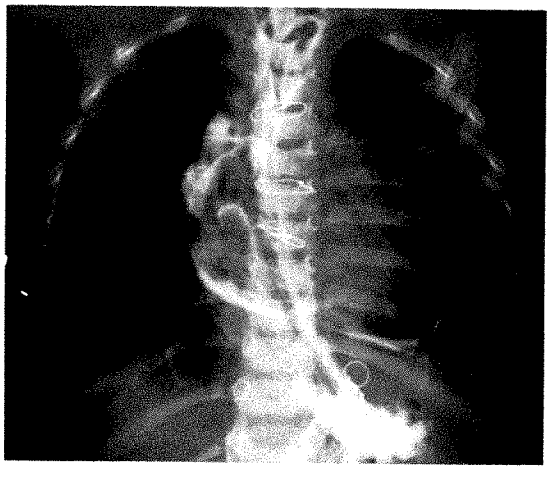


FIG. 3. Anteroposterior chest roentgenogram. Oral hypaque passes from the necrotic interposed retrosternal colon segment into the pericardial cavity. Air is also present in the pericardial cavity.

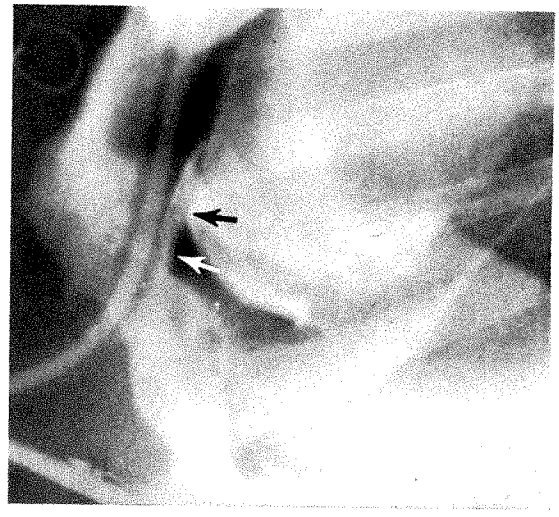


FIG. 4. Spot roentgenogram showing the colonic pericardial fistula (arrows). The colon is edematous, markedly ulcerated, and contains a tube.

with ease of mobilization, a diameter great enough that there is little likelihood of anastomotic stricture, an adequate blood supply, and resistance to acid peptic digestion.[2,9,11,13] The colon segment functions primarily as a passive conduit with gravity flow and may undergo progressive dilatation.[1,10,16] Gross states that the procedure should not be done before 1 year of age.[5] The segment elongates with growth of the child.[12] The intrathoracic colon transplant can be placed in the pleural cavity or in the retrosternal space. It may also be put in the retrocardiac position with retention of the esophageal hiatus.[2]

Complications include leakage or dehiscence at the esophagocolic or gastrocolic anastomosis. Small leaks at the cervical anastomosis can usually be managed conservatively.[7,16] Obstruction may occur at the various anastomoses or as the colon passes through the diaphragm. There may be kinking and obstruction of the redundant intrathoracic colon. Additional reported complications include: superficial wound and intrathoracic infections; recurrent malignancy; aspiration pneumonia; pulmonary embolism; air-way obstruction; pneumothorax; recurrent nerve injury; cystic dilatation of esophageal remnant; gastric atony; regurgitation of material from stomach; excessive oropharyngeal secretions; and diarrhea. Drug reactions, infectious hepatitis, cardiac failure, and myocardial infarction have also occurred.[1,4,13,14,16] Arterial insufficiency or venous obstruction may lead to bowel necrosis and perforation, poor anastomotic healing, or intravascular thrombosis. When necrosis appears late, the patient may have progressive dysphagia, and the bowel transplant may become a fibrous cord.[4] The necrosis can be localized or involve the entire transplanted colon.

In this reported case, necrosis of the colon segment resulted in mediastinitis, pericarditis and eventually a colonic pericardial fistula. The pericardium was not knowingly opened during the first surgical procedure.

A sudden increase in heart size should suggest pericarditis and associated mediastinitis in the absence of acute cardiac failure in colon transplant patients. Development of a pneumopericardium suggests a diagnosis of colonic pericardial fistula secondary to colon necrosis. The diagnosis should be confirmed by a contrast study with water soluble iodine containing contrast media. Other causes of pneumopericardium include recent open heart surgery, trauma, malignant tumors, gas-forming organisms, perforation into the pericardium by inflammatory foci from surrounding structures, perforation of a gastric ulcer into the heart directly or by formation of a subphrenic abscess, and dissection through the diaphragm into the pericardium. A primary suppurative pericarditis may perforate into the surrounding lung.[15] Pulmonary cavitation, pneumothorax adjacent to the heart, or mediastinal emphysema may be confused with pneumopericardium on plain chest roentgenograms.[3] Pneumopericardium can be distinguished by a shift in the air away from the dependent side on decubitus chest roentgenograms. Air in the pericardium does not rise above the aortic knob with the patient upright. The lateral chest roentgenogram can usually differentiate pneumomediastinum in which the air collects principally in the anterior mediastinum and along the great vessels. The thymus may be elevated away from the great vessels with pneumomediastinum in children.

## SUMMARY

A case of a child with necrosis of a colon transplant and resultant mediastinitis, pericarditis, and colonic pericardial fistula is reported. This was manifested roentgenologically by progressive enlargement of the cardiac silhouette and a pneumopericardium.

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VOL. 101, NO. 2

# EXTRAPERITONEAL PERFORATIONS OF THE GASTROINTESTINAL TRACT*

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MOST roentgenologists are constantly watchful for "free" or intraperitoneal air on reviewing roentgenograms of the abdomen. Intraperitoneal air is detectable in small amounts in roentgenograms obtained in the erect or decubitus positions and in larger amounts in the recumbent. Seldom is this intraperitoneal air overlooked. Radiologists who have reviewed roentgenograms on diagnostic retroperitoneal gas studies are familiar with the appearance of extraperitoneal air (or gas). It is frequently overlooked, however, when it is not expected, particularly when abdominal roentgenograms are obtained because of abdominal pain. At that time the attention of the roentgenologist may be diverted by numerous gas-filled loops of bowel. There have been recent reports of extraperitoneal perforations of the duodenum and rectum. However, no radiologic and only a few other references have appeared in the last 20 years pertaining to extraperitoneal perforations of the bowel between the ligament of Treitz and the rectum.

The purpose of this report is to present 5 cases of extraperitoneal perforation of the bowel together with the clinical and roentgen features. In 3 of these, extraperitoneal perforation was of the colon (above the rectum).

## REPORT OF CASES

CASE I. A 66 year old woman was admitted to the hospital with left lower abdominal cramps. She had had some left lower quadrant discomfort for 6 weeks, but this had recently become worse. For the 3 days preceding admission, diarrhea had been present. On admission the patient's temperature was 100° F. The abdomen was distended and tender and a vague mass in the left abdomen was thought to be a fecal-filled colon. The results of the stool examination for ova and parasites were negative. The clinical diagnosis was diverticulitis or carcinoma of the left colon. Plain roentgenograms of the abdomen were obtained (Fig. 1, *A* and *B*). One week later a barium enema examination was performed (Fig. 1*C*). The patient was treated by a diverting transverse colostomy and retroperitoneal drainage, and recovered satisfactorily. The barium enema examination was repeated 7 months later (Fig. 1*D*). Following this examination, the left colon was explored surgically. A small portion of the left colon, which the surgeon considered slightly thickened, was resected, but histologically this proved to be of normal colon. The colostomy wound was eventually closed and continuity of the bowel was re-established without complications. Although the cause of the perforation was not clearly established, the surgeon expressed the opinion that it was the result of a perforated solitary diverticulum, not identified at the exploration 7 months later.

CASE II. A 67 year old woman was admitted to another hospital on March 1, 1964 with sudden onset of lower abdominal pain and a temperature of 103° F. Clinically the condition was diagnosed as sigmoid diverticulitis and the patient was treated with antibiotics which resulted in only temporary improvement. On March 6, 1964, she was transferred to the San Francisco General Hospital. On examination the patient was confused and had a temperature of 101° F. Because of generalized abdominal distention with guarding and rebound tenderness over the lower abdomen, plain abdominal roentgenograms were obtained (Fig. 2, *A* and *B*). Laparotomy disclosed marked inflammation of the sigmoid colon, which was covered by a fibrinous exudate. Extensive gas in the sigmoid mesentery extended into the retroperi-

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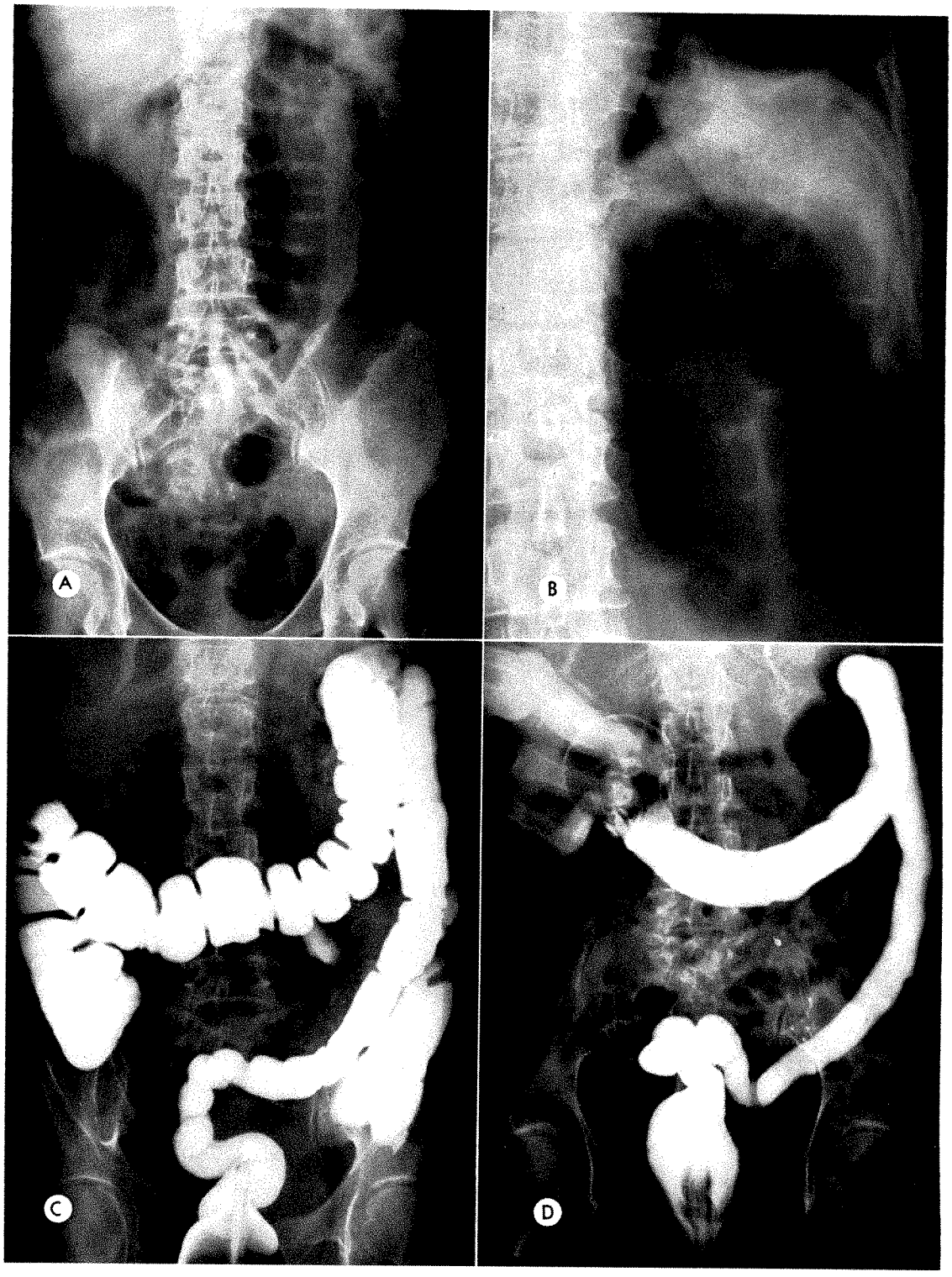



FIG. 1. Case 1. (*A* and *B*) Plain roentgenograms of abdomen. (*A*) Supine roentgenogram shows that the psoas shadow and the left extraperitoneal fat line are obliterated. Numerous small shadows of gas surround the upper descending colon. (*B*) Erect roentgenogram showing left upper quadrant. No "free" or intraperitoneal gas is seen. The small gas bubbles surrounding the upper descending colon are better shown. Note

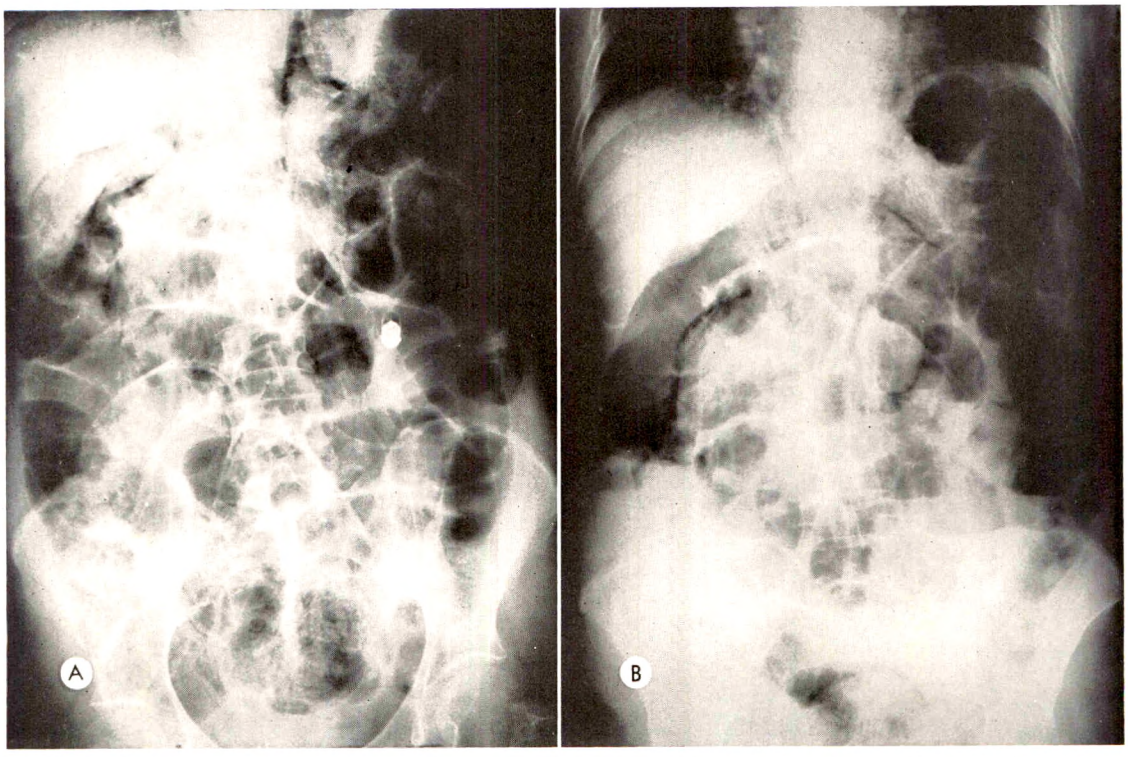



Fig. 2. Case II. (*A*) Supine and (*B*) erect abdominal roentgenograms. Numerous loops of both small and large bowel are distended by gas. No intraperitoneal gas is present. Retroperitoneal gas outlines both kidneys and both adrenal regions. The opacity seen to the left of the third lumbar vertebra in the supine roentgenogram and projected over the left ilium in the erect roentgenogram was a flashlight bulb that the patient had ingested.

toneal space. No pus was noted in the retroperitoneum, however, and the gas was thought to be the result of a perforation of the sigmoid and not of gas-producing organisms. A diverting transverse colostomy was performed and the marked distention of the small bowel was treated by decompression with a Leonard tube. A foreign body, which had been observed on the roentgenograms, proved to be a small flashlight bulb in the small bowel. Apparently the patient had ingested this bulb during her confused state. Although the sigmoid was not explored, the perforation was possibly caused by an additional ingested foreign body in the sigmoid. Postoperative bronchopneumonia developed and the patient died in cardiovascular collapse 21 days after the operation. Necropsy was not performed.

Case III. A 61 year old man was admitted to the hospital with a 6 day history of lower abdominal pain and bloody stool. The patient had noted increasing constipation for the previous 6 months. On physical examination, a tender mass was palpated in the left lower abdominal quadrant. The results of laboratory studies were normal except for a white blood cell count of 11,600. Abdominal roentgenograms were ob-

pleural fluid on the left and atelectasis in the base of left lung. The extraperitoneal perforation was overlooked on the initial interpretation of these roentgenograms. (*C*) Barium enema roentgenogram 7 days later. Barium has extravasated into the retroperitoneal space from a perforation in the lower descending colon. (The 3 densities in the right upper quadrant represent gallstones.) (*D*) Barium enema roentgenogram 7 months later. The colon is opacified from the rectum to the transverse colostomy. A small amount of barium remains in the retroperitoneal space from the previous perforation. No new extravasation has occurred.

tained (Fig. 3, *A* and *B*). On the first hospital day an abscess of the left lower abdominal wall was incised and drained. On culture of the abscess, coliform organisms were obtained. A laparotomy on the tenth day revealed a mass in the sigmoid colon. A left colectomy was performed. The pathologic diagnosis was that of a perforating carcinoma of the sigmoid colon.

CASE IV. A 79 year old woman was admitted with diffuse abdominal pain of 12 hours' duration. Nausea and intolerance to fatty food had been noted for the previous 3 months. The physical examination disclosed a temperature of 100° F. The abdomen was slightly distended, with tenderness and guarding in its upper portion. Clinically, the condition appeared to be one of acute cholecystitis. A nasogastric tube was passed and suction begun. An upright abdominal roentgenogram (not shown) revealed no evidence of free air. An intravenous cholangiography was performed (Fig. 4, *A* and *B*). On delayed roentgenograms (not shown) a normal common duct and gallbladder were apparent. The patient was given intravenous fluids and antibiotics, which improved the abdominal signs temporarily. The clinical impression was changed to that of possible bacterial endocarditis and, while this was being investigated, the patient suddenly became hypotensive and died. On necropsy, a perforated ulcer was present in the duodenum 2 cm. distal to the pylorus, with a focal area of organizing peritonitis around the ulcer.

CASE V. A 28 year old man was involved in an automobile accident in which the upper abdomen was struck by the steering column. Almost immediately, severe, generalized abdominal pain was experienced. On examination one hour after the accident, diffuse abdominal tenderness was present. No masses were pal-

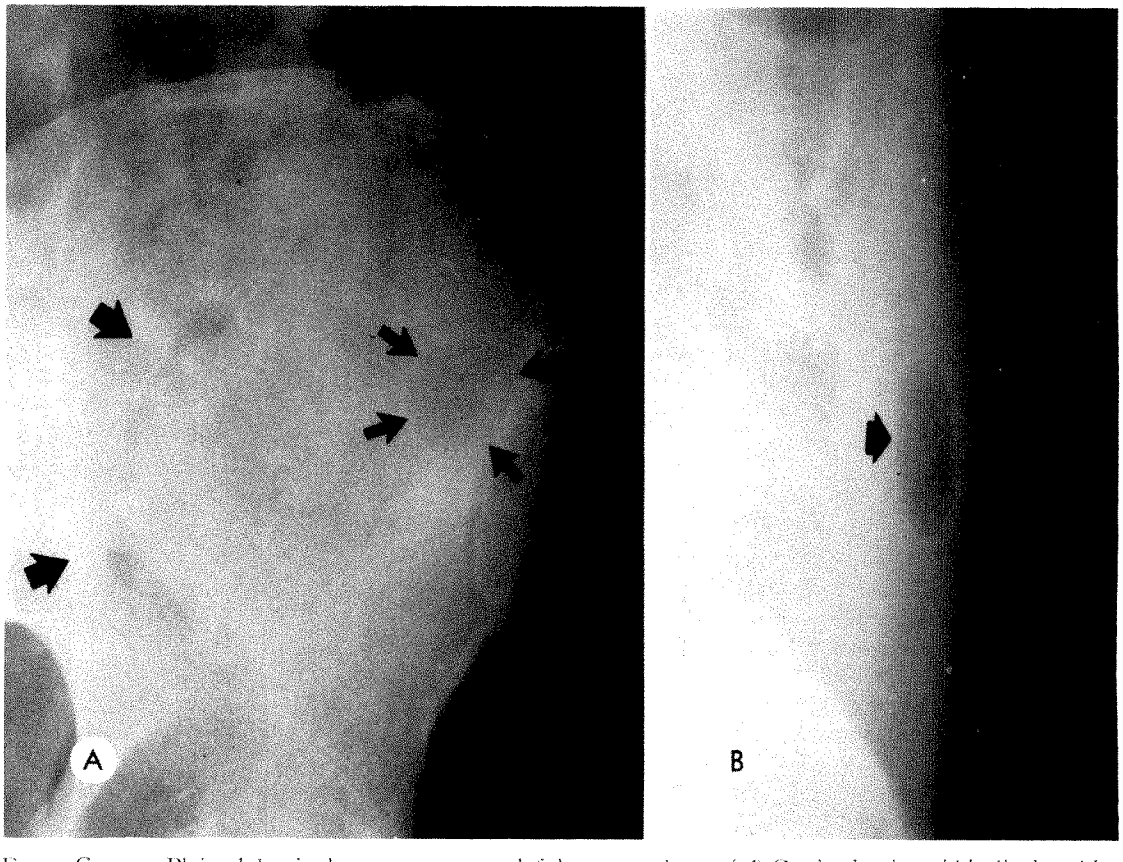



FIG. 3. Case III. Plain abdominal roentgenograms, left lower quadrant. (*A*) Gas in the sigmoid is displaced by a mass (2 large arrows). A collection of gas is lateral to the sigmoid and in the region of the mass (4 small arrows). (*B*) Tangential roentgenogram. The collection of gas (arrow) is seen to be extraperitoneal in the abdominal wall.

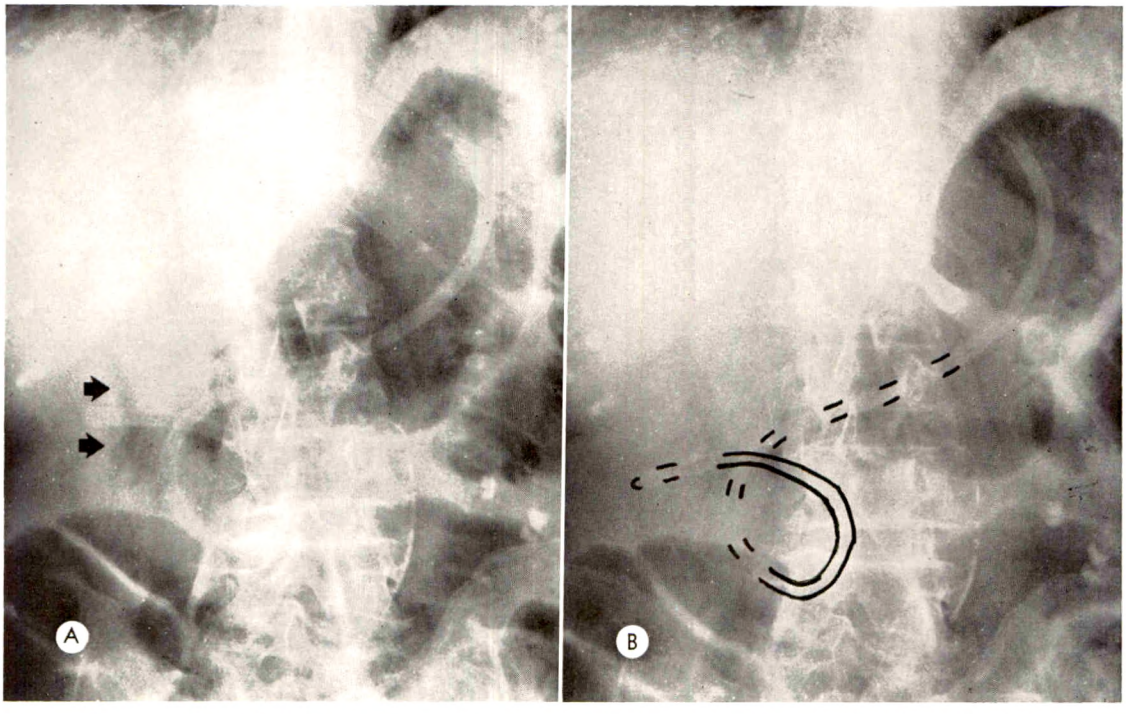



Fig. 4. Case IV. Abdominal roentgenograms obtained during an intravenous cholangiography. The biliary duct system has not yet opacified. Note the unusual position of the nasogastric tube, which appears coiled in the second portion of the duodenum with the tip of the tube extending toward the right. In *A* an unusual collection of gas is seen near the tip of the tube (arrows). In *B* the tube has been outlined in ink to better demonstrate it over the spine. The right psoas shadow is obliterated. The diagnosis of an extraperitoneal perforation of the duodenum was not made on the initial interpretation of these roentgenograms.

pable and peristalsis was absent. The abdomen became board-like within 2 hours. Roentgenograms of the abdomen obtained in the interval gave evidence of retroperitoneal gas (Fig. 5). On laparotomy after these roentgen examinations, a 2.5 cm. retroperitoneal perforation was found in the third portion of the duodenum. The perforation was repaired and a drain left in place. Recovery was satisfactory and the patient was discharged on the thirteenth postoperative day.

## DISCUSSION

The clinical entity of subcutaneous emphysema secondary to perforation of the gastrointestinal tract has long been known. The term, "surgical emphysema," is often applied. To be clinically apparent, large amounts of gas need to be present which will often need to dissect for a considerable distance from the site of perforation. Thus, clinical evaluation is less likely to be accurate than are roentgen studies in the detection of extraperitoneal perforations of the intestinal tract.

Oetting *et al.*[7] reviewed the world literature for cases of subcutaneous emphysema secondary to gastrointestinal perforation from 1853 to 1953 and collected 34 cases. The roentgen findings were not described. The sites of appearance of the emphysema were designated as the neck, anterior abdominal wall, and perineum. The authors noted correlation between the site of intestinal perforation and the appearance of the emphysema. Perforations of gastric and duodenal ulcers presented an emphysema in the neck. Emphysema from perforations of the colon and small bowel appeared in the anterior abdominal wall, and anorectal lesions caused emphysema in the perineum. In 2 additional cases, subcutaneous air was seen on roentgenograms in the anterior abdominal wall of one and in the retroperitoneal space in the other. The

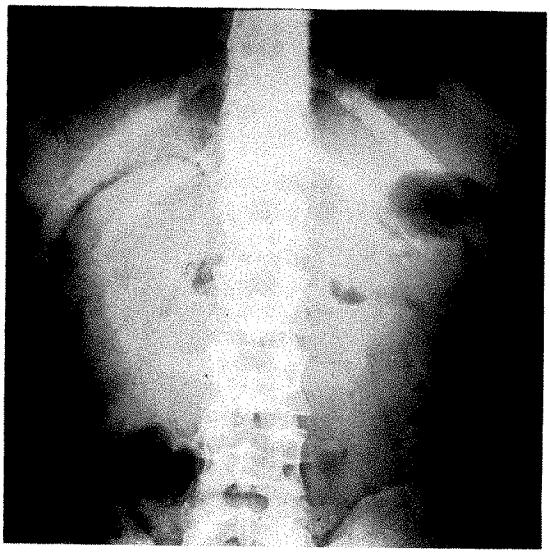


FIG. 5. Case v. Upright abdominal roentgenogram. Retroperitoneal gas outlines the right kidney. No evidence is seen of intraperitoneal gas under the diaphragm. The right psoas shadow is obliterated.

roentgen appearance in extraperitoneal perforations of the duodenum and rectum has been described several times. Sperling and Rigler,[9] in 1937, described retroperitoneal gas in traumatic rupture of the duodenum. Jacobson and Carter[4] reviewed 19 cases of intestinal rupture resulting from nonpenetrating abdominal injury. Most of these ruptures were found proximal to the ligament of Treitz and some of the perforations were extraperitoneal.[1] Few reports have appeared of the roentgen findings of extraperitoneal perforations of the bowel that were situated between the ligament of Treitz and the rectosigmoid junction. Lewis[6] described a single case of spontaneous perforation of the sigmoid colon in which a large amount of retroperitoneal gas was seen on abdominal roentgenograms.

In the present report, 5 cases of extraperitoneal perforation of the intestinal tract are described. In Case I a perforated solitary diverticulum of the descending colon may have been the cause. In Case II, an ingested foreign body was suspected.[2] The perforations in Cases III, IV, and V, were caused respectively by carcinoma, peptic ulcer, and trauma. Extraperitoneal perforations may occur from retroperitoneal portions of the gastrointestinal tract.[5,8] Such instances are illustrated in the descending colon by Case I and in the retroperitoneal duodenum by Cases IV and V. They may occur also from intraperitoneal portions of the gastrointestinal tract by perforation into the mesentery, with extraperitoneal dissection, as in Cases II and III. When the hole in the bowel straddles the junction of an intraperitoneal and extraperitoneal portion of the gastrointestinal tract, intraperitoneal and extraperitoneal perforations may occur simultaneously. These cases do not ordinarily present diagnostic problems, however, and the clinical and roentgen signs of intraperitoneal perforation are usually readily apparent and lead to surgical exploration.

SUMMARY

Extraperitoneal perforations of the gastrointestinal tract, although uncommon, may be fatal if not recognized.

The clinical and roentgen features of 5 cases of extraperitoneal perforation are described. In all, one or more of the following roentgen signs were seen: extraperitoneal gas, obliteration of normal extraperitoneal fat lines, and abnormal position of nasogastric tube. Usually an alert roentgenologist will be able to provide the diagnosis earlier than can the clinician.

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# A COMPARISON OF THE HORIZONTAL AXIAL VIEW AND THE CLASSICAL CHASSARD-LAPINE VIEW OF THE SIGMOID COLON

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THE Chassard-Lapine view of the sigmoid colon has been found to be very useful in disclosing otherwise unsuspected lesions in a redundant sigmoid. However, this view is frequently omitted in the elderly, less supple patient because of difficulty in positioning, the tendency to lose barium from the sigmoid, and the danger of the patient falling from the edge of the table during the roentgenographic procedure. To preclude these difficulties, many radiologists utilize a horizontal prone view with 30°–35° caudal angulation of the central roentgen ray as a substitute for the sitting-up projection. A recent publication by Dysart and Steward[1] stressed the value of this view and noted that it had apparently been in use for many years but had been neglected in the medical literature.

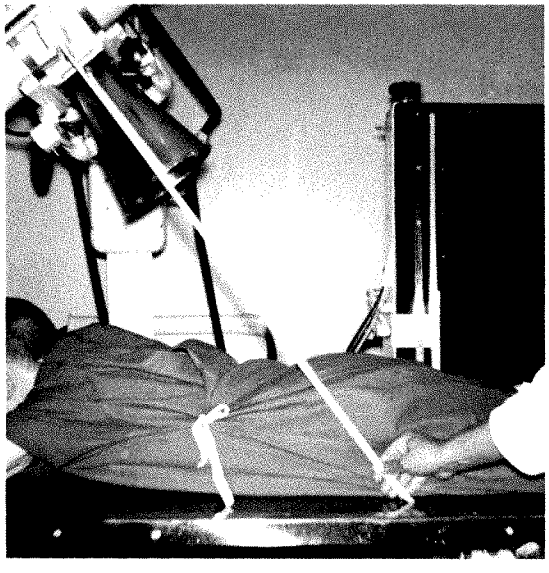


FIG. 1. The horizontal axial view is taken with the central roentgen ray centered on the lumbar spine at the level of the iliac crests, the tube being angled 30°–35° caudad. Additional projections with slight right and left oblique positioning can be useful in very difficult cases but are not routinely obtained.

This horizontal axial view was taught to one of us (B. B. G.) by Doctor Robert Powers in Palo Alto, California, about 1950.

## MATERIALS AND METHODS

Both the classical sitting Chassard-Lapine and the horizontal axial view of the sigmoid colon were obtained in 50 patients with varying degrees of redundancy of the sigmoid colon. The horizontal axial view is obtained (Fig. 1) by leaving the patient in the prone position with the central roent-



FIG. 2. The Chassard-Lapine view is taken with the patient sitting, bent forward and the central roentgen ray vertical. Slight anterior angulation of the tube may be necessary with the less supple patient.

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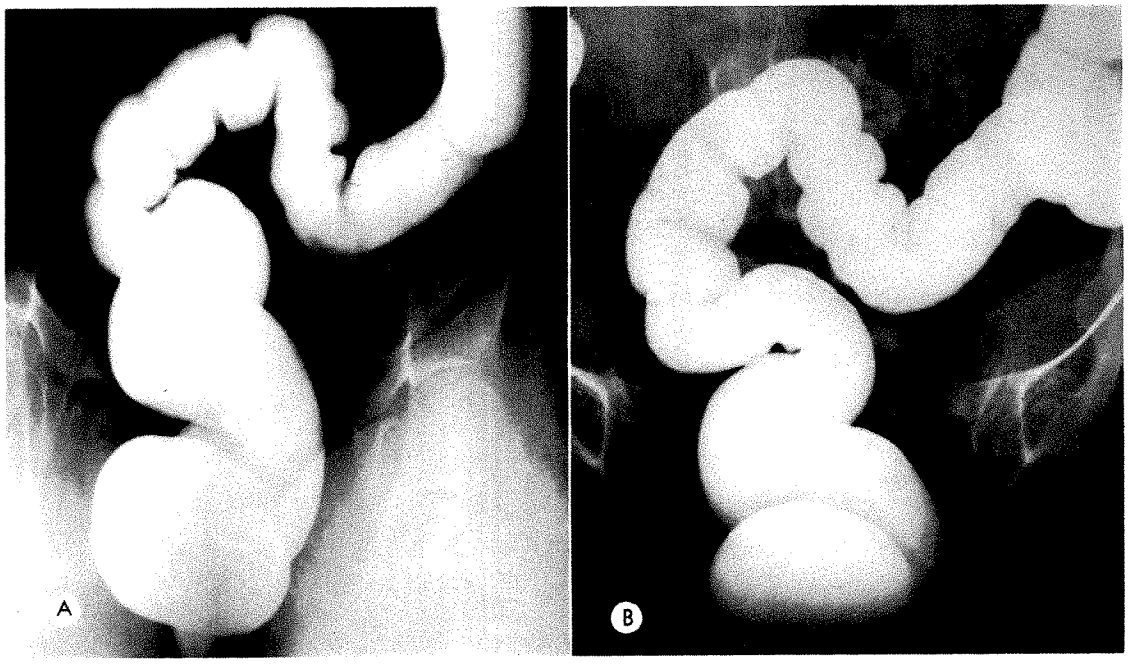



FIG. 3. A "simple" sigmoid well visualized in both the horizontal axial view (*A*) and the Chassard-Lapine view (*B*).

gen ray centered on the lumbar spine at the level of the iliac crests, the tube being angled 30°–35° caudad. The Chassard-Lapine view (Fig. 2) is obtained in the usual fashion with the patient sitting on the edge of the table bending forward as far as possible with the central roentgen ray vertical. The views are quite comparable as far as the

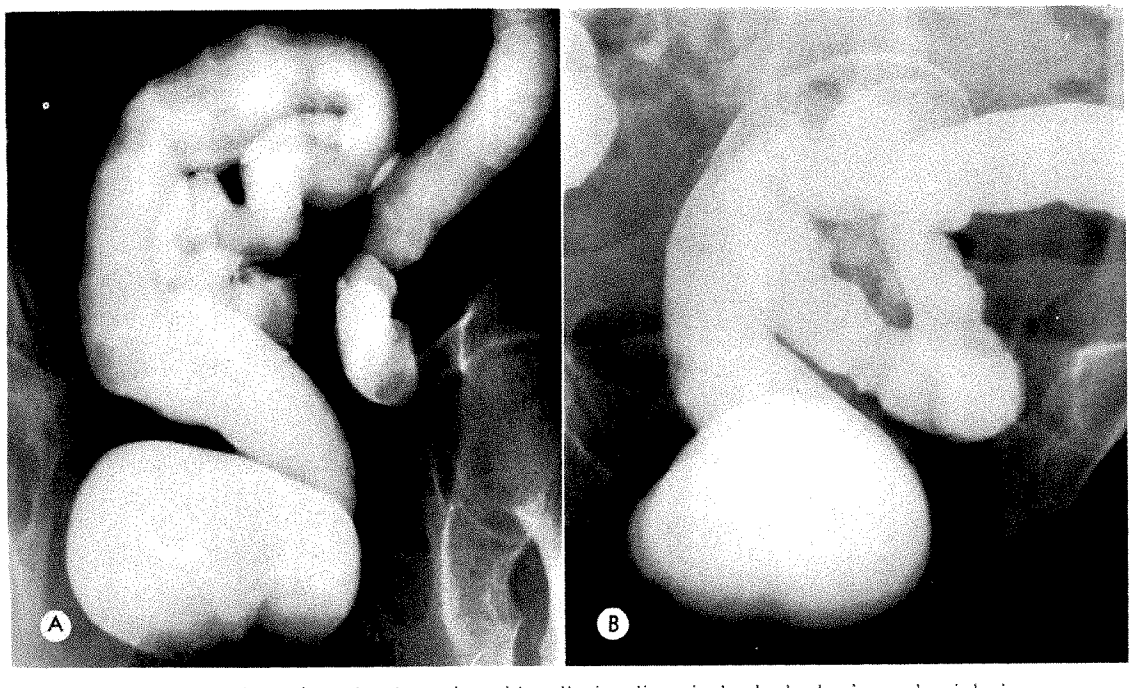



FIG. 4. A moderately redundant sigmoid well visualized in both the horizontal axial view (*A*) and the Chassard-Lapine view (*B*).

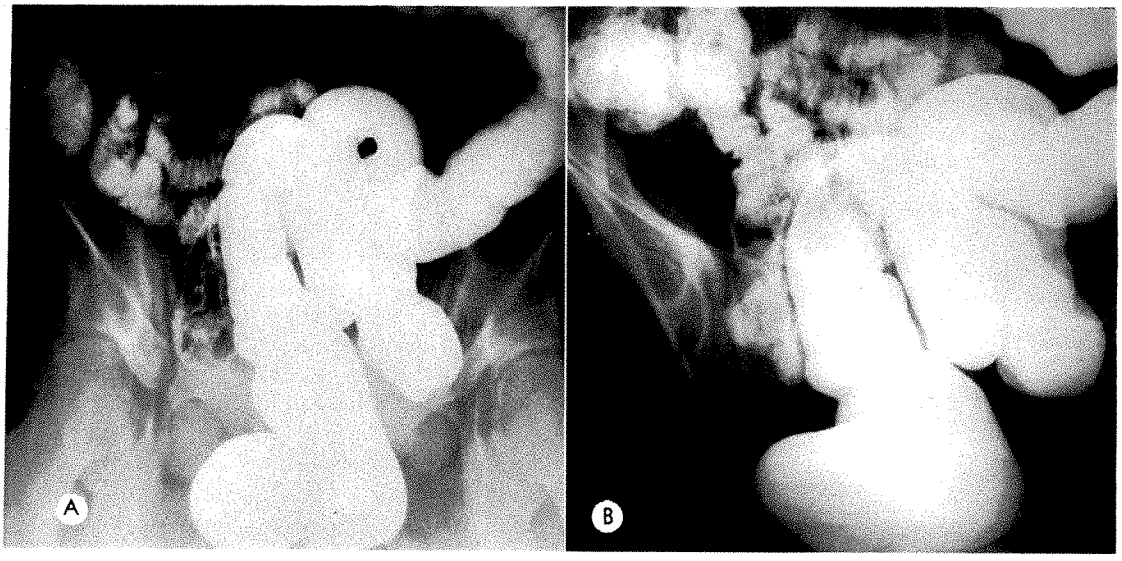



FIG. 5. A moderately redundant sigmoid in which both the horizontal axial view (*A*) and the Chassard-Lapine view (*B*) fail to project all loops free of each other.

relation of the central roentgen ray to the spine and pelvis is concerned. The technical factors are similar for the 2 projections, (120 kv., 200 ma., 0.2–0.6 sec.).

The roentgenograms were then evaluated as to adequacy of visualization of the sigmoid with interest primarily directed to which view projected the redundant loops free of each other with the greatest consistency.

## RESULTS

The 2 projections were judged to be essentially equal in "uncoiling" the sigmoid loops. Of the 50 cases, the 2 views were judged equal in 43 cases, the horizontal axial view was judged superior in 4 cases, and the sitting Chassard-Lapine projection was judged superior in 3 cases. The film density was felt to be slightly more consistent in the horizontal projection, and the Chassard-Lapine view was more frequently underexposed. This is no doubt due to the patient's varying ability to bend forward far enough to achieve the proper angle between the central roentgen ray and the spine.

Both projections were judged very useful and routinely visualized "simple" sigmoids (Fig. 3, *A* and *B*) and moderately redundant sigmoids (Fig. 4, *A* and *B*) well. Both views failed to project all loops free of each other in markedly redundant sigmoids (Fig. 5, *A* and *B*), but were judged very helpful in such cases due to improved visualization as compared with the routine projections and spot roentgenograms.

## SUMMARY

The horizontal axial view and the classical Chassard-Lapine view of the sigmoid colon have been compared and are believed to be equal in ability to separate redundant loops free of each other. The horizontal axial view is felt to be more easily obtained, especially in elderly, less supple patients.

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# PERFORATION OF AN ATRETIC COLON DURING BARIUM ENEMA EXAMINATION*

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PERFORATION of the colon in the newborn is uncommon. Atresia of the colon is quite rare. We are reporting a patient with colon atresia in whom the distal segment of the colon was perforated by a barium enema.

## REPORT OF A CASE

A male infant, product of a normal pregnancy, developed abdominal distention and vomited bile-stained material on the second day of life. Three meconium stools were described as normal. On physical examination there was abdominal distention and no bowel sounds. Twenty ml. of bile-stained material was aspirated from the stomach through a nasogastric tube.

Roentgenograms of the abdomen showed intestinal obstruction with a markedly distended loop of bowel in the right abdomen (Fig. 1, *A* and *B*). A barium enema study was performed using USP barium sulfate in the concentration recommended by the manufacturer. The barium container was placed approximately 2 feet above the table top. The distal colon was small from disuse and contained multiple tiny filling defects. There was no passage of contrast medium beyond the distal transverse colon for approximately 20 seconds. Then barium sulfate suddenly flowed freely into the peritoneal cavity (Fig. 2, *A* and *B*). The examination was terminated. The diagnosis was bowel obstruction with perforation of the colon. The patient shortly thereafter underwent abdominal exploration. The large air-filled structure on the right was the cecum and a portion of terminal ileum which had undergone volvulus. The distended hemorrhagic cecum and terminal ileum

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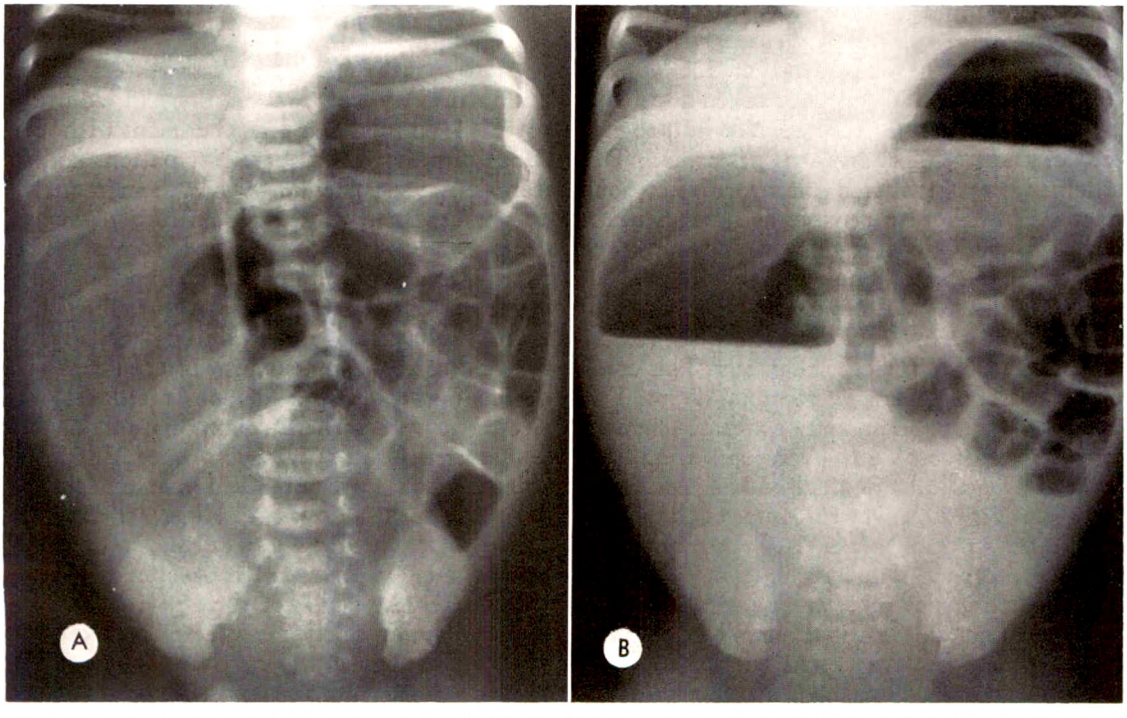



FIG. 1. (*A*) Supine and (*B*) erect abdominal roentgenograms showing evidence of bowel obstruction with a markedly dilated segment of bowel on the right.

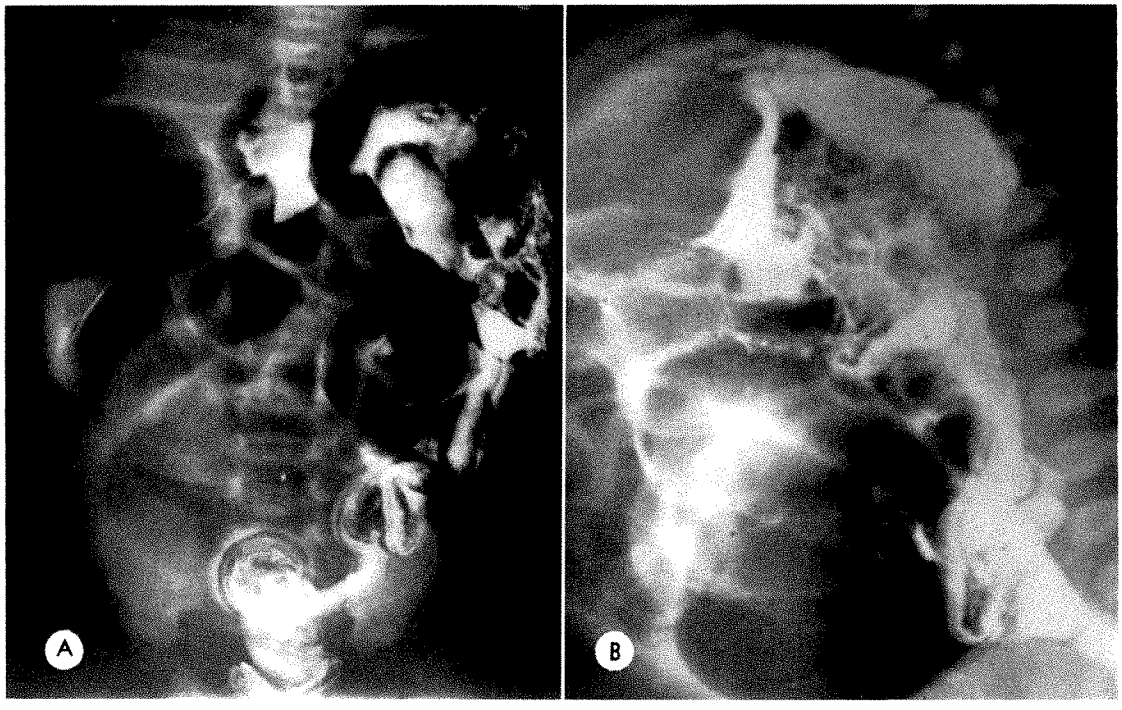



FIG. 2. (*A*) Anterior and (*B*) lateral views with barium sulfate having passed out through the atretic end of the distal transverse colon into the peritoneal cavity. The distal colon segment is small and unused.

were removed. There was complete absence of the colon between the cecum and the splenic flexure. Barium sulfate was leaking through a perforation in the blind end of the splenic flexure. The splenic flexure was taken down and the proximal 3 cm. resected. An end-to-end ileo-descending colostomy was performed. The abdomen was irrigated first with copious amounts of saline to remove as much barium sulfate as possible and then with 1 per cent kanomycin. A gastrostomy was performed.

The surgical specimen consisted of a distended hemorrhagic cecum which measured 14 cm. in length and 7 cm. in diameter. This communicated with 10 cm. of terminal ileum. The cecum and a portion of ileum contained hemorrhagic viscid meconium. A second specimen, the 3 cm. of blind proximal end of the distal colon segment, on microscopic examination showed thinning and absence of the muscularis in many areas. The perforation occurred through an area devoid of muscle.

The patient did reasonably well in the immediate postoperative period with the exception of frequent loose bowel movements and lack of weight gain. He had intermittent mild rectal bleeding and ileus. A roentgenogram of the abdomen 2 weeks after surgery showed residual barium sulfate in the peritoneal cavity (Fig. 3).

At 10 months of age he was readmitted with phimosis. Physical examination, in addition, revealed an undistended abdomen with evidence of previous surgery. The patient had an iron deficiency anemia with a hematocrit of 27 per cent and a hemoglobin of 7.7 gm. per cent. There was no clinical evidence of bowel obstruction. A barium enema and upper gastrointestinal examination again showed residual barium sulfate in the peritoneal cavity (Fig. 4). A stricture at the anastomosis of the ileum and descending colon was demonstrated. It was elected to resect the anastomosis and an abdominal exploration was performed. Innumerable adhesions were noted and as many as possible were broken down. The distal ileum was dilated to approximately 8 cm. in diameter while the colon measured approximately 3 cm. in diameter. A portion of the ileum and proximal descending colon was resected and an end-to-end anastomosis was carried out. The postoperative course was uneventful and the anemia corrected. The patient has been seen in the clinic and has done well for the past year.

## DISCUSSION

Perforation of the colon in the newborn period has numerous etiologies.[6,7] Inflammatory disease, distal obstruction, strangulation of the colon secondary to volvulus or intussusception are some of the predisposing factors. The cause of the perforation may not be apparent. Iatrogenic perforation from enemas, thermometers and instrumentation usually occurs in the rectum or rectosigmoid. Thomas and Brockman[6] reviewed 26 cases of colon perforation of obscure etiology. In their series colitis, frequently due to coliform organisms, was considered the major underlying factor. The bacterial flora may have been altered by broad spectrum antibiotics in some of the patients. Although atresia of the colon is rare and relatively few barium enema studies are reported,[4] the ease with which the distal segment was perforated during the barium enema examination in our case was surprising to us. That portion of the blind distal colon through which the perforation occurred was devoid of muscle. We would postulate this as the explanation of why the rupture took place. It is known that the microscopic appearance at the end of an atretic segment is variable.



FIG. 3. Abdominal roentgenogram 2 weeks later, after surgical exploration and irrigation of the peritoneal cavity. Residual intraperitoneal barium sulfate is seen.

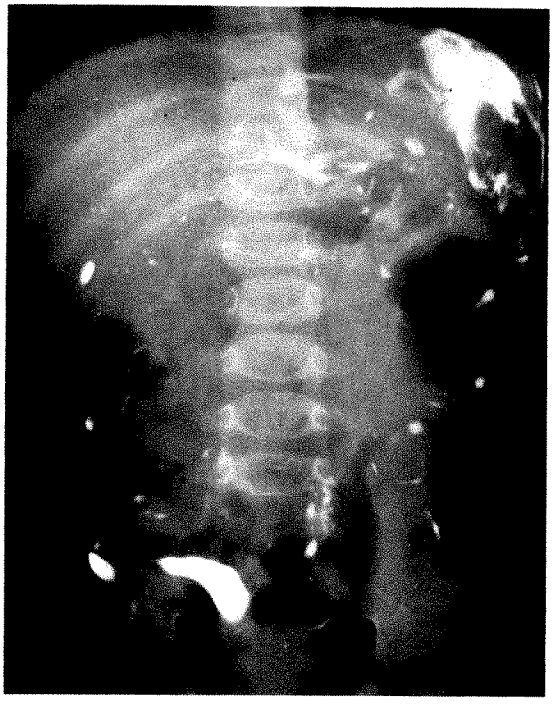


FIG. 4. Abdominal roentgenogram 10 months later with residual barium sulfate persisting in the peritoneal cavity.

Contrast enema studies are generally recommended in the newborn period in distal bowel obstructions.[2] Barium sulfate in the peritoneal cavity sets up a foreign body reaction that can lead to adhesions and eventual bowel obstruction even in the absence of infection.[3] Certain commercial barium sulfate preparations are more injurious to the peritoneal cavity than others.[1] Iodinated contrast media have little histologic effect on the peritoneal cavity of experimental animals.[5] They can produce death in experimental animals if sufficient volume of these hypertonic solutions is introduced into the peritoneal cavity. In view of the different histologic peritoneal responses to barium sulfate preparations and iodinated contrast media it

may be in the patient's best interest to use the latter for contrast enemas in suspected distal bowel obstruction in the newborn period. Careful fluoroscopic monitoring of the enema study should be made to prevent significant quantities of iodinated contrast media from entering the peritoneal cavity if perforation did occur.

SUMMARY

A patient with atresia of the colon is reported in whom the distal colonic segment was perforated during a barium enema examination. The perforation occurred through an area devoid of muscle.

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# COLONIC ILEUS AND ITS DIFFERENTIAL ROENTGEN DIAGNOSIS*

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COLONIC ileus is a syndrome of diffuse or segmental colonic dilatation due to a variety of causes. A list of the reported etiologies is given in Table I. Notwithstanding the cumulative experience in the roentgen diagnosis of the acute abdomen, it must be recognized that the criteria for the plain roentgenogram differential diagnosis of colonic ileus and colonic obstruction are largely inadequate. Many authors[2,11,15,20] have stated that the roentgen picture of colonic ileus is indistinguishable from that of mechanical colonic obstruction.

It is against this background that a comprehensive analysis of the roentgen findings of colonic ileus and colonic obstruction was undertaken with special emphasis on the plain roentgenogram of the dilated colon and its contents. The purpose of this communication is to develop the roentgen criteria for the differential diagnosis of these entities based on the patterns of colonic dilatation.

TABLE I

REPORTED CAUSES OF COLONIC ILEUS

1. Acute appendicitis[21]
2. Acute cholecystitis[21]
3. Acute gastroenteritis[21]
4. Acute pancreatitis[2,20]
5. Cardiac failure[21,22,25]
6. Electrolyte disturbances with either raised or lowered serum potassium[21]
7. Ganglionic blocking agents[6]
8. Idiopathic[5,6]
9. Lead poisoning[21]
10. Mesenteric thrombosis[11]
11. Morphine overdosage[21]
12. Myxedema[1]
13. Parkinson's disease (especially on anti-Parkinson's medications)[4]
14. Pelvic or extraperitoneal infection[22]
15. Peritonitis[22]
16. Porphyria[21]
17. Psychosis[13,24]
18. Pulmonary embolism[22,25]
19. Renal failure[21,25]
20. Sepsis[16,22]
21. Surgery, especially pelvic surgery[16,19]
22. Trauma[22]
23. Urinary calculus[22,25]

## MATERIALS AND METHODS

Fifty cases of colonic ileus and 70 cases of mechanical distal colonic obstruction (*i.e.*, distal to the splenic flexure) due to carcinoma or diverticulitis constitute the material for the present investigation. As noted in Table II, our cases of colonic ileus include a variety of etiologic factors. In most instances, the categorization of colonic ileus as either spastic or adynamic is difficult[5,15,21,22,25] and for this reason no such division was made in this study. Cases with small bowel dilatation were considered as colonic ileus, if the colonic dilatation was predominant.

Each case was reviewed in detail for the following salient points: pattern and continuity of gaseous colonic dilatation; thickness of the colonic wall; inner contour of the bowel; haustrations; septa; amount and appearance of the retained fluid; and amount and appearance of the retained fecal matter.

The adoption of these roentgen criteria for the evaluation of the various causes of colonic dilatation was based upon favorable personal experience in the diagnosis of colonic ileus during the past few years. These favorable results were obtained by the application of a few empirical propositions which we had made in determining the significance and the relative value of

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TABLE II

CAUSES OF COLONIC ILEUS IN THIS SERIES OF CASES

| | |
|---|---|
| Idiopathic | 8 |
| Cardiac failure | 7 |
| Urinary calculus | 6 |
| Acute cholecystitis | 6 |
| Surgery | 5 |
| Mesenteric thrombosis | 4 |
| Acute pancreatitis | 3 |
| Acute appendicitis | 2 |
| Trauma | 2 |
| Pelvic abscess | 2 |
| Sepsis | 2 |
| Parkinson's disease | 2 |
| Acute gastroenteritis | 1 |
| Total | 50 |

various roentgen features. It would seem helpful to discuss these propositions as a basis for the understanding of colonic dilatation.

PROPOSITIONS

(1) *The colon proximal to an obstructing lesion is gradually altered by the early partially obstructive manifestations, so that when it becomes dilated secondary to more complete obstruction, its appearance differs from that of a dilated colon that is otherwise normal.*

Partial obstruction causes colonic muscular hypertrophy. Early, before chronic distention develops, this muscular hypertrophy may be indicated roentgenographically by accentuation of the haustrations and increase in the number of septa which in certain areas appear numerous and closely packed. Later, due to chronic distention, the haustrations become shallow or obliterated with a decrease in the number of septa.

Muscular hypertrophy also causes thickening of the colonic wall, but evaluation of thickening of the colonic wall roentgenographically is difficult because both the actual inner margin and the actual outer margin of the colonic wall cannot be delineated with certainty. Fluid or fecal matter between the bowel lumen and the colonic wall may stimulate thickening. The outer margin is only occasionally delineated by the pericolonic fat, and intraperitoneal fluid or omental edema may stimulate thickening.

As partial colonic obstruction becomes more chronic, the inner colonic contours lose their usual sharp definition and smoothness and become irregular and ragged. This is apparently due to mucosal edema, and adherent fecal matter and secretions. Despite chronic distention, a few of the colonic septa remain. They are, however, altered in appearance with varying degrees of thickening, irregularity and poor definition. The thickening may be partially secondary to muscular hypertrophy with the remainder of the septal abnormalities due to mucosal edema, and adherent fecal matter and secretions.

Colonic obstruction usually produces varying degrees of fecal retention. However, more important, the colonic fecal content is frequently altered from its normal semisolid or solid consistency. In many cases of chronic obstruction, the retained colonic fluid has a grey, finely mottled appearance on roentgenograms. This is due to small particles of fecal matter finely suspended in the retained fluid. This finding can be seen in the recumbent studies but is best evaluated in the erect or decubitus roentgenograms.

The greater the severity and chronicity of the obstruction, the more prominent will be the above findings. In relatively acute colonic obstruction, only a minimal degree of abnormality of the dilated colon will be detectable.

(2) *In colonic ileus, the appearance is frequently that of acute dilatation of a previously normal colon. Preserved haustrations, thin well defined septa, smooth inner colonic contour and thin colonic wall are the cardinal features of the acuteness of the process.*

The amount of fecal matter varies depending upon the amount of feces previously present in the colon. The fecal matter, however, usually has a relatively normal consistency with fecal boluses in the left colon and mottled semisolid feces in the right colon.

Some cases of colonic ileus show a some-

what more severe, less acute pattern. This may be indicated by greater colonic dilatation with some haustral and septal loss The septa may appear slightly thickened due to edema or muscular hypertrophy. The retained fecal contents may be more fluid with adherent feces and secretions producing slight irregularity of the inner colonic wall. The roentgen picture of colonic ileus in the more severe less acute cases thus merges to an extent with that seen in the less severe more acute cases of colonic obstruction.

(3) *The relative amount of fluid and gas in the dilated colon is related to the intraluminal pressure with predominant gaseous distention indicating a more acute process with lower intraluminal pressures and predominant fluid distention indicating a more chronic process with higher intraluminal pressures.*[23] *Therefore, predominant fluid distention of the colon is indicative of colonic obstruction.*

Roentgen evaluation of the fluid-filled colon may be difficult unless attention is paid to the ill defined fluid-filled masses, usually the cecum and ascending colon. The fluid may contain little fecal matter and may thus be homogeneous and blend with other unit-density intra-abdominal structures. A long air fluid level in the erect or decubitus roentgenogram, small collections of gas trapped between the septa, or gaseous dilatation of the transverse colon may direct attention to the fluid-filled right colon.

This pattern is not seen in the colonic ileus, in which dilatation is acute and intraluminal pressures are low. Cecal perforation due to the high intraluminal pressures developing in the dilated cecum in colonic obstruction[3,14] is extremely rare in colonic ileus.[15,19] Gaseous distention predominates with relatively small amounts of fluid. The distended colon can, therefore, usually be followed in continuity from the cecum to the distal dilated segment. In the overwhelming majority of cases, the colonic dilatation extends into the left colon and the maximum dilatation in the supine roentgenogram involves the transverse colon. The cecum may be severely dilated in the prone roentgenogram or in the supine roentgenogram when it is mobile and anteriorly located.

Some cases of colonic ileus due to intra-abdominal inflammatory processes with peritonitis show somewhat more fluid.[9] The amount of fluid, however, is less than that seen with colonic obstruction, so that a completely fluid-filled right colon is not observed. In addition, the features of the gas-filled colon are those of an acutely dilated unaltered colon—the picture characteristic of colonic ileus.

Small bowel dilatation in colonic obstruction is generally considered to be due to incompetence of the ileocecal valve.[9,23] At times it is the predominant finding. The dilated colon, however, usually can be detected and shows the features of colonic obstruction outlined above.

## ROENTGENOGRAPHIC FINDINGS

Figure 1 illustrates colonic ileus of the acute type (etiology-pelvic surgery). The dilatation involves the entire colon and can be followed in continuity from the cecum to the rectum. Figure 2 illustrates a case of colonic ileus with segmental dilatation extending to the splenic flexure (etiology-renal failure). Figure 3 shows a similar case with segmental dilatation to the mid-decending colon but with more fecal matter in the ascending colon (etiology-cardiac failure). The colon in these 3 cases is significantly dilated but its anatomic features are normal. The haustrations are relatively preserved and regular. The septa are thin and well defined. The inner colonic contour is sharp and smooth. The gas-filled colon can be followed in continuity due to the presence of minimal fluid. The fecal matter present appears to be of normal consistency.

Figure 4 demonstrates severe distention in colonic ileus (etiology-hip surgery). Despite the huge dilatation, some haustrations are present and the septa remain thin and sharply defined. Gaseous distention allows visualization of the dilated colon in continuity.

Figure 5 illustrates colonic ileus of the

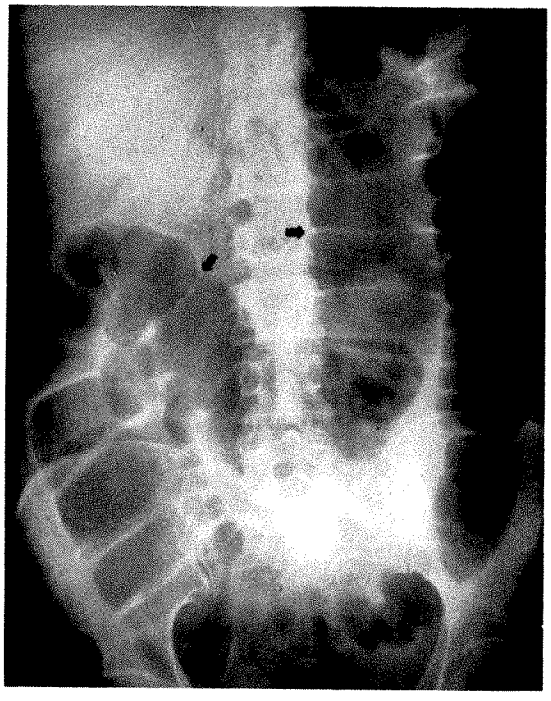


FIG. 1. Colonic ileus (etiology-pelvic surgery). The entire colon shows gaseous distention and can be followed in continuity from the cecum to the rectum. The haustrations are preserved and regular and the septa are thin and sharply defined (arrows)

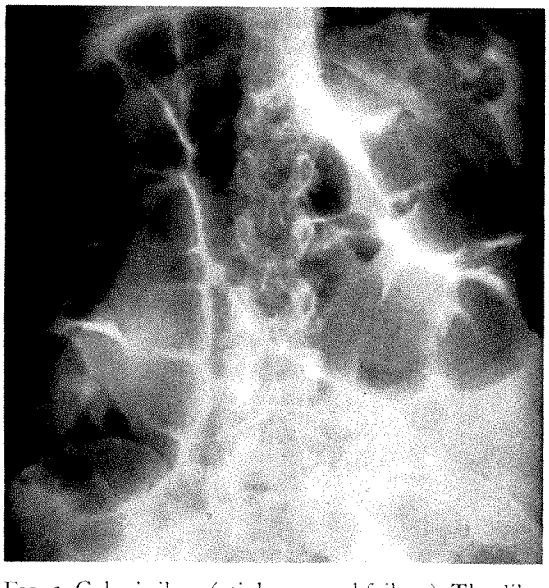


FIG. 2. Colonic ileus (etiology-renal failure). The dilatation of the colon is segmental, extending from the cecum to the splenic flexure. There is slight small bowel dilatation. The haustrations are relatively preserved. The septa are thin and the inner colonic contour is smooth.

more chronic type (etiology-idiopathic). Haustrations are shallower and less frequent than in the previous cases. Many of the septa are slightly thickened and less well defined. There is slight irregularity of the inner cecal contour.

Figure 6 shows a case of colonic ileus demonstrating a larger amount of fluid (etiology-acute cholecystitis with peritonitis). The ascending colon contains a moderate amount of fluid but its gas content is visible. The appearance of the transverse colon, however, is consistent with the acute type of colonic ileus.

Figure 7 illustrates a pattern of chronic colonic obstruction with marked abnormality of the dilated colon. There is marked gaseous distention, but with loss of haustrations, occasional thick, rather irregular ill defined septa and a ragged inner contour of the colonic wall.

Figure 8 illustrates less chronic obstruc-

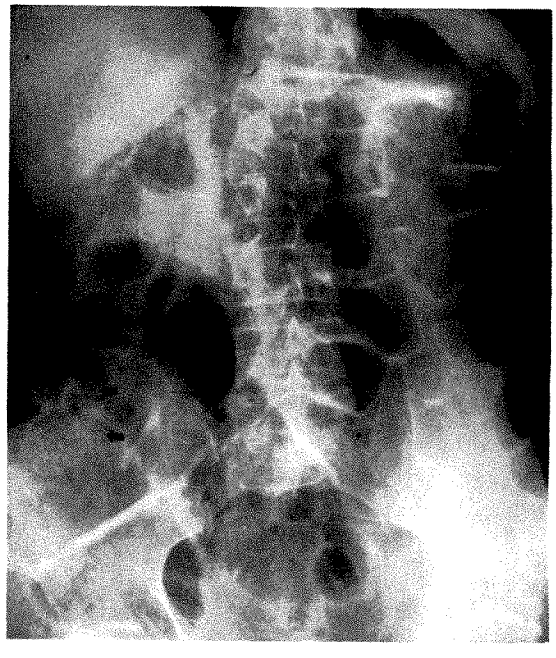


FIG. 3. Colonic ileus (etiology-cardiac failure). The colonic dilatation extends to the mid descending colon. The appearance of the dilated colon is similar to the cases illustrated in Figures 1 and 2. The semisolid appearing fecal matter (arrows) in the ascending colon is of normal consistency for this area.

tion with gaseous distention but with haustral and septal obliteration.

Figure 9 shows a case with greater fluid retention. The retained fluid fecal matter is grey and mottled in appearance.

Figure 10 illustrates the ragged inner contour of a dilated cecum apparently due to adherent fecal matter, secretions and mucosal edema.

Figure 11 illustrates relatively acute colonic obstruction with predominant gaseous distention and absence of fecal matter. Some haustrations are present and are somewhat accentuated in the distal transverse colon. The septa in the hepatic flexure are closely packed.

Figure 12 illustrates a case with predominant fluid distention. The gas is localized to the transverse colon. The dilated fluid-filled cecum and ascending colon are seen as a mass density in the right side of the abdomen with small collections of gas trapped at the apices of the haustrations.

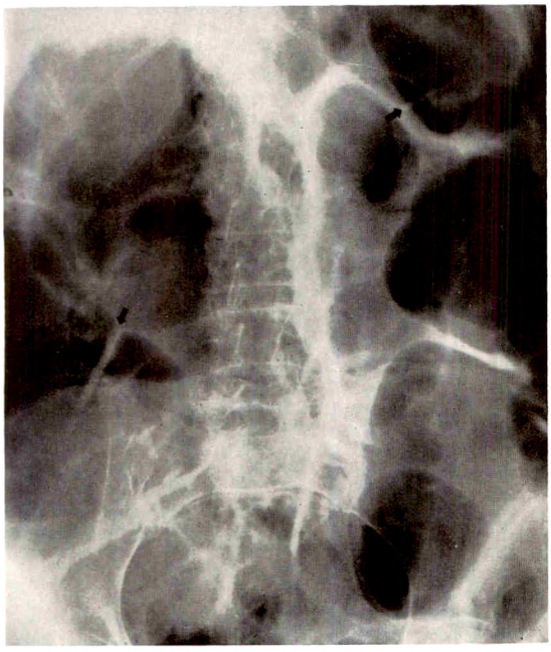


Fig. 4. Colonic ileus (etiology-hip surgery). The colon is markedly distended. Despite the huge dilatation, some haustrations are preserved and the septa (arrows) remain thin and sharply defined. Gaseous distention allows visualization of the dilated colon in continuity.

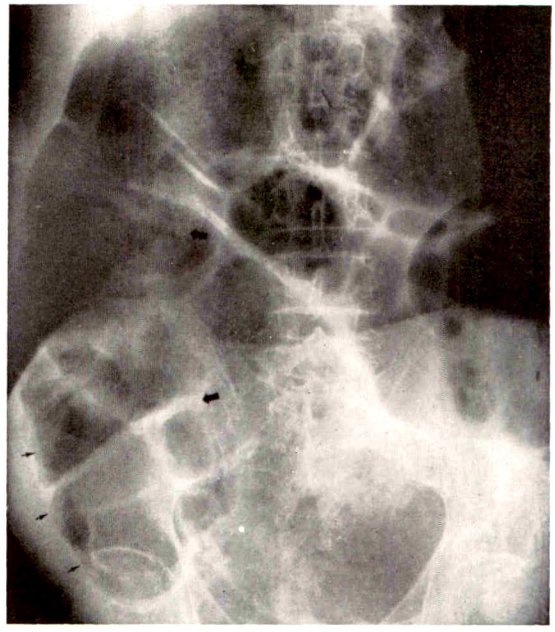


Fig. 5. Colonic ileus (etiology-idiopathic). Colonic distention is marked. Many of the septa (large arrows) are thickened, slightly irregular and less well defined. There is slight irregularity of the inner cecal contour (small arrows).

## DIFFERENTIAL DIAGNOSIS

(1) *Volvulus*. In volvulus, the colonic obstruction is of relatively acute onset and the dilated colon is similar in appearance to that in colonic ileus. A number of reports[3,4] mention that sigmoid volvulus may be an occasional complication of colonic ileus due to twisting of the dilated sigmoid. The two entities are thus seen to merge in some cases. Although cecal volvulus has been reported as a complication of mechanical neoplastic obstruction, this is a rare complication and infrequently a diagnostic problem.[18]

The roentgen picture in volvulus, therefore, resembles the more severe cases of colonic ileus in the features of the dilated loops. The diagnosis of sigmoid volvulus, however, is suggested by the prominent dilatation of the smooth walled ahaustral sigmoid, extending from the pelvic cavity into the upper abdomen.[6,8,9,17] In cecal volvulus, the ectopic location of the cecum with its characteristic configuration is diagnostic.[7,9]

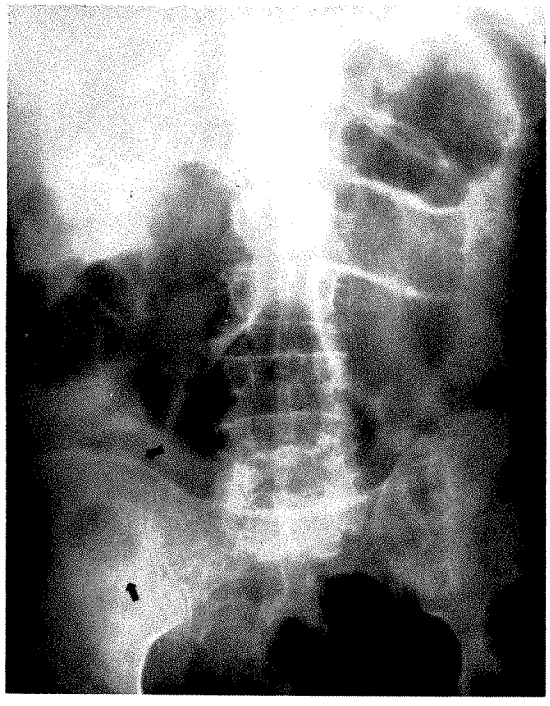


FIG. 6. Colonic ileus (etiology-acute cholecystitis with peritonitis). The colonic distention extends to the splenic flexure. The ascending colon and cecum contain a moderate amount of fluid producing a soft tissue density interspersed with collections of gas (arrows). The transverse colon shows the features of acute colonic ileus.

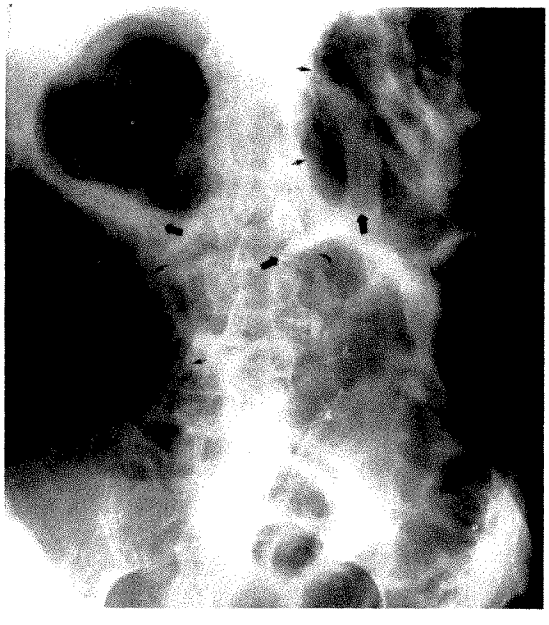


FIG. 7. Colonic obstruction due to sigmoid carcinoma. This is a pattern of chronic obstruction with severe abnormality of the dilated colon. There is total loss of haustrations, occasional thick irregular ill defined septa (large arrows) and a ragged inner contour of the colonic wall (small arrows).

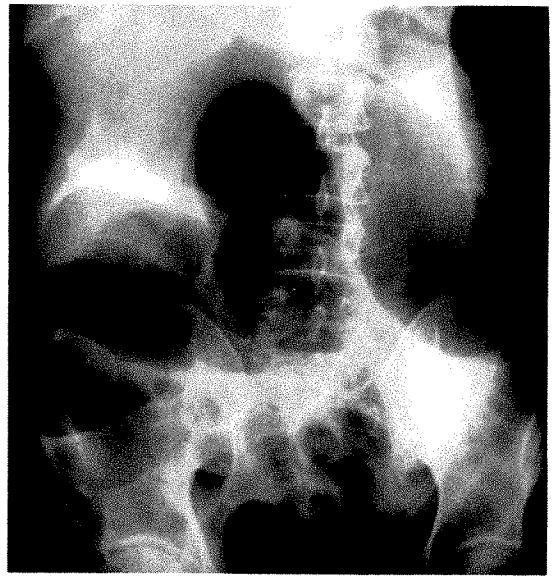


FIG. 8. Colonic obstruction due to sigmoid carcinoma. There is gaseous distention, but with obliteration of the haustrations and most of the septa.

(2) *Toxic dilatation during the course of ulcerative colitis.* In toxic dilatation, the dilated colon shows more severe abnormalities than are seen in colonic obstruction.[26] The important findings are: (a) irregular inner contours of the colon with multiple deep projections indicative of ulceration; (b) numerous nodular pseudo-polypoid intraluminal protrusions; and (c) relative shortening of the colon. A rare complication of an obstructing neoplasm is acute colitis in the proximal colon.[10,12] The changes in the colon in this entity are less prominent than in ulcerative colitis and there are no reports of their recognition on plain roentgenograms as in toxic dilatation.

## DISCUSSION

The previously discussed propositions indicate that, in the overwhelming majority of cases, the findings on the plain roentgenogram in colonic ileus do not resemble

colonic obstruction. In colonic obstruction, the colon proximal to the lesion is first altered in varying degrees in its anatomy and contents by the developing lesion and then by mechanical obstruction. In colonic ileus, the involved colon is relatively normal prior to the acute episode and then becomes dilated by a reflex mechanism[15, 20,21] which probably differs significantly from the mechanical mechanism of colonic obstruction.

From the standpoint of plain roentgenogram differential diagnosis of colonic ileus and colonic obstruction, the cases can be divided into 3 categories: (1) most likely colonic obstruction; (2) most likely colonic ileus; and (3) indeterminate. Based on the criteria previously discussed and reviewed below, 86 per cent of our cases of colonic obstruction would fall into Category 1, 87 per cent of our cases of colonic ileus in Category 2, and 14 per cent of our cases of colonic obstruction and 13 per cent of our cases of colonic ileus in Category 3.

In Category 1 are those cases with two or more of the following findings: noncontinuous gaseous colonic distention; fluid-filled right colon; accentuation of haustra-

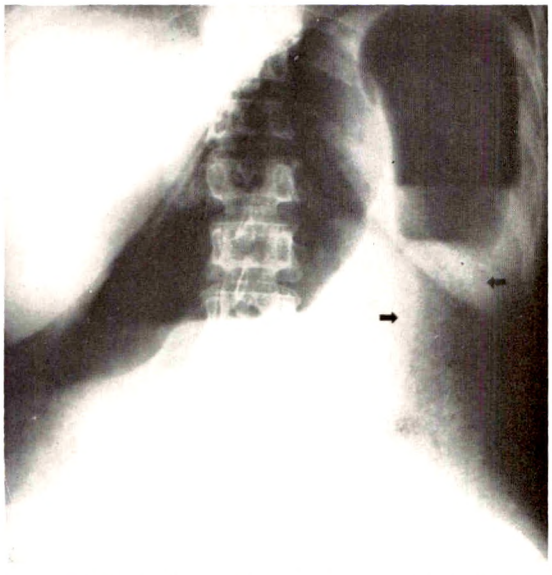


Fig. 9. Colonic obstruction due to sigmoid carcinoma. Erect roentgenogram. The retained colonic fluid has a grey mottled appearance (arrows).

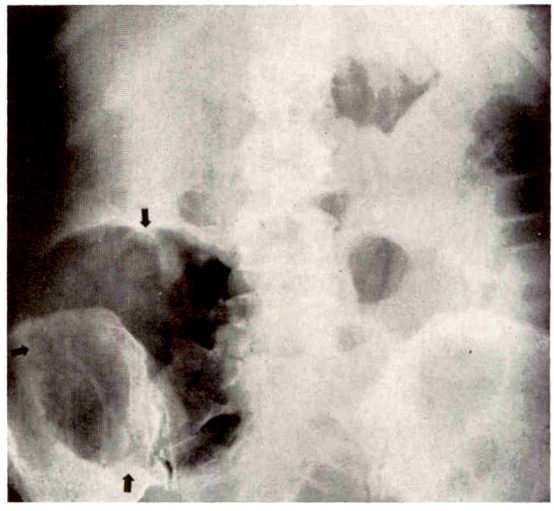


Fig. 10. Colonic obstruction due to carcinoma of the splenic flexure. The gas distended cecum (arrows) shows a ragged inner contour. The hepatic flexure and portions of the transverse colon are fluid filled.

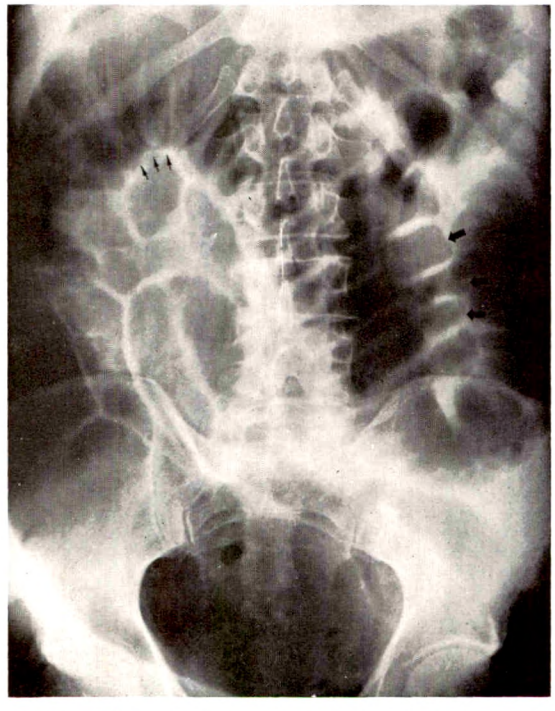


Fig. 11. Colonic obstruction of the relatively acute type due to carcinoma of the descending colon. There is predominant gaseous distention and absence of fecal matter. The haustrations are somewhat accentuated in the transverse colon (large arrows), but are obliterated in the ascending colon. In the hepatic flexure, the septa are closely packed (small arrows).

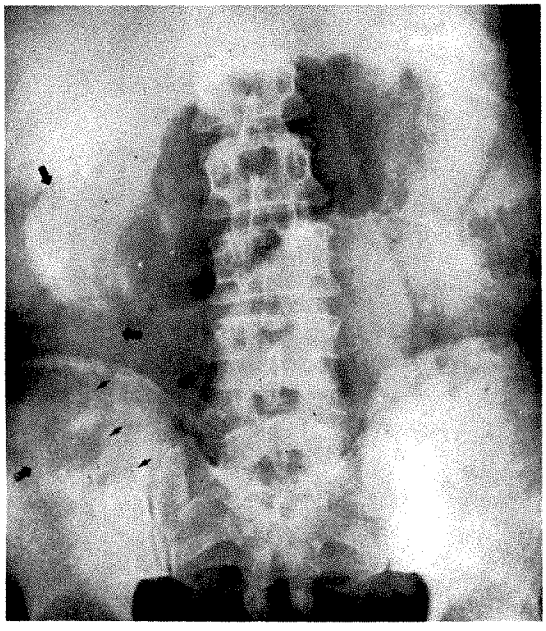


FIG. 12. Colonic obstruction with predominant fluid distention due to carcinoma of the descending colon. The gas is localized to the transverse colon. The cecum, ascending colon and hepatic flexure appear as a mass density in the right side of the abdomen (large arrows) with small collections of gas trapped at the apices of the haustrations (small arrows).

tions with numerous closely packed septa; loss or considerable shallowness of haustrations; thickened ill defined, irregular septa; large amounts of fecal retention; ragged inner colonic contour; and grey mottled fluid. In these patients, sigmoidoscopy and barium enema examination on an urgent basis are indicated. This can usually be performed without any further jeopardy to the patient. If the patient's condition is such as to contraindicate these studies, the findings are sufficient to indicate to the surgeon that the most expeditious colonic decompressive procedure is necessary. In some cases, with large amounts of fecal retention and some gaseous distention, barium enema study will show no obstructing lesion. These are patients with chronic constipation and fecal impaction usually on a neurogenic or psychogenic basis.

In Category 2 are those cases with the following findings: continuous gaseous distention with minimal fluid; preserved and regular haustrations; thin well defined septa; smooth inner colonic contour; and normal consistency of fecal contents. Further help can be obtained from the clinical history and findings indicating the probable cause of the colonic dilatation. Barium enema examination on an urgent basis is not necessary in these cases. In some conditions, such as severe cardiac disease or pulmonary embolism, it may involve significant jeopardy to the patient. The clinician can be assured that the plain roentgenogram findings favor colonic ileus and that the further diagnostic course and treatment of the patient should be considered from this standpoint.

In Category 3 are those cases with the following findings: severe gaseous colonic distention but with small amounts of fluid; slight haustral loss; slight septal thickening; and slight irregularity of the inner colonic contour. Barium enema examination is necessary in these cases for further evaluation and surgery should not be contemplated prior to this study.

## SUMMARY

Mindful of the inadequacy of the existing roentgen criteria of colonic ileus, a systematic analysis of the plain roentgenogram findings on 50 such cases was undertaken, with special emphasis on the patterns of colonic dilatation pertaining to the acuteness or chronicity of the process and the nature of the colonic contents. While its pathogenesis varies, colonic ileus did present diagnostic features. These are: continuous gaseous dilatation; minimal fluid; preserved regular haustrations; thin well defined septa; smooth inner colonic contour; and normal consistency of the fecal contents.

Concurrent study of 70 cases of low colonic obstruction due to carcinoma or diverticulitis indicated the validity of certain criteria which favor obstruction. These are: noncontinuous gaseous colonic distention; fluid-filled right colon; accentuation of haustrations with numerous

closely packed septa or loss or shallowness of haustrations; large amounts of fecal retention; thickened ill defined irregular septa; ragged inner colonic contour; and grey mottled fluid.

Realization of these characteristics may enable us to arrive at the correct diagnosis. The advantage of such a possibility should not be underestimated as either sigmoidoscopy or barium enema examination may become extremely precarious if not harmful under certain circumstances.

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# ANGIOGRAPHY IN CROHN'S DISEASE OF THE SMALL BOWEL AND COLON*

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SINCE Crohn's first description in 1932 of regional ileitis,[6] the same histopathology has been repeatedly recognized by different authors in all parts of the intestinal tract from the stomach to the rectum.[2,7,8,9,11] The multiplicity of sites of involvement and pathologic patterns makes it clear that no single generic term encompasses the entire disease. Thus in the panel concerned with this entity in the May, 1966, Annual Meeting of the American Gastroenterological Association, the moderator was prompted to predict that the arbitrary designation of Crohn's disease would probably secure a more firm position in the literature, by default.

Crohn's disease affects all bowel layers, frequently extends into the mesentery, and shows a tendency to follow along vessel paths.[14] Moreover, vascular changes with consequent luminal compromise occur regularly.

## MATERIAL AND METHODS

Fourteen patients were diagnosed in the following ways: 8 by microscopic examination of operative specimens; 6 by clinical signs and symptoms reinforced with typical barium study findings in the small bowel, colon, or both. Of the 14 patients, 10 had sigmoidoscopy, small bowel series, and barium enema examination, 2 had sigmoidoscopy and small bowel series and 2 had sigmoidoscopy and barium enema examination.

Selective arteriography of the superior and inferior mesenteric arteries was performed in 8 cases. In 5 additional cases investigation was confined to the superior mesenteric artery and in 1 case to the inferior mesenteric artery.

Percutaneous catheterization of the visceral arteries was performed using the Seldinger technique. In most cases requiring its study, the colon was distended with air immediately prior to arterial contrast injection to effect optimal separation of the small arteries for their clear depiction. More recently saline has been substituted for air in order to equalize body density in the area of interest, thereby giving a remarkably uniform photographic field for vessel study.

## RESULTS

### SMALL BOWEL

Barium small bowel series led to recognition of abnormality characteristic of Crohn's disease in 10 cases. The least severe changes were found in the proximal small bowel and increased in severity as the terminal ileum was approached; *i.e.*, edema of the mucosal folds was characteristic in the jejunum and there was a variably severe luminal narrowing of the distal ileum with a gradient between them.

The superior mesenteric arteriography showed the following findings in the loops recognized as edematous by barium studies: the branches of the superior mesenteric artery were dilated until abruptly tapering at or slightly before reaching the bowel. The branches immediately distal to tapers were characterized by right angle bifurcations (in contrast to the acute bifurcation patterns of the normal) and numerous narrowings (Fig. 1*A*; and 2*A*). In the capillary phase a dense accumulation of contrast

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FIG. 1. Superior mesenteric angiography in a 17 year old male with early Crohn's disease of the small bowel. (*A*) Arterial phase shows dilated intestinal branches with abrupt tapering in the periphery. Irregular distribution of peripheral branches. (*B*) Venous phase with contrast medium of high concentration in dilated veins.

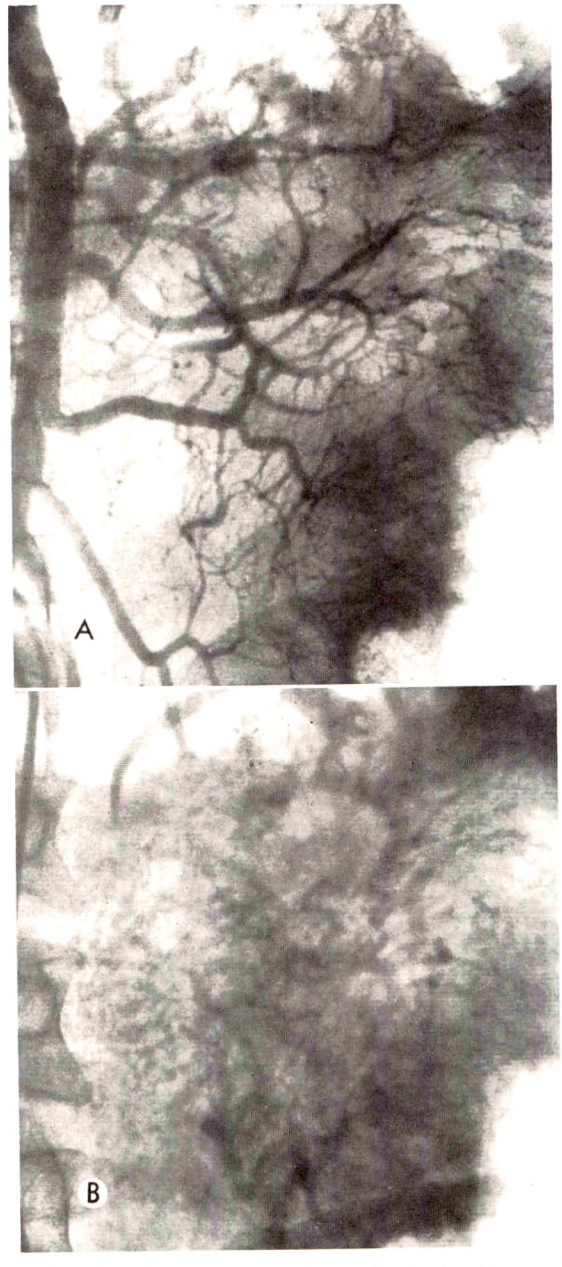



FIG. 2. Superior mesenteric angiography in a 21 year old male with early Crohn's disease involving the small bowel. (*A*) Arterial phase shows dilated intestinal branches with irregular lumen, right angle bifurcations and tapering in the periphery. (*B*) Venous phase shows contrast medium of rather high concentration in irregular veins.

medium was seen in the diseased bowel wall, whereas in normal bowel no accumulation was apparent. The venous drainage of



FIG. 3. Inferior mesenteric angiography in a 17 year old woman with early Crohn's disease of the colon. Note the wide arteries and the vasa recta which do not taper toward the periphery.

the diseased segments was both distinctly premature and remarkably dense compared with the normal (Fig. 1*B*; and 2*B*).

Advanced Crohn's disease presented a substantially different aspect. The arteries to the narrowed bowel were of normal or slightly reduced diameter except the terminal branches, which were abnormally tiny, irregular in outline, right angled in their bifurcations, and tortuous (Fig. 4). The contrast blush of bowel wall in the capillary phase was absent and venous opacification was scarcely discernible. In no instance did superior mesenteric arteriography fail to provide complete localization of small bowel disease already recognized in the barium study. More important, in 3 of the cases arteriography showed additional areas of abnormality not reaching the threshold of barium study recognition.

COLON

In 3 cases barium enema examination showed typical Crohn's disease of the colon; in 3 cases the findings were pathologic but nonspecific; in 1 case they were suggestive of ulcerative colitis. Sigmoidoscopy disclosed mucosal ulcerations in 2 cases in which barium enema study ap-



FIG. 4. Superior mesenteric angiography in a 10 year old boy with advanced Crohn's disease involving the terminal ileum and ascending colon. The arteries supplying the involved area are rather tiny, show irregular lumen and right angle bifurcations.

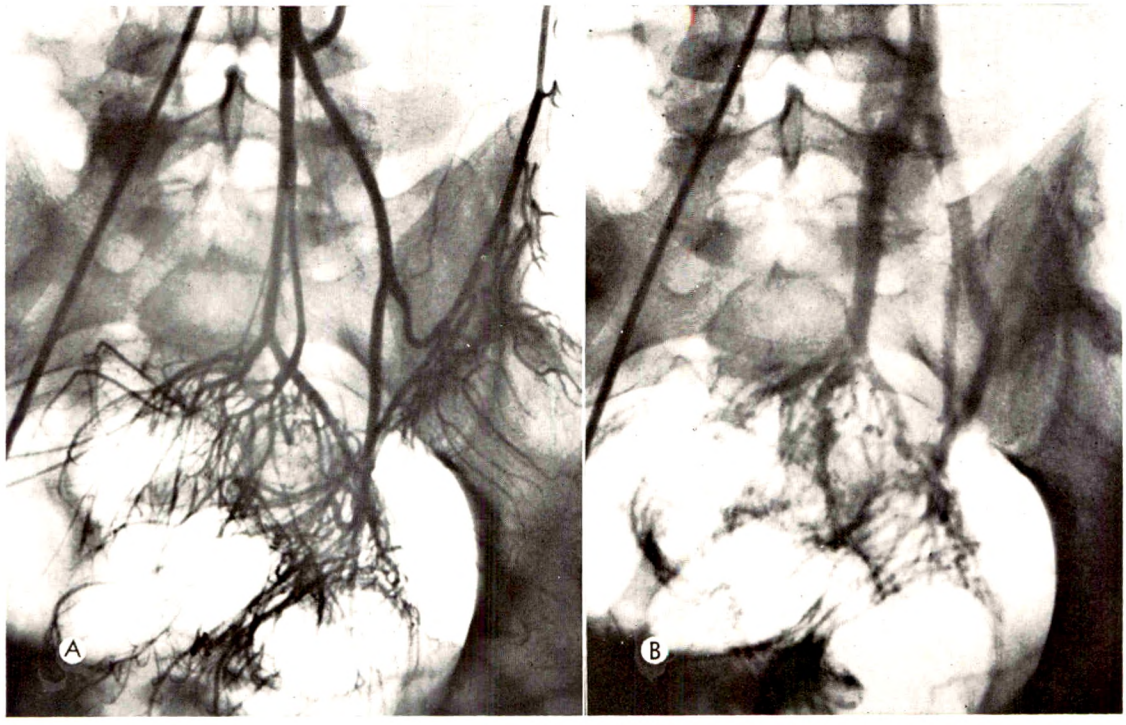



Fig. 5. Inferior mesenteric angiography in a 26 year old male with rectosigmoid extension of Crohn's disease which first appeared in the distal ileum. (*A*) The arteries supplying the rectosigmoid area are wide and the vasa recta do not taper toward the periphery. (*B*) Draining veins are wide and the concentration of contrast medium is high.

peared to be normal. Barium demonstration of mucosal edema and ulcerations indicated early disease and narrowing was pathognomonic of advanced disease.

Selective mesenteric arteriography in the 2 cases negative by barium enema examination but positive by sigmoidoscopy showed dilated rectal and sigmoid arteries, most strikingly in the vasa recta. The arterial contours were smooth (Fig. 5*A*). Contrast medium appeared with unusual density and rapidity in veins draining the area incriminated by sigmoidoscopy (Fig. 5*B*).

In the case in which barium enema examination suggested ulcerative colitis (Fig. 3), the colon likewise was supplied by widened, smooth arteries. The vasa recta, also very wide, did not taper toward the periphery. Venous visualization was premature, overly-dense, and the veins were dilated. This patient was operated upon with a working diagnosis of ulcerative colitis, but microscopic study of the operative specimen showed clear evidence of Crohn's disease.

The arteries to a narrowed part of the colon had a normal or slightly decreased diameter with the exception of the peripheral branches which were tiny, showed lumen irregularities, right angle bifurcations and a tortuous course (Fig. 4). In these parts no accumulation of the contrast medium in the bowel wall was noted during the capillary phase and the low concentration of the contrast medium in the veins made them scarcely perceptible.

## DISCUSSION

In the several cases in which Crohn's disease involved almost the entire small bowel, it was possible to correlate the disease's varying severity as determined by small bowel series with corresponding angiographic patterns (Table 1).

In those lesions recognized as early in

TABLE I

ANGIOGRAPHIC CHARACTERISTICS

| | Arterial Findings | Venous Findings |
|---|---|---|
| Normal | Smooth arteries which taper successively toward the periphery. Acute angle bifurcations and regular distribution of peripheral branches | Low concentration of contrast medium in the veins which can scarcely be outlined |
| Ulcerative Colitis | Dilated arteries without lumen irregularities. Wide vasa recta which do not taper toward the periphery | Increased concentration of contrast medium in dilated early filled veins |
| Early Crohn's Disease | *Small bowel.* Dilated arteries which abruptly taper when they reach the bowel wall. Peripheral branches show right angle bifurcations, bizarre distribution, and small luminal irregularities<br>*Colon.* Dilated arteries without lumen irregularities. Wide vasa recta which do not taper toward the periphery | Increased concentration of contrast medium in dilated early filled veins |
| Advanced Crohn's Disease | *Small bowel* and *colon.* Normal or reduced arterial calibers, right angle bifurcations and irregularities of lumen | Low concentration of contrast medium in veins of normal diameter |

the small bowel series, *e.g.*, mucosal edema and slight dilatation, the bowel was supplied by arteries which in their mesenteric courses exhibited dilatation, right angle bifurcations, and abrupt tapering just prior to reaching the bowel wall. The mural arborization was bizarrely arranged, and minor irregularities were noted in the small vessels. Dense contrast material loading of involved bowel wall occurred during the capillary phase, a finding not seen in normal cases. Premature and unusually dense venous opacification was also a distinct departure from the normal.

In more advanced lesions, *e.g.*, narrowed bowel, the mesenteric arterial calibers had reduced from their early phase dilatation to normal or slight narrowness and had become irregular. The bifurcations continued to be at right angle. The contours of mural arteries were more strikingly irregular than in early disease. Contrast material accumulation in the bowel wall was absent in the capillary phase and venous drainage was almost impossible to interpret because of its faintness.

The arterial changes in the colon in early and advanced Crohn's disease were similar to those described for the small intestine with one exception: in the early stage the mural arteries were unusually wide, and were straight and smooth, resembling the changes seen in ulcerative colitis,[3,4,5,10,12,15,16,17] thus rendering differentiation impossible. In advanced cases, however, Crohn's disease in the colon showed narrowing of the arteries and lumen irregularities—findings not seen in long standing ulcerative colitis.

Boijsen and Reuter,[8] in reporting the angiographic findings in 11 patients afflicted with this disease, noted slight dilatation of the arteries and veins with increased blood flow. The vasa recta of the diseased bowel were slightly irregular and some tapered abruptly. A thickened bowel wall was seen during the capillary phase. These findings corresponded to those in our cases of early disease which had not progressed to bowel narrowing and rigidity.

Brahme,[4] described the angiographic findings in 5 patients with Crohn's disease which were somewhat at variance with those reported by Boijsen and Reuter. He described arteries of normal width, slight tortuosity, and slight irregularity. In dis-

ease of the terminal ileum, the arteries were numerically reduced. Blood flow was decreased and mural contrast opacification absent. Bacaner,[1] also reported the blood flow to be reduced in Crohn's disease as determined radioisotopically. His case was of unspecified chronicity but was in agreement with our findings in long standing disease in which bowel narrowing was present.

A unifying explanation is suggested by our microscopic blood vessel studies in the early and late stages of Crohn's disease. Early in the disease there is dilatation and hyperemia of the submucosal vessels and to lesser degree also of the mucosal capillaries. Later, thickening of the vessel walls, together with lumen encroachment is seen, explaining the decrease of flow and volume capacity of the vascular bed. As a consequence, the channels of the more central mesenteric arteries serving those segments are reduced. Selective arteriography is advantageous in demonstrating the luminal irregularities, permitting classification and staging of the findings.

Bockus *et al.*[2] discussed the rectosigmoid extension of Crohn's disease in patients whose findings first appeared in the distal ileum. Two of our cases who were examples of this, had negative barium enema studies but positive sigmoidoscopy and selective arteriography. There were increased vascularity and premature, overly dense venous opacification. Lindner *et al.*[13] reported that the secondary colonic lesions healed after the affected small bowel was resected. In 1 of our 2 cases, the patient's diseased terminal ileum was resected without relief or other change in the inflammatory findings at repeat angiography 8 months postoperatively. The second patient was not operated upon or studied further by arteriography.

Most patients with Crohn's disease of the colon have concomitant involvement of the small bowel. If the changes do not reach the threshold of recognition in small bowel series, an incorrect diagnosis of ulcerative colitis may be made. The angiographic investigation is of particular value in showing early inflammatory change that would otherwise escape detection. This fact results in at least two important corollary implications: firstly, the surgeon will be guided in choosing lines of resection; and secondly, if arteriography shows extremely wide, early inflammatory involvement in addition to that of surgical interest, this would deter the clinician from operative intervention and suggest medical treatment.

SUMMARY

Selective mesenteric arteriography in 14 cases of Crohn's disease more completely delineated disease extent than did conventional barium studies. It also permitted to reliably distinguish early from late disease, as well as a fully developed Crohn's disease of the colon from ulcerative colitis.

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# TEN YEARS EXPERIENCE WITH CELIAC AND UPPER ABDOMINAL SUPERIOR MESENTERIC ARTERIOGRAPHY*

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AS THE geographers have their incompletely explored land masses of Africa and the Polar Cap, so we in medicine have the pancreas. Statistically, carcinoma of the pancreas represents only 2 per cent of all malignancies; however, this presentation is concerned not only with malignant and benign pancreatic processes, but with other uses of visceral and primarily celiac angiography as well.

It is disturbing to note the following: (1) carcinoma of the pancreatic head can be diagnosed in only 50 per cent of cases by the conventional diagnostic methods of radiology, yet the pancreatic head is involved in 70 per cent;[26,60] (2) of all pancreatic malignancies only 25 per cent are diagnosed preoperatively by radiography[59] and the five year survival is less than 1 per cent;[46] (3) both of the above are even more disturbing when one considers the 12 per cent increase in men and the 22 per cent increase in women afflicted with carcinoma of the pancreas over the period 1949–1960.[55]

The need for better diagnostic techniques is dramatically indicated by the 22 year surgical experience which demonstrated that only 10 per cent of those patients who were operable, were candidates for removal of the neoplasm;[37] and of these, fewer than 13 per cent can be expected to survive 5 years or more.[18,47,61]

In the face of these facts the despair among physicians is easily understood, but it should only point out the need to investigate every reasonable diagnostic method to facilitate earlier detection. Only in the rare insulinoma, adenocarcinoma of the ampulla of Vater, Zollinger-Ellison syndrome and the cystadenocarcinoma are early symptoms of such characteristics as to enable early diagnosis. Pancreatic carcinomas often produce vague symptoms for 6 to 8 months and the physician often requires another 6 months to establish the diagnosis.

For these reasons the radiologist has long investigated various roentgenologic related techniques to study the pancreas. It is interesting that in 1951, Bierman *et al.*[10] first performed selective celiac angiography as well as superior mesenteric arteriography via arteriotomy in the carotid and brachial arteries. Rigler and his co-workers, in 1953[50] and 1954,[49] found gross translumbar angiography of the liver and spleen to be of value and thus was close to attempting angiography of the pancreas.

This paper will give the results of the use of selective celiac and superior mesenteric angiography for the past 10 years in 900 cases performed by one operator (B.P.S.), with surgical or autopsy proof in 400 of these cases. The mortality, morbidity and indications will be described with the older and the newer techniques. The latter have not been altered over the past 4 years. Equipment needed for both the small and the large hospital will be briefly discussed.

## CASE MATERIAL AND TECHNIQUE

Twenty-one years ago one of the great American radiologists[48] reviewed the fifty years of roentgenology and stated that

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visualization of the pancreas, liver and spleen remained an unsolved problem. In 1965, another excellent radiologist[22] could not give any indication of a change in this situation; only angiography has offered some hope. It now appears that the surgical therapy has exceeded our diagnostic acumen.

Following introduction of the ingenious percutaneous arterial catheterization method of Seldinger in 1953,[52] Ödman in 1956[40] gave a detailed description of the technique of selective celiac angiography and 2 years later reported his findings in 38 cases.[39] The excellent anatomic study of the vascularity of the upper abdominal organs by Michels in 1955[35] was undoubtedly of tremendous value to these early pancreatic angiographers. Odnoralov[41] and Berrett *et al.*[8] in 1964 have broadened the anatomic basis for interpretation. Boijsen, in 1961[13] and 1964[12] demonstrated the technical feasibility of the simultaneous catheterization and injection of the celiac axis and superior mesenteric arteries. The following year Lunderquist[32] reported on extensive studies of this simultaneous biarterial catheterization technique in pancreatic carcinoma. It was thought that such an approach would circumvent the problem of the 45 per cent[5,35] of patients who have anatomic variations of the celiac and superior mesenteric arteries.

Various techniques have been developed to alter the vascularity of the pancreas. Sgalitzer in 1937[53] reported the first use of a vasodilator in such an attempt, but the agent papaverine hydrochloride employed by him was without success. Reactive hyperemia following arterial compression was found by Sgalitzer to be of value and he also showed diathermy and sympathetic block to be of some value. Success was described with histamine acid phosphate (histamine phosphate) and tolazoline hydrochloride (prescoline).[32] Increased contrast with gas both inside the stomach and retroperitoneally was reported by Macarini and Oliva.[33] Many of these techniques have not only destroyed simplicity of a potentially valuable procedure but have been of little help in actual case material where they have been used. Secretin combined with an oral fatty emulsion has long been employed by Swedish angiographers, but the effectiveness of this technique remains in doubt. At present many angiographers are evaluating the various techniques and the larger the series, the more uniform the technical factors and results become. This is especially true in studies of over 100 cases. The valuable angiographic signs in benign and malignant disease are rapidly becoming apparent.

Over the period from 1956 to 1966 selective angiography of the upper abdominal viscera has been evaluated in 900 patients, with 400 of these studies having been judged on the basis of surgery or autopsy. This limitation in follow-up proof at three large hospitals where this study was performed indicates one of the great difficulties in correlating the results of angiography in pancreatic carcinoma. The loss of so many of these patients to follow-up was due in large part to the advanced stage of growth when they were examined. Thus many were obviously inoperable and were cared for in their terminal state at home. In those operated upon, one had difficulty in evaluating the "negative" operative findings for, as Ingelfinger[28] has indicated, palpation and even biopsy may be incorrect. Over the 10 year period we have had several patients who died of pancreatic carcinoma who had "negative" surgical exploration findings following positive celiac angiography for clinically suspected carcinoma. This is in keeping with the 11 to 36 per cent diagnostic error reported by biopsy of the pancreas.[11,15,23]

Initially, only the celiac axis was catheterized using conventional fluoroscopy and the impregnated polyethylene catheter as described by Ödman.[40] Lead markers placed over the vertebral bodies aided fluoroscopic localization of the faint catheter tip and filming was in the anteroposterior projection only. These markers

are no longer necessary with image amplification and/or television. Over the next 2 years many of the diagnostic or technical failures appeared to be due to the dual blood supply of the pancreas; *i.e.* the varying volumes of blood supplied from the celiac and/or superior mesenteric arteries.

In 1958, we attempted to solve this problem by first catheterizing the celiac trunk, obtaining frontal serial angiograms; and then catheterizing the superior mesenteric artery and repeating the angiography. In the second patient, a 36 year old male studied by this method, severe symptoms of vascular insufficiency of the bowel developed, lasting for 12 hours before they cleared. At that time an excessively large yellow Ödman catheter was used and we now know that it was the intimal damage by this large catheter rather than the type or small volume of contrast agent used that produced this complication. Nonetheless, this experience made us change back to celiac injection only, and it was at this time (1958) that the smaller, so-called green, Ödman catheter was chosen as more preferable. This catheter selection has remained the same to date, the one exception being the very small or young female where a red Ödman catheter is used. Anteroposterior and, after repeat injection, lateral angiograms of the celiac vascular complex are obtained, 50–65 cc. of sodium diatrizoate (hypaque 50 per cent) being used in the frontal and 15–25 cc. in the lateral projection. No adverse reactions were encountered either in patients who were normal, or had acute or chronic pancreatitis, or pancreatic carcinoma.

In 1960, we added superior mesenteric angiography in the frontal projection, if the celiac visualization was not diagnostic or if the extent of the malignancy was in question. For the superior mesenteric artery injection 25–35 cc. of sodium diatrizoate is employed and again no evidence of untoward angiographic reactions was encountered.

The use of a single catheter, the determination of the direction of the blood flow in the pancreatic arcade, and the complete visualization of the vascular supply of the pancreas seem ideal. The diagnostic accuracy with this technique has been increased and a satisfactory angiographic method appears to have been established.

This technique has been employed for the past 6 years unchanged with two exceptions: (1) a right posterior oblique projection is sometimes preferred to the lateral projection especially in suspected cases of carcinoma of the head of the pancreas, but always a lateral projection is preferred when the initial frontal projection suggests pancreatitis; and (2) for 6 months in 1963, having heard of Boijsen's work,[12] the simultaneous opacification of the celiac and superior mesenteric arteries was accomplished with little technical difficulty. This had the disadvantage of (a) greater arterial risk associated with the local punctures, (b) loss of directional or collateral study, and (c) the nonvisualization of a short segment of occluded artery since, unless the roentgen-ray beam is perpendicular to the short obstruction, filling from both directions may obscure a short segmental occlusion. The increased density in the pancreas is also partially lost from the increase in background density of the overlying stomach and small bowel. In insulinoma suspects and probably in gastric masses such as leiomyomas the double catheter technique may be indicated. We have no positive cases in this category.

Filming for 95 per cent of cases is adequate at 2 films per second for 3 seconds, 1 film per second for 3 seconds, and 1 film every 4 seconds for the remainder of the 12 cassettes, permitting use of the simpler changers such as the Sanchez-Perez cassette changer. If available, the Franklin or Schönander cassette changers, enabling exposure of more films over the 18 to 20 second interval, are ideal for the larger, busier department, since the typical findings are occasionally, although rarely, seen on only 1 or 2 films of a fairly large series. Due to the fact that contrast material delivery rates of 10 cc. per second are ade-

quate for the selective work and only 20 cc. per second are occasionally required for a supplementary catheter aortogram, almost any of the automatic injectors is satisfactory.

We have found that, with very little experience and a proper lordotic secondary curve on the catheter, the anterior positioning of the primary or end curve is easily accomplished with the patient in the supine position. Where a loss of the lordotic curve occurs in this position, plastic sponges may be inserted under the lumbar vertebrae to produce this curve or a catheter without a secondary curve may be used. The celiac axis is better catheterized for only a short distance to avoid subintimal injection and false narrowing of the artery at its origin that can resemble cuffing. It is of interest that in the cases where there was subintimal injection there were no adverse sequelae except for the discomfort of the patient which lasted some 5 to 20 minutes. If the catheter at this low injection pressure does recoil into the aorta another catheter with a larger primary or tip curve is inserted by means of the guide wire through this same puncture site. It is essential to always have a back flow of blood from a selectively catheterized organ to avoid intramural injections or embolization. A right side hole 6 to 8 mm. from the tip gives a more even distribution of the contrast material in the celiac axis than does a single end hole since more contrast agent usually is seen in the splenic artery.

Preparation of the patient throughout the 10 year period has consisted of hydration, thorough explanation to the patient of the procedure, and premedication with intravenous sodium pentobarbital (nembutal) and diphenhydramine hydrochloride (benadryl) immediately before beginning this study. If the clinician insists on an analgesic, morphine sulfate is preferred since meperidine hydrochloride (demerol) may lower the blood pressure and the possibility of thrombosis could be increased.

In approximately 3,600 percutaneous catheterizations 3 local arterial occlusions were the only severe complications encountered. These occlusions were all successfully corrected by local endarterectomy and all resulted in return of the dorsalis pedis pulse. Since 2 of the 3 occurred in the first 300 cases at which time the thin wall No. 16 needle and yellow Ödman catheter were used, a thin wall No. 18 needle and green Ödman catheter have been substituted for the past 8 years to give the smallest possible needle-catheter combination. This is thought to be of considerable value as the only other occlusion was in fact partial, in a young woman. At surgery her problem was shown to be due to fibrin which rolled up on the outside of the catheter as the latter was withdrawn from the artery producing a filling defect at the site of puncture.

Heparin sodium is not used except in thoracic aortography or cerebral vascular selective catheter injections. For procedures with long catheters rather frequent saline-heparin flushes are employed. Heparin sodium was used for the first 3 years and one can now appreciate a significant decrease in the size of the hematomas since this agent was omitted. Of course, the one thrombosis in the past 8 years, although it was an incomplete or fibrin ball obstruction, might have been avoided by the use of heparin sodium.

In hypertensive patients hematomas may be a real problem, since usually digital compression is used until all leakage has stopped. However, only 2 serious hematomas were observed in our entire group: an axillary hematoma which liquefied and required evacuation 10 days later; and an obstruction of the femoral vein which developed from a femoral artery leak that occurred 3 hours after it had sealed. Both of these complications were in very old hypertensive patients. Hematoma or loss of control of the puncture site presents no difficulty as long as the inguinal ligament is carefully studied and the puncture site chosen 1 to 2 cm. inferior to this line. The

use of a large sand bag or dressing produces much larger hematomas and it is not thought to be a good technique since only with the hiding of the hematoma can an arterial leakage continue without the patient's or physician's notice.

Indications for arterial, capillary and venous angiography include various pathologic states of the pancreas, liver, spleen and small bowel as well as the diagnosis of upper quadrant masses. These will be discussed under angiographic findings and in the illustrations.

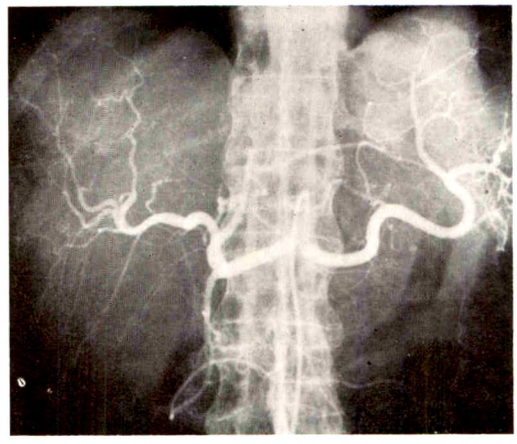


Fig. 1. Normal anteroposterior celiac arteriogram for orientation. Following the percutaneous insertion under local anesthesia of a preformed catheter, the tip is inserted into the celiac axis. The hepatic artery comes off to the right. The gastroduodenal artery originates perpendicular to the hepatic artery and descends parallel and slightly to the right of the vertebral column. The splenic artery courses to the left in a "lazy S" curve with loops above and below a line extending from the origin of celiac artery to the hilus of the spleen. The hepatic artery distal to the origin of the gastroduodenal artery extends directly into the liver without deviation. Note the relatively small size of the pancreaticoduodenal arteries, and the origin of the right gastroepiploic artery which arises from the gastroduodenal artery and extends along the greater curvature of the stomach.

### ANGIOGRAPHIC FINDINGS

A thorough knowledge of the normal vascular anatomy of the liver, spleen and pancreas is paramount to any accuracy in visceral angiography. The variations must also be known in view of the 45 per cent of cases which have a different blood supply from the remainder. The best anatomic dissection references are given in the excellent paper by Lunderquist[32] and there is no need to repeat these in this paper (Fig. 1).

The angiographic findings in various pathologic states will be described primarily from the authors' series rather than the literature which is in considerable disagreement primarily because of the small series having been reported and the variation in technique. One cannot stress the importance of the technique enough, as one is dealing with avascular pathology for the most part and a slight over- or underpenetration of the roentgenogram and minimal motion will obscure the typical findings. Most of the interest in visceral angiography has centered around the diagnosis of pancreatic carcinoma and a brief review of the pathology of this tumor will indicate the problem.

Pancreatic malignancies are almost all adenocarcinomas and most frequently originate from the ductal system.[20,36] This results in a scirrhous type of neoplasm with or without cystic or gelatinous degenerative components.[26] The softer medullary tumors, originating primarily from acinar tissue, undergo necrosis and hemorrhage early. More detailed pathologic findings are readily available[20,36,54] for the interested reader. Thus the problem in pancreatic angiography is that we are dealing almost entirely with a tumor which necrotizes and forms cysts or gelatinous material; therefore, demonstrable pathologic vessels are few. The vascular cellular tumors seen in the kidney and brain are represented in the pancreas by a very small proportion of the total number of malignancies. Secondary signs, rather than tumor stains or vessels, will remain the mainstay of angiography for pancreatic carcinoma until a suitable pharmacologic agent is found to enhance this sparse vasculature.

A study of the salient angiographic findings in 15 pathologic entities studied by celiac angiography is of value in indicating

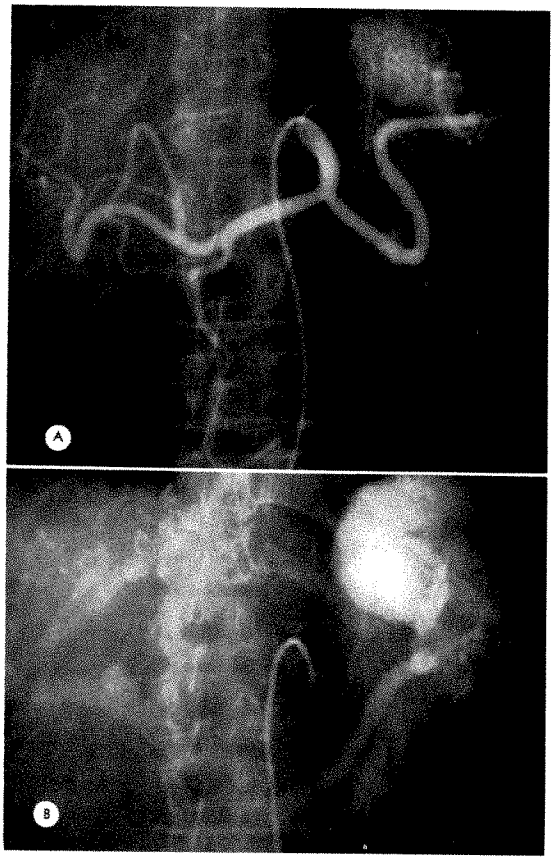


FIG. 2. (*A* and *B*) Splenic vein displacement and invasion by carcinoma. The splenic vein is depressed by a mass in the tail of the pancreas. There is slight irregularity of the splenic vein suggesting invasion. The arterial phase (*A*) demonstrates cuffing of the gastroduodenal artery confirming an extensive carcinoma.

a field which is large and which requires rapid interpretation of the film during angiography to determine what other injections should be done at that sitting. A brief outline is as follows:

A. Adenocarcinoma of the pancreas (usual type)
   1. Displacement of the arteries or especially of the veins such as the splenic (Fig. 2, *A* and *B*; and 6, *A* and *B*)
   2. Encasement (localized stenosis) of the arteries or the veins, primarily the arteries in the head and body of the pancreas and the veins in the body and tail (Fig. 2, *A* and *B*; 3*C*; and 4)
   3. Complete obstruction of arteries or veins due to invasion (Fig. 5, *A* and *B*)
   4. Tumor vessels—uncommon
   5. Tumor "stains" due to retention of contrast material in the capillary phase (Fig. 6*B*)
   6. A surrounding area of pancreatitis is often seen—this is a difficult problem at present

B. Cystadenoma, malignant or benign, and angiosarcoma
   1. Marked displacement of vessels without cuffing or obstruction
   2. Marked increase of vascularity
   3. Prominent capillary stain with slow clearance time
   4. Prominent venous drainage pattern

C. Islet cell tumors
   1. No displacement of arteries
   2. Occasional minimal irregularity of the arterial branches in the tumor
   3. Prominent and very prolonged capillary stain

D. Pancreatitis (Fig. 7, *A–C*; 8, *A–C*; and 9, *B* and *C*)
   1. Increased number, tortuosity, and size of anatomically identifiable arteries
   2. Displacement of the arteries proportional to number 3
   3. Increased volume of the pancreas
   4. Increased capillary stain with irregularity and mottling
   5. Early arteriovenous shunting
   6. Straightening of the arteries and less capillary blush in the burned-out or quiescent chronic pancreatitis case
   7. Pseudocyst formation (Fig. 9, *B* and *C*)
      a. Increased number of arteries but these are spread over a larger area
      b. Decreased diameter of the arteries
      c. Often surrounding area of 1 to 5 above
   8. Irregularity of the arterial lumen—very rare

E. Primary hepatic neoplasm
   1. Uncommonly numerous large tumor vessels, arteriovenous shunting, capillary stain, increased size of hepatic artery, and portal vein invasion
   2. Displacement of the arteries, small or no tumor vessels demonstrable and tumor stain uncommon (exception—malignant carcinoid)

F. Common duct carcinomas
   1. Elevation of the hepatic artery

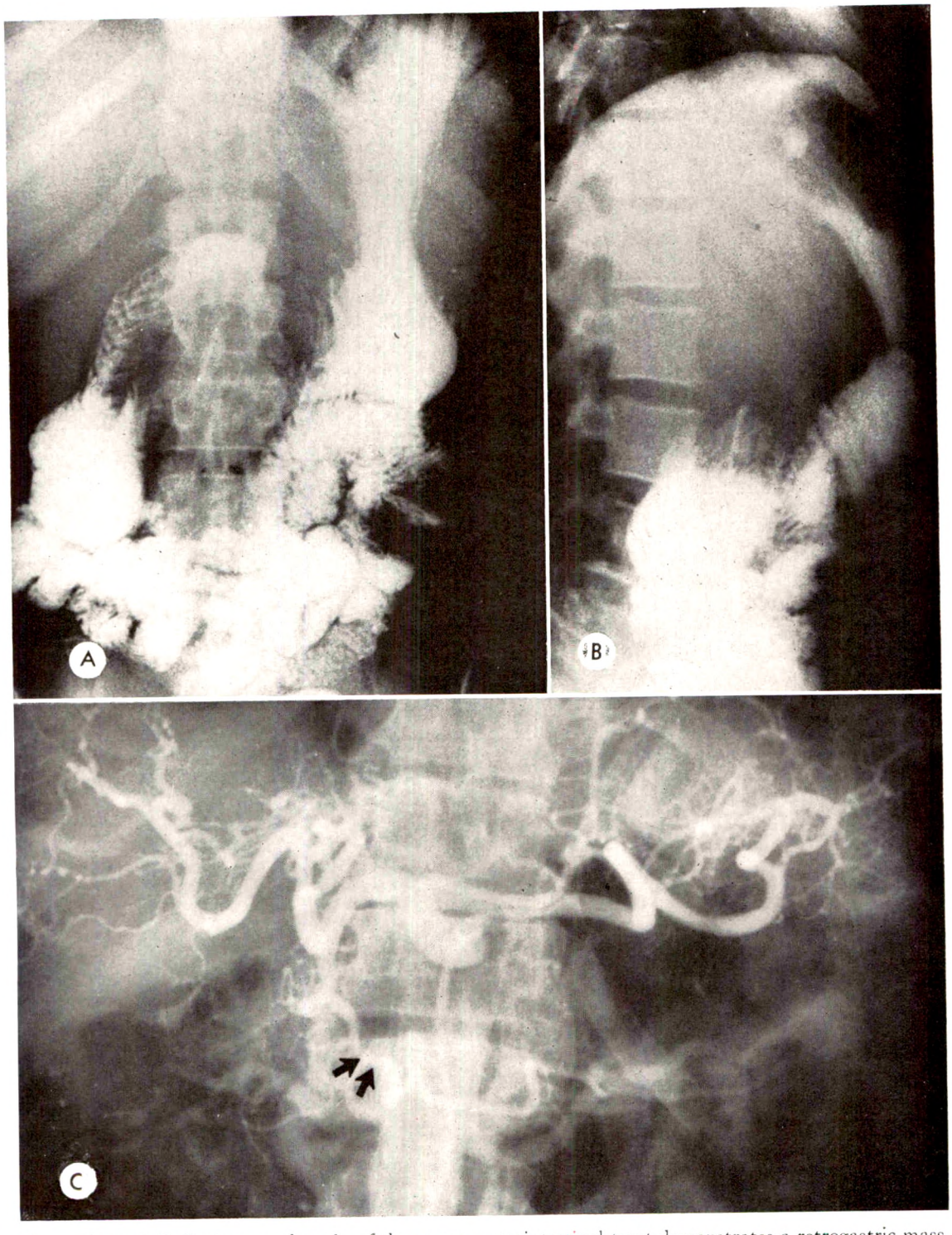



Fig. 3. (*A* and *B*) Barium meal study of the upper gastrointestinal tract demonstrates a retrogastric mass. (*C*) Mesenteric arteriography shows extensive carcinoma, with segmental, fairly symmetric arterial narrowing (cuffing, arrows) seen in the distal gastroduodenal and proximal gastroepiploic arteries. Minimal eccentric cuffing is also noted in the distal splenic artery below the 11th rib indicating extensive carcinoma subsequently confirmed by surgery. These examples of cuffing (localized lumen constriction) lack the irregularity of atherosclerosis.

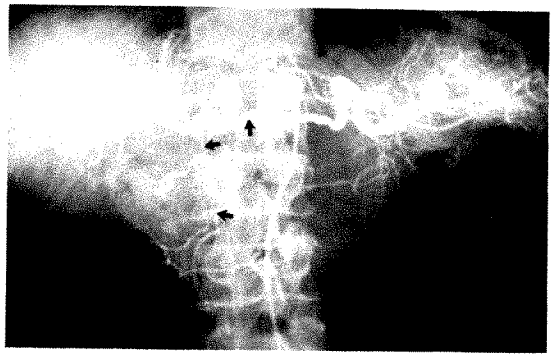


FIG. 4. Carcinoma. Cuffing of the proximal hepatic artery (tumor encasement, upper mid arrow) and severe cuffing of the gastroduodenal and pancreaticoduodenal arteries (left arrows).

2. Straightening of the gastric duodenal artery due to 1
3. Capillary stain rare

G. Cirrhosis of the liver
1. Stretched or straightened hepatic arteries (early or fatty stage)
2. Increased tortuosity of the hepatic arteries (late or fibrotic stage)
3. Mottling in the capillary or hepatogram phase
4. Venous signs of portal hypertension
5. Enlargement of the splenic artery and its radicles

H. Cholangitis and hepatitis
1. Enlarged and straightened arteries
2. Dense hepatogram
3. Severe mottling during the capillary phase

I. Portal hypertension
1. Increased size of the splenic and portal veins
2. Increased size of the spleen
3. Decreased linear flow rate or reverse flow in the portal circulation
4. Collateral venous pathways
5. Splenic or portal vein obstruction

J. Splenic rupture
1. Displacement of the spleen
2. Mottled opacification of the spleen with larger nonopacified areas near the rupture site
3. Increased flow rate through the spleen
4. Pooling of contrast agent in the spleen or adjacent area
5. Defect in the splenic contour

K. Celiac and hepatic aneurysms

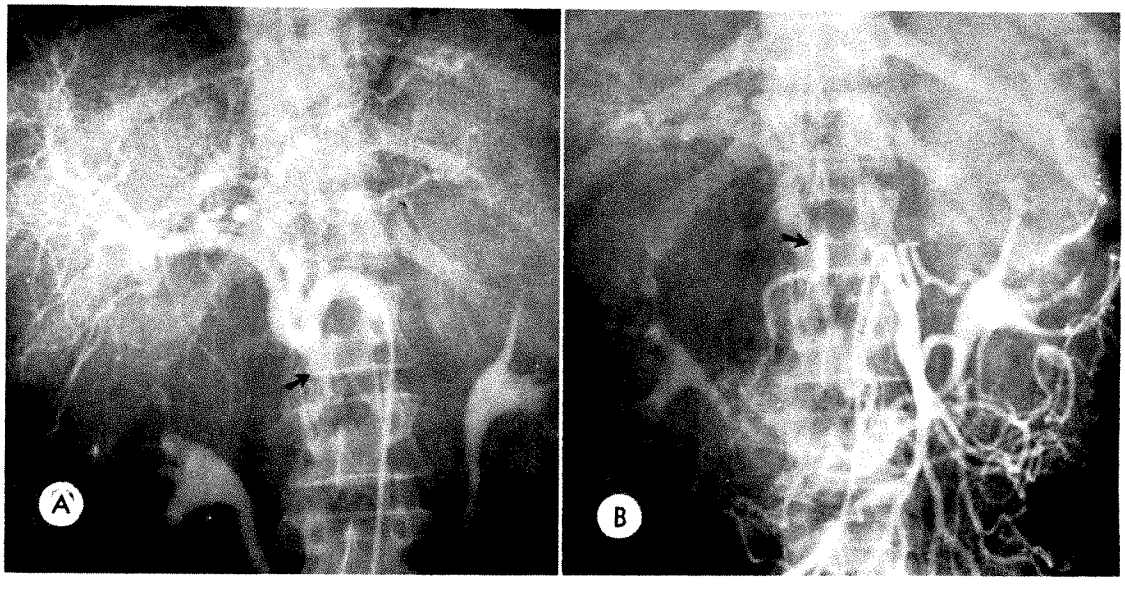



FIG. 5. Vessel obstruction due to carcinoma. (*A*) Selective hepatic arteriogram shows occlusion of the gastroduodenal artery (arrow). (*B*) Occlusion of the gastroduodenal artery (arrow) is demonstrated with retrograde filling following superior mesenteric artery injection. A short segmental obstruction like this can be observed by consecutive (not simultaneous) injection of the celiac and superior mesenteric vessels. Increased vascularity is present in the uncinate process and there are also a few sinusoids demonstrated, thereby further corroborating the diagnosis of carcinoma.

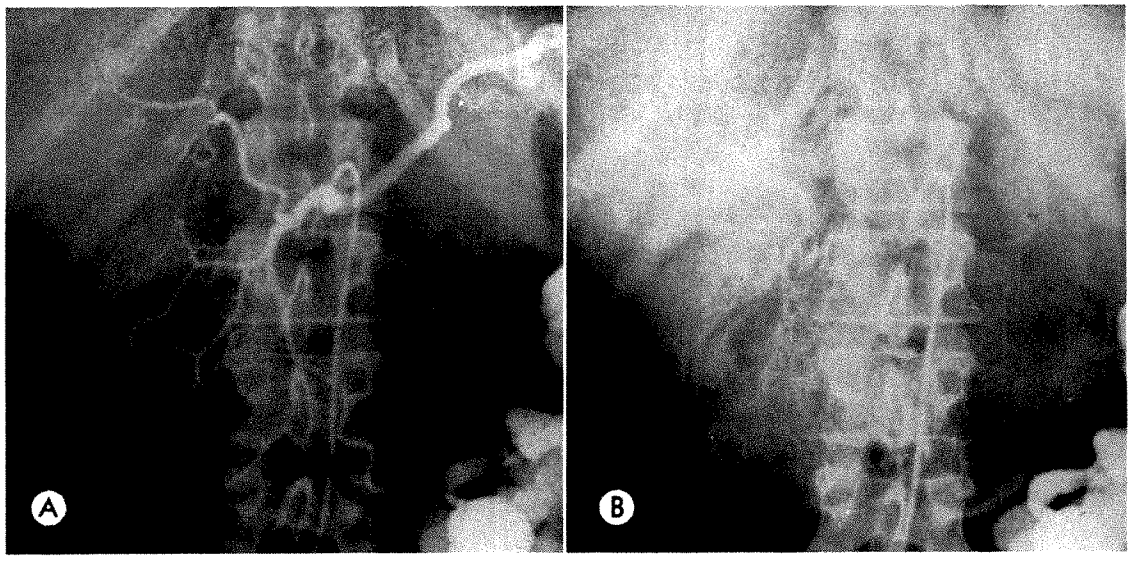



FIG. 6. (*A* and *B*) Increased vascularity due to cystadenocarcinoma. Marked increase in size of the gastroduodenal artery and the pancreaticoduodenal artery with displacement and spreading. There is a very poorly defined tumor stain (*B*).

L. Upper gastrointestinal hemorrhage
   1. Visualization of the mucosa after the normal capillary phase has ended
   2. Local persistent pooling of the contrast agent intraluminally

M. Postoperative status of splenorenal and portacaval venous shunts

N. Postoperative status of splenorenal arterial shunts

O. Left upper quadrant retroperitoneal sarcomas and adrenal cortical carcinomas (Fig. 10, *A* and *B*)
   1. Very few tumor vessels
   2. The splenic artery often supplies blood to these invasive tumors
   3. Very bizarre tumor vessels that are supplied from several organs in addition to the splenic and lumbar arteries

There are many other entities that have been diagnosed by celiac angiography, but this oversimplified outline is intended to cover the uses in which clinicians have shown the most interest during this 10 year period. We have cited a few pertinent articles in the bibliography on the more uncommon lesions included in this outline; however, all diagnostic signs have been found in the series presented with the exception of the insulinomas.

## ANGIOGRAPHIC CORRELATION WITH SURGICAL OR AUTOPSY FINDINGS

In the early part of this study errors were not uncommon with most of these due to: (1) the expectation of vascular capillary stain in pancreatic malignancy as one finds it in renal and cerebral malignancies; (2) staining of the normally overlapping duodenal wall in the region of the pancreatic head or staining of the jejunal wall in the pancreatic tail area. The right posterior oblique projection was of great help in the former and the latter remains troublesome especially in the presence of anomalous blood supply.

When it became apparent that pancreatitis generally produces similar angiographic signs as one sees in brain and kidney malignancy except for the absence of tumor vessels correct interpretation became much more accurate. This now approaches 85 per cent for carcinoma of the head of the pancreas and 60 per cent for all pancreatic carcinomas proven by surgery

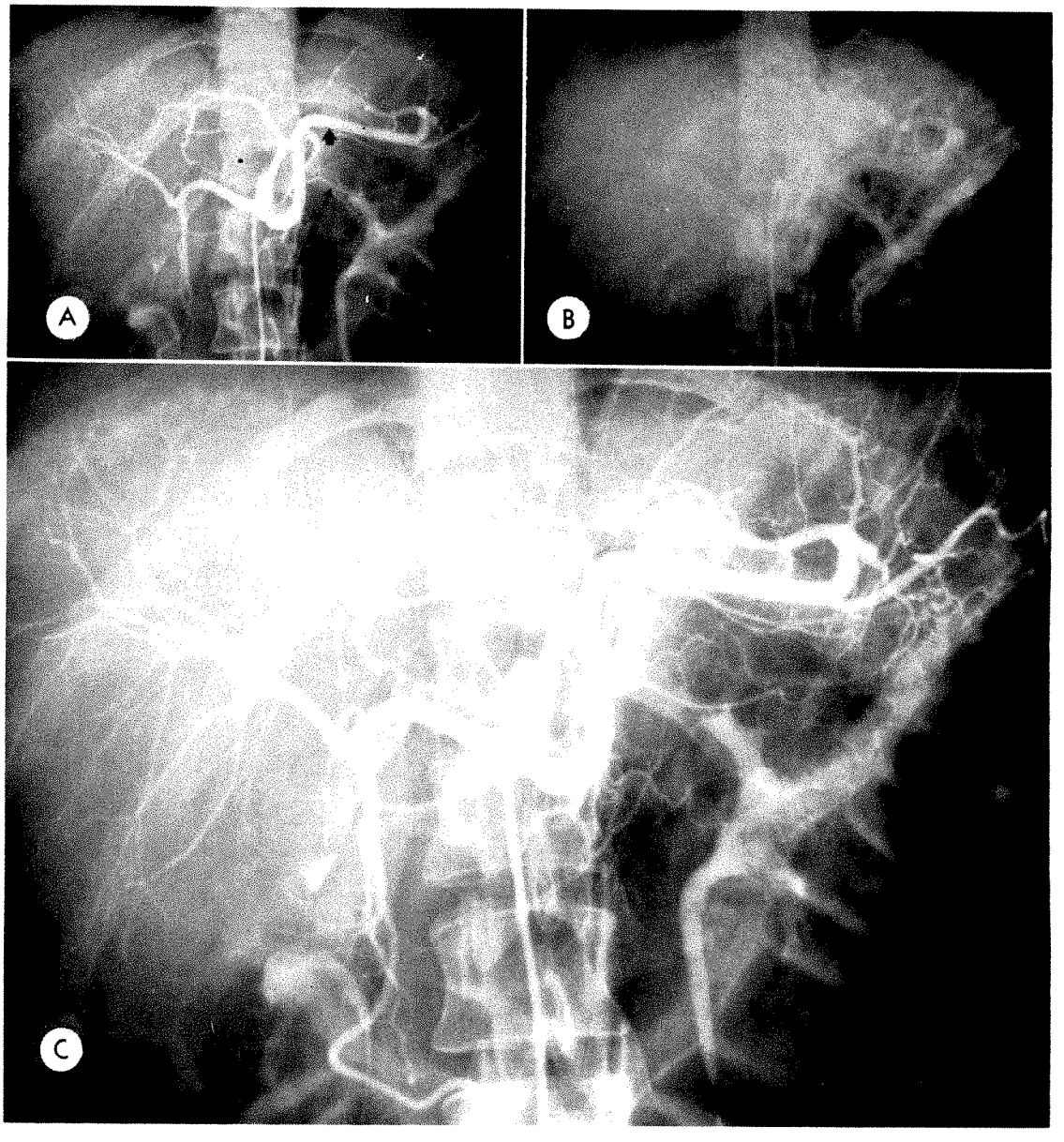



FIG. 7. (*A* and *B*) Increased vascularity due to pancreatitis. There is increase in the size of identifiable transverse pancreatic artery and the branches of the dorsal pancreatic artery, with elevation of the splenic artery and splenic vein (thick arrow). There is likewise increase in number of the smaller arteries (thin arrows). The diagnosis was pancreatitis in the tail extending into the body. (*C*) Close-up view of *A* demonstrating the numerous dilated vessels to better advantage.

or autopsy. Diagnosis of pancreatitis is possible in approximately 90 per cent of the cases when one accepts surgical and strong laboratory data as confirming evidence. The diagnosis of secondary pancreatitis distal to a small obstructing carcinoma remains a real problem and perhaps the use of pharmacologic agents will be the solution. Likewise, the diagnosis of small pancreatic carcinomas is unsatisfactory from the standpoint of the patient, referring physician, and angiographer. The other entities such as splenic and portal vein pathology, cirrhosis, hepatic malig-

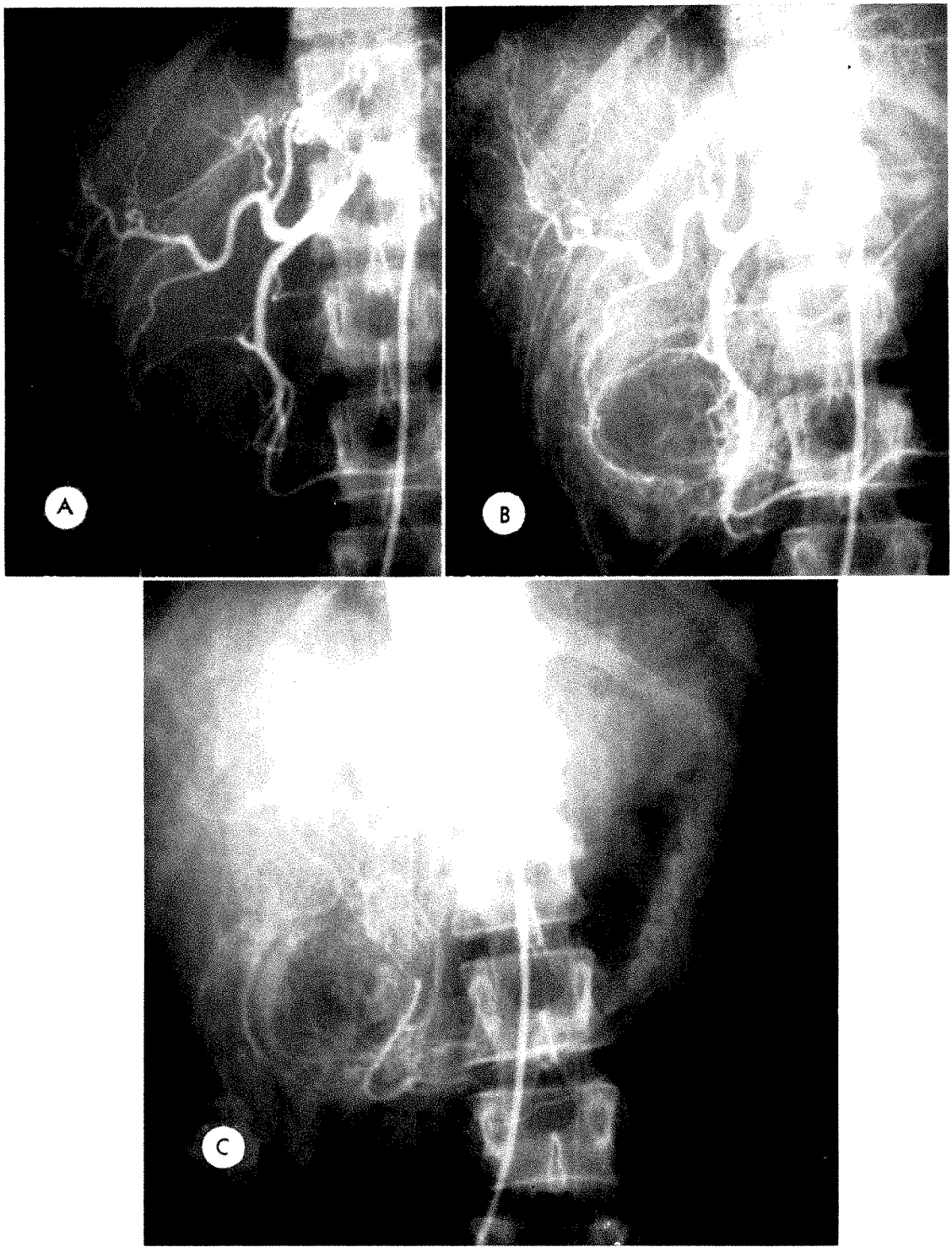



Fig. 8. (*A–C*) Hemorrhagic pancreatic cyst and pancreatitis. A young nurse with injury to the duodenum from a steering wheel revealed hemorrhagic pancreatic cyst which is outlined by dilated vessels. It is of interest that the laboratory data were continually negative in respect to pancreatitis, although she had marked symptomatology.

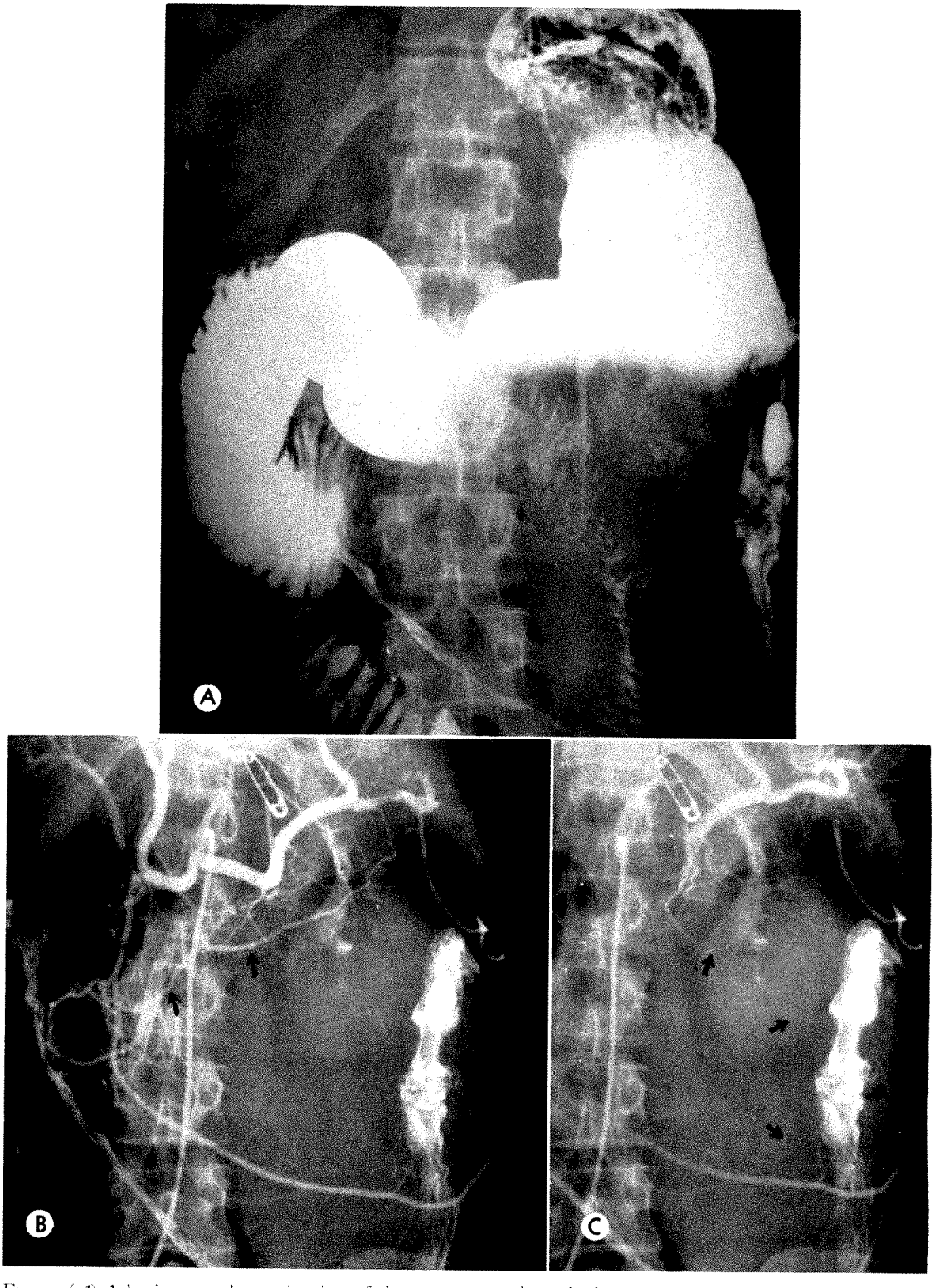



FIG. 9. (*A*) A barium meal examination of the upper gastrointestinal tract demonstrates a mass in the left midabdomen of undetermined nature. (*B* and *C*) Celiac arteriograms reveal a pathognomonic finding of pseudocyst of the pancreas, with long, narrow, stretched branches from the dorsal pancreaticosplenic

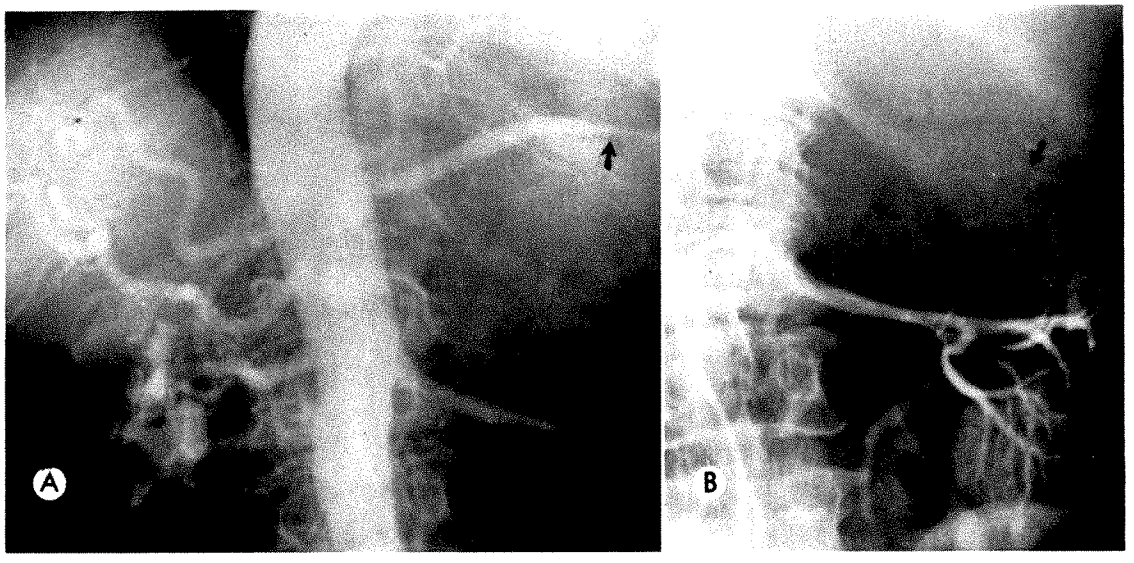



FIG. 10. (*A* and *B*) Retroperitoneal sarcoma. Cuffing of the spleric artery (arrow in *A*) without real significant displacement, and a very great displacement of the renal artery. Sinusoids in the tumor mass (arrows in *B*). The retroperitoneal sarcoma received blood from the tail of the pancreas and the upper pole of the kidney.

nancies, islet cell tumors, pancreatic cyst adenomas, splenic rupture, upper gastrointestinal hemorrhage, and hepatic or celiac aneurysms constitute about 80 per cent, the primary difficulty being the false diagnosis in portal venous hypertension due to reverse flow from intrahepatic block or a Budd-Chiari (veno-occlusive) syndrome. Injection of the superior mesenteric artery after reviewing the celiac axis angiograms is necessary, unless portal vein patency is demonstrated.

The tremendous strides in neuroradiology have occurred largely because of the expanded knowledge of normal anatomy. Similarly, one can expect more accuracy as angiographers and anatomists depict the *in vivo* anatomy of the celiomesenteric complex more accurately. One can never expect to use the very valuable modality of vessel diaplacement, as one does in intracranial lesions when the visceral vessels vary 45 per cent in their distribution and equally so in their spacial relationships. Generally speaking, the above listed angiographic findings must be relied upon rather than the easier measurement techniques of other diagnostic angiographic procedures.

## DISCUSSION

Over the course of 10 years we have been greatly encouraged by the use of visceral angiography in the difficult upper abdominal diagnostic problems. The observation of only 3 significant local complications in 3,600 percutaneous catheterization studies attests to the safety of the method. A steady increase in diagnostic acumen, as the needed meticulous technique and interpretative skill increased, has also been apparent.

The patient with pancreatic carcinoma often presents late with a multifaceted

artery that anastomose with the gastroepiploic artery (arrows in *C*). Note the tremendous transverse pancreatic and ascending pancreaticoduodenal artery (arrows in *B*) indicating associated pancreatitis in the head and body of the pancreas.

clinical picture, and the laboratory and radiographic procedures usually do not yield the needed accuracy in the diagnosis of a large number of cases.

The upper gastrointestinal barium study is still the safest, easiest and for late stages of malignancies the best study. We believe that all patients with upper abdominal complaints should have angiography or secondly duodenal drainage study only after a satisfactory upper gastrointestinal examination has been carried out. If this examination gives equivocal or negative results in the face of strong clinical findings, then visceral angiography appears to be an excellent choice. One of the most outstanding articles on conventional barium evaluation is the recent excellent contribution of Beranbaum.[6]

Raskin[46] reported that the moderately difficult and tedious duodenal aspiration combined with secretion and cytologic studies is the most valuable. Others[24] disagree. However, it is unlikely that this complex and uncomfortable procedure will become commonplace in the smaller institution where many of these cases are first seen. Having done both, we find visceral angiography faster and considerably easier technically.

Radioisotope techniques are still wanting for a satisfactory isotope with specific uptake in the pancreas and peristalsis may be a great source of trouble.[30,46]

The greatest problem for angiography of the pancreas remains in the primary carcinoma-secondary pancreatitis combination. That this combination does occur has been pointed out on autopsy studies by Brown *et al.*[16] An excellent recent monograph by Dreiling *et al.*[19] and the 1963 Symposium on Pancreatitis[51] in Marseilles indicate the lack of specific tests to evaluate pancreatic disease.

Cystadenomas of the pancreas, whether benign or malignant, are characterized by their very large vessels as described above and only the angiosarcoma could possibly be confused with them as Brunschwig[17] has mentioned. However, these tumors are rare, but their identification, localization and blood supply are readily determined by angiography.[57] Olsson[42] and Bookstein and Oberman[14] have given evidence of this.

SUMMARY

Of 3,600 percutaneous arterial catheterizations over the past 10 years, 900 have been for abdominal visceral problems, primarily pancreatic; 400 of these have been proven by surgery or autopsy.

A summary of the technique of celiac and superior mesenteric arteriography, together with the morbidity, correlation with tissue proof, and the pertinent useful signs is presented. Various pathologic entities are briefly described.

In pancreatic carcinoma, early diagnosis is difficult because of the variability in the clinical picture. Especially with carcinoma of the tail and body of the pancreas symptoms of sufficient severity are often late in appearance, and this important fact is directly related to the poor result.

Once clinical suspicion of pancreatic carcinoma is raised, and barium gastrointestinal study is negative, celiac and, if indicated, superior mesenteric angiography would appear to be the next basic step. Our results with such a procedure are discussed.

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# GALLSTONE ILEUS*

## A REVIEW OF THE ROENTGENOGRAPHIC FINDINGS AND REPORT OF A NEW ROENTGEN SIGN

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THE plain film roentgenographic diagnosis of gallstone ileus is usually based on the demonstration of any one or all of the following signs: (1) gas in the gallbladder or biliary tree; (2) ectopic location of a gallstone; and (3) partial or complete small bowel obstruction.

These roentgen signs are not difficult to demonstrate and when gallstone ileus is considered in the differential diagnosis, the roentgen diagnosis is frequently made.

The growing number of geriatric patients and the redistribution of this older population from municipal to private hospitals will result in a different group of physicians seeing the patients who most frequently have gallstone ileus. It is the purpose of the authors to emphasize a new roentgen sign which will alert the radiologist to the presence of gallstone ileus and to review the roentgen signs and clinical features of this disease.

The new sign is called the "loop obturation pattern," consisting of a characteristic appearance of the loops of the small bowel in the supine roentgenogram of the abdomen. There is minimal to moderate gaseous distention of the fluid-filled loops of bowel. The valvulae conniventes are prominent and do not show evidence of extreme effacement. Loops of bowel containing only fluid are well delineated. The gas-containing loops not infrequently assume a radical configuration. Essentially the sign pertains to the characteristics of obstructed small bowel in which there is a small amount of gas associated with moderately distended, fluid-filled, obstructed small intestine (Fig. 1).

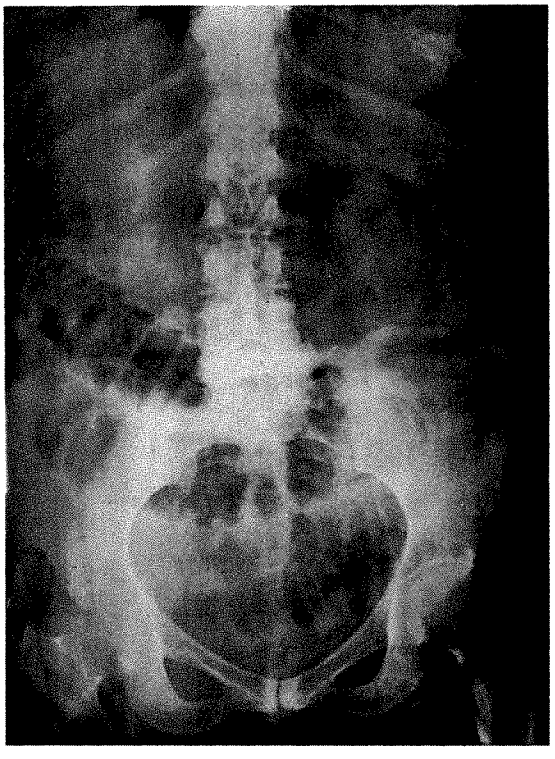


FIG. 1. Gallstone ileus due to mid ileal cholelith. This supine roentgenogram shows well outlined fluid-filled loops of small bowel. Minimal to moderate gas is present. The valvulae conniventes are well outlined. The loop obturation pattern is positive.

Sloan[11] described the gross mucosal appearance of fixed specimens of the mesenteric small bowel. With marked distention of the jejunum, a consistent pattern was demonstrated in which the mucosal folds assumed a circular or semicircular configuration. The distance between each fold increased as distention of the loops of jejunum increased. These folds were much less constant in the distended ileum and were only occasionally seen here.

Frimann-Dahl[5] in describing obstructed loops of small bowel which contain much fluid and little gas indicated that the gas

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collected between the mucosal folds and was visible as parallel translucencies. When the loops were circular or semicircular, the roentgen pattern assumed a radical arrangement, an appearance that he compared to rosary beads. This was often seen in cases of small bowel volvulus in his series of bowel obstructions.

Williams'[13,14] description of the supine roentgenogram of simple small bowel obstruction with only moderate gas accumulation distinguished the same finding but referred to it as a "positive stretch sign." The sign is due to trapping of gas beneath the stretched valvulae conniventes and reliably indicates dilated small bowel, calling attention to the fluid-filled loops. When only a minimal amount of gas is present, the roentgenograms are also diagnostic but the findings are more subtle.

In characterizing the small bowel appearance in gallstone obstruction, Rigler *et al.*[8] described unusual visibility of the small intestinal segments with classic distention of several loops of small bowel by gas and relative absence of gas in the colon in most cases. Occasional cases did not show characteristic distention of the small bowel because obstruction was partial or intermittent.

In reviewing a series of supine roentgenograms of 35 instances of gallstone ileus with the obturating stone in the mesenteric small bowel, the loop obturation pattern was frequently encountered. A series of parallel radiolucencies outlined the stretched valvulae conniventes, frequently little gas accumulated in the small bowel and the fluid-filled loops of the small bowel were exceptionally well delineated. The pattern conforms to Frimann-Dahl's description of much fluid and little gas seen in the volvulus, and Williams' description of a positive stretch sign as a manifestation of minimal gas accumulation in a fluid-filled obstructed bowel.

The characteristic loop obturation pattern was present in 30 instances (86 per cent). Gas was present in the gallbladder or biliary tree in 21 instances (60 per cent). An ectopic calculus was present in 9 instances (25 per cent). The loop obturation pattern was present in 11 instances (31 per cent) where the other 2 signs were negative. Two patients had 2 separate gallstone obstructions.

## CLINICAL FEATURES

The incidence of gallstone ileus varies from 0.4 to 5.0 per cent of all cases of bowel obstruction.[1,2,6,7] Welch[12] reviewed the relative frequency of different types of obstruction in 1,035 consecutive cases of small bowel obstruction at the Massachusetts General Hospital from 1947–1955. He reported gallstone ileus frequency as 0.8 per cent. This equaled the incidence of mesenteric thrombosis and obstruction caused by internal hernia; was half the frequency of volvulus and obstruction caused by regional enteritis; and was ⅛ as frequent, as was intussusception.

Welch in 1958, felt that gallstone ileus was decreasing in incidence as compared with the period prior to 1929 because of the more frequent performance of cholecystectomy. This decreased figure will probably be balanced by the gradually increasing life expectancy.

The above incidence figures which are classically referred to in most gallstone ileus reports may be misleading when faced with a diagnostic problem. Brockis and Gilbert[1] reviewed 179 cases in the literature and showed that no fewer than 24 per cent of small bowel obstructions, excluding strangulation obstruction in patients over 70, were due to gallstone ileus. The figures for the 50–60 and 60–70 age groups were 10.1 per cent and 14.5 per cent incidence respectively. Eighty-eight per cent of the instances of gallstone ileus occur in women. This compares with the incidence of cholecystitis in women.

Symptoms vary with the site of perforation of the gallstone into the gastrointestinal tract, size of the stone, site of lodgement in the intestinal tract, and the degree of obstruction. Vomiting, pain and tenderness in the abdomen, abdominal pain

other than right upper quadrant occur in half of the patients. Up to 50 per cent of the patients have antecedent gallbladder disease. Many have an obscure clinical course with periods of vague abdominal discomfort.[4,7]

The intermittent attacks of abdominal cramps, nausea and vomiting occur for several days. When the stone impacts, a full blown syndrome of bowel obstruction may become clinically recognizable.

"Ileus" may be an inexact term to describe obstruction because this would suggest paralysis of the bowel and not dynamic obstruction. The paralysis may eventually occur in the late stages of obstruction as it frequently does in other types of mechanical obstruction. Despite this lack of preciseness most physicians understand the implications of mechanical obstruction.

## ROENTGEN SIGNS

### GAS IN THE BILIARY TREE OR GALLBLADDER

Flat, upright, left lateral decubitus, prone and coned roentgenograms frequently demonstrate gas in the biliary tree (Fig. 2). The left lateral decubitus roentgenogram is particularly valuable. Occasionally when the biliary tree is not outlined gas may fill the gallbladder. A stone which leaves a biliary fistula need not obstruct the bowel. The presence of internal biliary fistula alone is not pathognomic of gallstone ileus. Incidence of artificial biliary fistulae of as high as 3 per cent is reported in patients operated upon for biliary tract disease. Other causes are perforation of a peptic ulcer, and extension of a malignant tumor from the pancreas, stomach or colon.[2]

Gas in the biliary tree may also be caused by gas-forming organism infection, sphincterotomy, stone passage through the sphincter of Oddi and patulous sphincter of Oddi seen in aged patients.[9]

The suspicion of gallstone ileus or the finding of gas in the biliary tree is sufficient indication for the use of barium or water soluble contrast medium.

An interesting finding in internal biliary fistula with intravenous cholangiography has recently been reported by Shehadi.[10] The biliary ducts assume a frothy pattern due to admixture of gas with the contrast agent. The use of cholangiography is not suggested as a routine method of diagnosis, but a bizzare clinical picture should certainly prompt the performance of this examination early in the work-up.

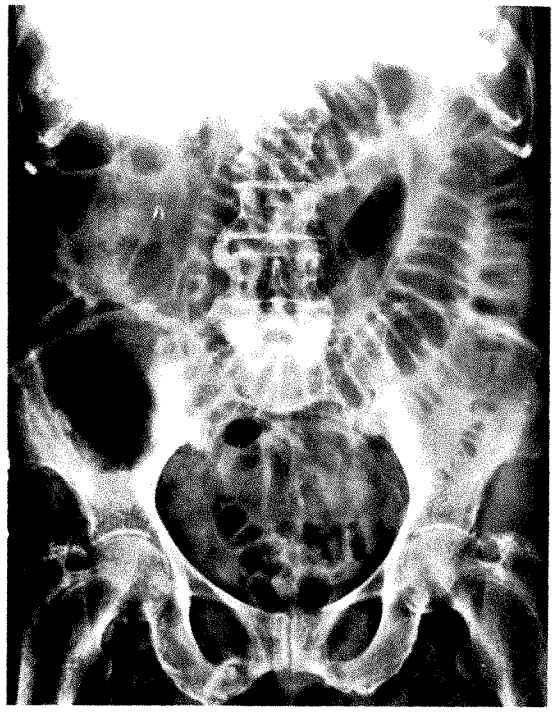


Fig. 2. Gallstone ileus due to obturation by mid ileal gallstone. Gas is present in the gallbladder and suggested in the biliary tree. The cecum contains gas indicating partial mechanical obstruction. The exceptionally well delineated loops of small bowel contain moderate gas and assume a radial configuration.

### ECTOPIC BILIARY CALCULUS

Frimann-Dahl[5] localized the obturating gallstone in 50 per cent of his series. Rigler *et al.*[8] demonstrated the stone in 4 of 10 cases and detected changed position from previously known sites in 2 of 10 cases (Fig. 3).

The presence of the roentgen triad: mechanical small bowel obstruction, internal biliary fistula and ectopic gallstone, is considered pathognomonic. Further ex-

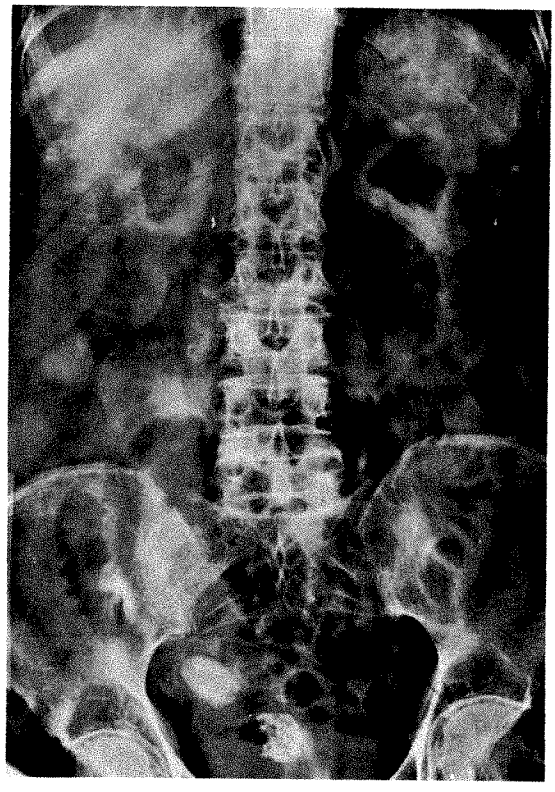


FIG. 3. Gallstone ileus. The laminated biliary calculus is in an ectopic location in the right lower quadrant of the abdomen. Fluid-filled loops of small bowel indicate obstruction in a supine roentgenogram.

amination with contrast material when these findings are all present is time wasting. However, when only 1 or 2 of the triad is noted, use of opaque contrast material is certainly helpful. The biliary fistula will be demonstrated in all but a few cases in which the gallstone may have migrated through the common bile duct or where the fistula has become scarred and spontaneously closed. The disappearance of previously identified stones from the gallbladder area is uncommon, but when demonstrated is most helpful (Fig. 4, *A–C*).

### OTHER TYPES OF MECHANICAL OBSTRUCTION

The supine roentgenographic findings of other types of mechanical small bowel obstruction are well documented in the radiologic literature. The presence of minimal gas in distended fluid-filled loops of bowel in simple obstruction is an infrequent occurrence.[14] Moderate gas accumulation in simple obstruction is usually attributed to either "high" or early obstruction. The classic description of simple nonstrangulated obstruction is that of moderate to severe gaseous distention of the small bowel (Fig. 5). This is in distinction to the lesser amount of gas accumulated in gallstone ileus.

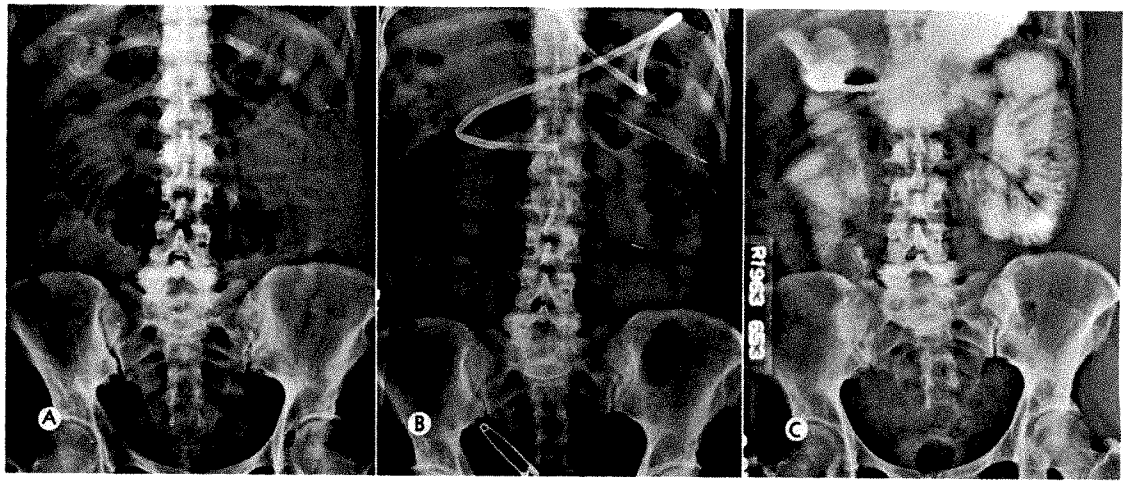



FIG. 4. (*A*) Gallstone ileus due to impacted gallstone in the terminal ileum. Loop obturation pattern present. Note laminated calculi in the right upper quadrant. (*B*) Sixteen days postoperatively abdominal cramping was present for 20 hours. Gallstones are absent from the right upper quadrant. One stone is demonstrated in right mid abdomen. (*C*) Oral water soluble contrast medium outlines the calculus and demonstrates the biliary duodenal fistula. Two gallstones were found in the terminal ileum 6 inches proximal to previous enterotomy. (Courtesy of George Jacobson, M.D.)

Strangulated obstruction frequently shows fluid-filled loops of intestine. The specific criteria of pseudotumor, "coffee bean" loop and loss of normal mucosal pattern are reliable criteria for strangulation (Fig. 6).

## DISCUSSION

Kirkland and Croce[6] in a review of 154 cases between 1940 and 1960 state that the correct preoperative diagnosis was made in 54 instances (35 per cent). Using the criteria mentioned by Rigler, plain roentgenograms and opaque contrast studies will result in 80–90 per cent accuracy in preoperative diagnosis.

The frequent appearance of the loop obturation pattern in gallstone ileus should alert the radiologist to the necessity for additional roentgenograms or opaque contrast studies to confirm the diagnosis, particularly in the patient with a bizarre clinical picture when only a supine roentgenogram of the abdomen has been taken.

Frimann-Dahl[5] described a slow progression of the obstruction in gallstone ileus. In the early stages fluid levels were frequently absent. The fluid-filled coils of bowel were seen as long dense bands in the supine roentgenograms.

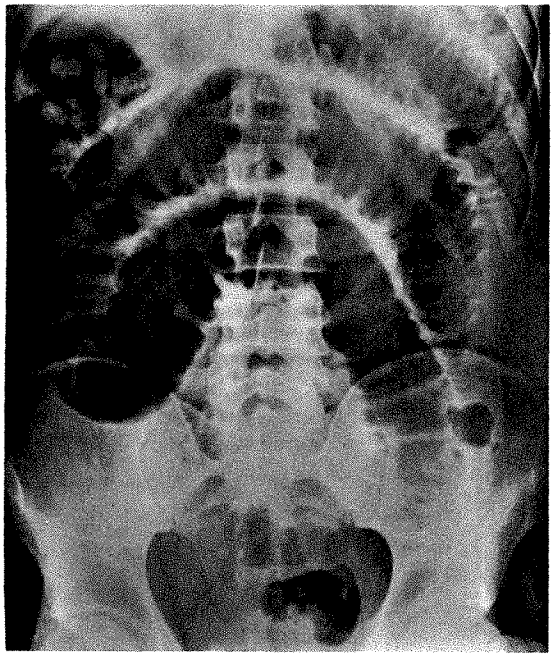


Fig. 5. Distal ileal obstruction due to multiple adhesions. The large amount of gas present in the distended loops of small bowel in this supine roentgenogram is infrequently seen in gallstone ileus.

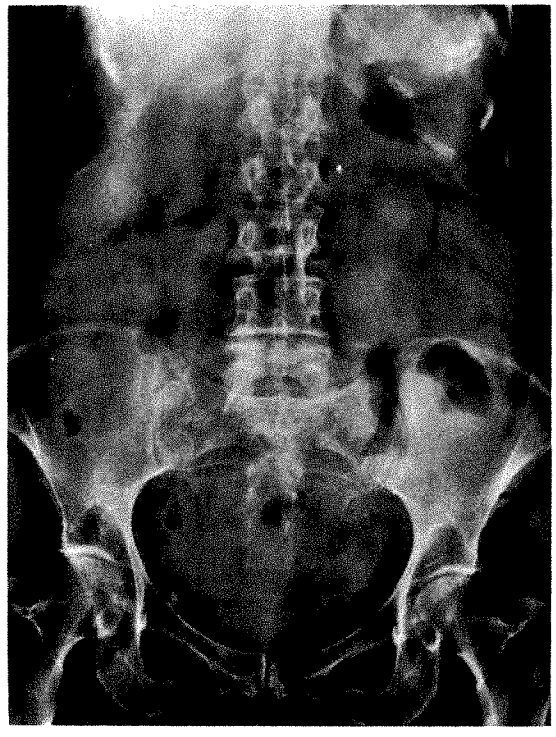


Fig. 6. Gangrenous small bowel obstruction. This plain roentgenogram shows minimal gas in the small intestine. Fluid-filled loops are visible throughout the abdomen. The gas fails to outline the valvulae conniventes. The only similarity to the loop obturation pattern is the clear delineation of fluid-filled loops of bowel.

Copious vomiting, electrolyte depletion in the older individual and resulting atonia of the bowel may explain the frequent appearance of the loop obturation pattern. The intermittent obstruction and perhaps some associated reflex inhibition caused by the obstructing stone are other possible explanations.

The underlying mechanism for the loop obturation pattern is the relatively small amount of gas and moderate amount of fluid in the distended small bowel. Although this pattern is present in obstruction other than gallstone ileus, its occurrence is infrequent.

Awareness of the frequency of this type of obstruction in older patients is further stressed because of the estimated increase in population over 65 years of age from 18,000,000 in 1960 to 25,000,000 in 1985.[3]

SUMMARY

A new roentgenographic sign of gallstone ileus is described. This is called the "loop obturation pattern" and refers to the characteristic appearance of the small bowel occurring in the supine plain roentgenogram of the abdomen. There is minimal to moderate gaseous distention of the small bowel; prominent valvulae conniventes which do not show extreme degrees of effacement; clear delineation of the fluid-containing loops of bowel and frequently a radial configuration of the loops of bowel.

The loop obturation pattern occurred in 86 per cent of 35 proven instances of gallstone ileus. In 31 per cent, the loop obturation pattern was present in the absence of gas in the biliary tree or an ectopic calculus. Recognition of the loop obturation pattern is not pathognomonic of gallstone ileus but should alert the radiologist to its presence and provide indication for additional plain roentgenography or contrast medium study for confirmation.

The clinical and supine roentgenographic criteria for gallstone obstruction are reviewed.

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VOL. 101, NO. 2

# THE USE OF BARIUM SULFATE SUSPENSIONS IN THE STUDY OF SUSPECTED MECHANICAL OBSTRUCTION OF THE SMALL INTESTINE*

*By* SIDNEY W. NELSON, M.D.,† *and* ANTHIMOS J. CHRISTOFORIDIS, M.D.‡
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MOST surgeons admit that mortality and morbidity are still distressingly high in patients with mechanical obstruction of the small intestine.[1,2,3,7,9,10,14,17,18,21,22] Although there has been a steady decrease in mortality during the last 25 years, it is likely that most centers still have a mortality rate around 10 per cent. Most patients tend to procrastinate in seeking the help of a family physician who then may have difficulty in making a clinical diagnosis. These delays often mean that the obstruction has been present for a day or two before the patient reaches the surgeon. Since mortality and morbidity are so closely related to early operative relief of the obstruction, it is the purpose of this communication to report our experiences in making earlier radiologic diagnoses of mechanical obstruction of the small intestine by the use of ordinary barium sulfate suspensions which are administered orally.

## THE REASONS FOR A MORE AGGRESSIVE APPROACH TO THE ROENTGENOLOGIC STUDY OF SUSPECTED OBSTRUCTION OF THE SMALL INTESTINE

It is well known that plain roentgenograms of the abdomen can clarify many diagnostic problems in patients with acute abdominal symptoms. In fact, intestinal obstruction of the mechanical type is probably diagnosed on the basis of plain roentgenographic findings about 50–60 per cent of the time.[9] However, in the remaining patients the plain roentgenograms are either normal or show such minimal abnormalities as to be of no value in making a definitive radiologic diagnosis.

With respect to the abnormal findings on plain roentgenograms of the abdomen, it is of great interest that Sloan[15,16] has shown experimentally that the roentgenologic changes following the production of nonstrangulating obstruction of the small intestine lag far behind the pathologic changes. This experimental work is important because it confirms the fact that plain abdominal roentgenograms may not be diagnostic of an obstruction of the small intestine until many hours have elapsed after its onset.

It is unfortunate that the diagnostic problem is often complicated by the fact that the clinical symptomatology may often be atypical or almost completely lacking in patients with intestinal obstruction. Thus, we now strongly encourage the oral administration of ordinary commercially available barium sulfate suspensions to clarify the question of a mechanical obstruction of the small intestine as soon as possible after the patient's admission to the hospital, particularly when the plain roentgenograms of the abdomen are not diagnostic (or may even be normal). If a mechanical obstruction in such patients remains undiagnosed during a temporizing regimen which may last 24–48 hours (or longer), a significant deterioration in electrolyte and fluid balance can occur, and the operative morbidity and mortality increase significantly as the duration of the obstruction increases.[9]

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Since even in the best hands the diagnosis of an obstruction of the small intestine often presents great difficulty[9,22] it seems only logical that we radiologists should further exploit a useful and relatively unexplored field of radiologic diagnosis in an effort to make earlier and more accurate diagnoses; *i.e.*, the use of inert well-suspended barium sulfate. We have been astonished and gratified by the diagnostic precision and safety of this method, and we wish to emphasize the relatively short period of time (30 minutes to 6 hours) usually required to make such positive diagnoses.

## PREVIOUS STUDIES

In previous experimental studies[12,13] comparing the use of a barium sulfate suspension and a water-soluble iodinated compound we have shown that the barium sulfate suspension is the safest and by far the

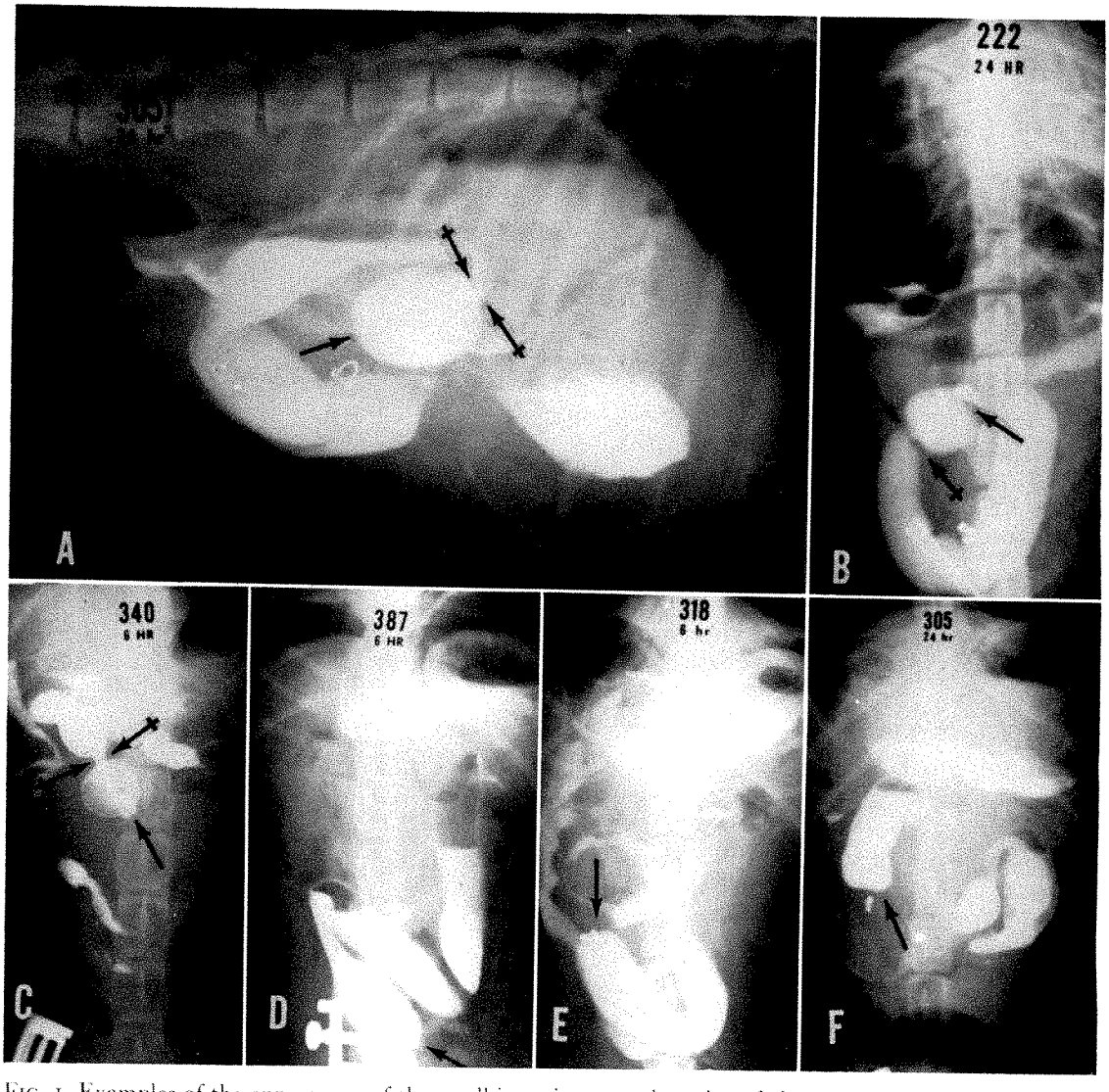



FIG. 1. Examples of the appearance of the small intestine near the point of obstruction (→) as shown in experimental studies on dogs with surgically produced simple mechanical obstruction. Note the "snakehead" appearance in those instances in which a deep peristaltic contraction (↔) happened to be close to the point of obstruction when the roentgen exposure was made (*A*, *B* and *C*). Compare with the more club-shaped appearance (→) of the loop proximal to the obstruction when the exposures happened to be made at a moment when a peristaltic wave did not happen to be present near the point of obstruction (*D*, *E* and *F*).

most reliable medium for demonstrating an obstruction of the small intestine (Fig. 1, *A–F*). Furthermore, our studies and those of others[8] convince us that the water-soluble iodinated compounds by virtue of their osmotic effects and tendency to cause emesis can produce minimal, but probably significant deleterious changes in the already precarious acid-base and fluid balance of patients with protracted emesis due to intestinal obstruction or other diseases. Conversely, the relatively inert and osmotically weak conventional barium sulfate suspensions do not have these disadvantages, and we have been using them with increasing frequency during the past 6 years in the study of patients in whom the plain roentgenograms do not clarify the diagnosis in suspected obstruction of the small intestine.

### OUR TECHNIQUE OF EXAMINATION

In those patients in whom the plain roentgenograms are not diagnostic, we now encourage the use of a 50 per cent W/V aqueous or saline suspension of commercially available barium sulfate preparations in ordinary amounts (200–300 cc.). Roentgenograms of the abdomen are obtained at approximately 1 hour intervals thereafter until the barium has entered the colon or until its progress becomes arrested in the small intestine.

Once a cupful of the barium sulfate suspension has been given to a patient, a fluoroscopic examination of the stomach and duodenum may be done if it appears that a high obstruction is likely. We use manual palpation to express barium from the stomach into the duodenum and jejunum in amounts adequate to fill the proximal part of the jejunum rather than to rely upon the patient's own gastric peristalsis to accomplish this, inasmuch as gastric peristalsis may be ineffective due to nausea or other factors. The manual method of getting the suspension to the point of obstruction can then result in a clearcut demonstration before the suspension is lost due to emesis. If vomiting of the suspension occurs before the diagnosis has been established we make another effort to have the patient ingest more of the suspension.

Following the fluoroscopic and spot filming procedures, roentgenograms of the abdomen are obtained at intervals determined by the suspected level of the obstruction: every 30–60 minutes if high obstruction is suspected (Fig. 3, *A* and *B*; 5, *A* and *B*; 6, *A* and *B*; and 8, *A* and *B*) and less frequently if a more distal obstruction is suspected (Fig. 2, *A–C*; 4, *A* and *B*; and 7, *A* and *B*). If the radiologist is available and has the time, he can frequently clarify a minimal or nonspecific finding seen on one of the roentgenograms by re-fluoroscoping the patient in an attempt to displace loops overlying any questionable finding or to spread suspicious loops apart in an attempt to get spot roentgenograms to help clarify a morphologic abnormality or a loop of intestine which appears larger in caliber than normal.

### THE ROENTGEN DIAGNOSTIC SPECTRUM OF POSSIBILITIES FOLLOWING THE ORAL ADMINISTRATION OF BARIUM SULFATE SUSPENSIONS WHEN OBSTRUCTION OF THE SMALL INTESTINE IS SUSPECTED

Since the symptomatology of intestinal obstruction can so frequently be mimicked by other conditions, a wide range of roentgen diagnostic possibilities exist in patients at first suspected of having mechanical obstruction of the small intestine. This realm of possibilities has not been adequately explored because of the erroneous idea that the use of barium sulfate must be avoided in any patient in whom there is the remotest possibility of a mechanical obstruction of the small intestine. However, well suspended commercial preparations of barium sulfate have been proven to be completely safe[5,6,11,12,13] and do *not* become inspissated proximal to a mechanical obstruction of the small intestine, but barium sulfate should not be given orally if there is a possibility of an obstruction in the distal one-half of the colon. When such a possibility seems likely, a roentgen study of the small intestine

should be preceded by a barium enema examination.

In Table 1 are seen the diagnostic possibilities which may be encountered in those patients with normal or slow transit times from the stomach to the cecum, and those associated with a complete arrest of the barium sulfate in the small intestine. We have learned that partial or temporarily complete mechanical obstruction can be demonstrated roentgenologically in patients with normal (Fig. 2, *A–C*; 5, *A* and *B*; and 8, *A* and *B*) or slow (Fig. 4, *A* and *B*; and 7, *A* and *B*) transit times. Therefore, a normal or slow transit time by itself does not rule out a temporary or partial mechanical obstruction of the small intestine. Since the point of a partial obstruction may be obscured by overlapping barium-filled loops of small intestine both proximal and distal to the lesion, we try to spread or displace these loops using fluoroscopic control, compression pads, etc., in our search for the obstructed point, particularly when there are loops of large caliber or questionable morphologic findings on the large roentgenograms of the abdomen.

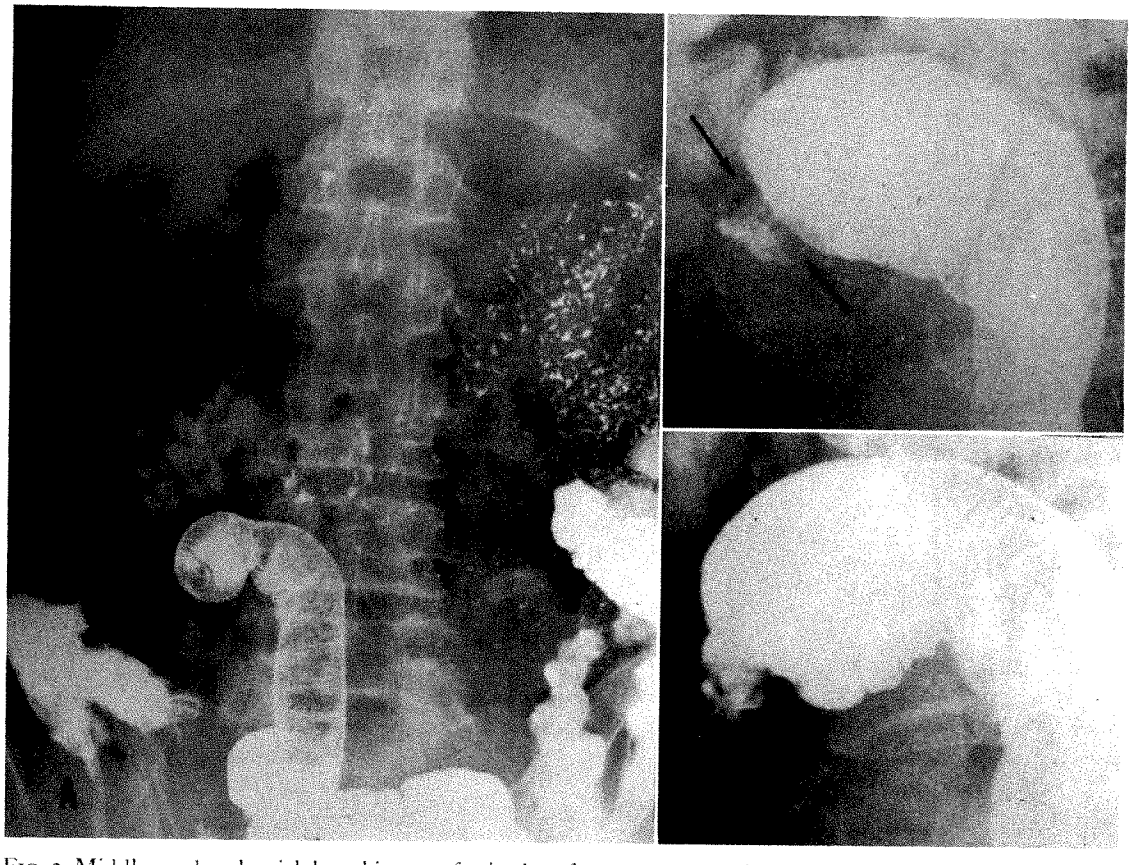


FIG. 2. Middle aged male with long history of episodes of severe crampy abdominal pain for which no explanation had been found on previous plain roentgenograms, upper gastrointestinal series, and barium enema study. During the last acute episode prior to operation a roentgenogram of the abdomen 2½ hours after the administration of barium sulfate showed the obstructed point (arrow in *A*) with the typical "snakehead" appearance. Spot roentgenograms (*B* and *C*) showed an extrinsic linear compression (between arrows in *B*) typical of an adhesion. Note the normal parallel mucosal folds (between arrows in *B*) where adhesion compresses the bowel. A few moments later during palpation a small amount of barium sulfate was made to pass into the narrow lumen (*C*). Subsequent serial roentgenograms were normal because all of the barium sulfate passed the point of obstruction and entered the colon during the next 2 hours. Thus, what was temporarily a complete obstruction suddenly "released" to allow the entire barium meal to enter the colon. However, the diagnosis of obstruction due to adhesion had been clearly shown.

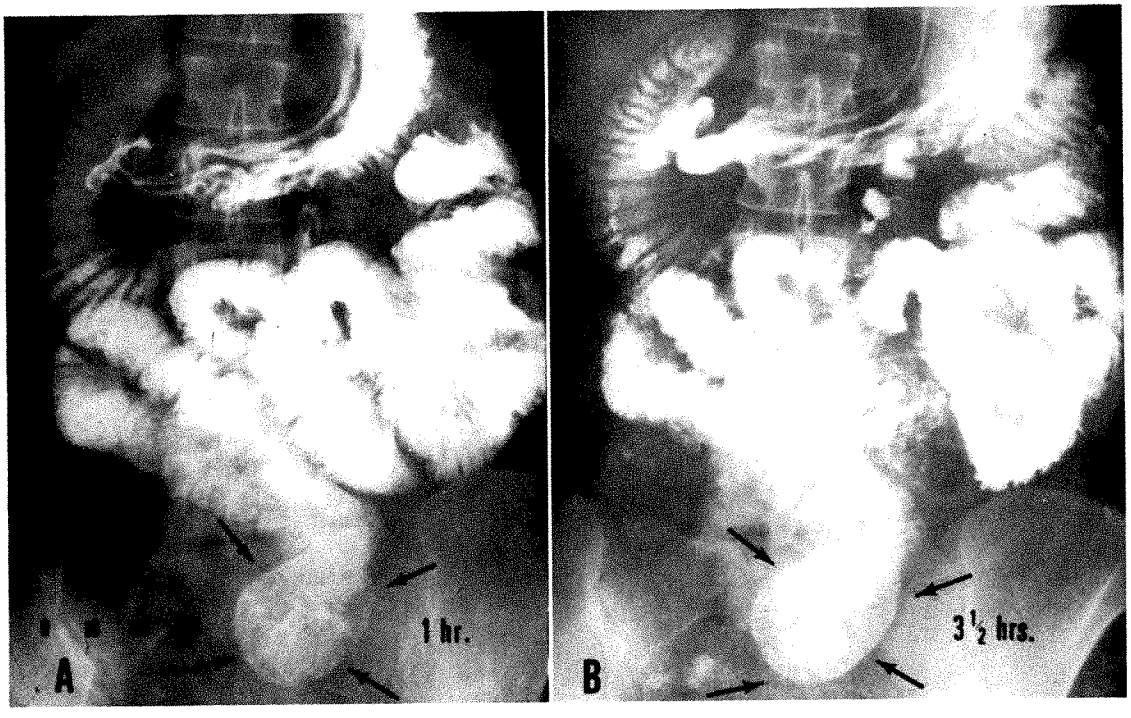



FIG. 3. This middle aged female had signs and symptoms which indicated a possible mechanical obstruction of the small intestine. Plain abdominal roentgenograms in supine and erect positions were normal. An examination of the small intestine with a barium sulfate suspension showed a club-shaped obstructed loop in the pelvis on the 1 hour roentgenogram (arrows in *A*). Barium did not pass beyond this point during the next 3½ hours (arrows in *B*). This appearance is typical for a complete mechanical obstruction. Note that the barium has become mixed with fluid and bowel contents proximal to the obstruction.

After perusing Table 1, it is obvious that the *one* diagnostic finding which is meaningful at the present state of our knowledge is the roentgenologic identification of the actual *point* of a partial or complete mechanical obstruction (categories indicated by asterisks in Table 1). In this type of case the radiologist's contribution can be prompt and decisive. Conversely, it is much more difficult, if not frequently impossible, to interpret the significance of the arrest of the contrast material in the small intestine in the absence of the demonstration of a specific *point* of obstruction.

### ROENTGENOLOGIC EVALUATION OF THE PATHO-PHYSIOLOGY OF INTESTINAL OBSTRUCTION FOLLOWING THE ADMINISTRATION OF BARIUM SULFATE

The use of barium sulfate in animals and man has helped evolve useful radiologic criteria with respect to the precise identification of the obstructed point, the nature of the obstructing lesion, and the understanding of some of the abnormal physiologic phenomena which occur proximal to it. For instance, in animals we learned that a short, widened club-shaped loop of bowel with a somewhat rounded distal end was characteristic of the appearance immediately proximal to the nonstrangulating mechanical obstructions we had produced (Fig. 1, *A–F*). Furthermore, as the duration of the obstruction increased, the caliber and length of this loop gradually increased as its tonus and peristaltic activity diminished. If the obstruction continues for more than 48 hours, the dilatation gradually spreads proximally to involve those portions of the small intestine which for the first 2 or 3 days after the onset of the obstruction were often of normal caliber. In fact, in the case of obstructions of less than 48 hours duration in dogs the peristaltic activity and caliber of the intestine proximal to a

simple nonstrangulating mechanical obstruction often remain relatively normal except for the moderate dilatation of the short club-shaped loop immediately proximal to the obstruction.

During our animal studies it was easy to recognize peristaltic activity on the roentgenograms (Fig. 1, *A–F*) and when a strong deep contraction happened to be roentgenologically visible close to the obstructed point, it looked like the "neck" of a snake, because the distended short segment of the intestine between this strong peristaltic wave and the obstructed point frequently resembles the "head" of a snake. Although the "snakehead" appearance (Fig. 1, *A*, *B* and *C*) is not observed in all obstructions since it is a somewhat coincidental event which may not be seen on every roentgenogram, its appearance is, nevertheless, so characteristic and striking that it is worth remembering. This appearance of the isolated barium-filled dilated club-shaped loop immediately proximal to the obstruction is diagnostic of a *mechanical* obstruction (Fig. 1–7).

## ROENTGEN DEMONSTRATION OF INCOMPLETELY OBSTRUCTING LESIONS OF THE SMALL INTESTINE

Although an incomplete obstruction may appear to be almost complete during a certain phase of the roentgenologic examination (Fig. 2, *A–C*; and 5, *A* and *B*), the barium suspension often passes beyond the point of partial obstruction. We have been misled by what appeared to be an almost complete mechanical obstruction (Fig. 5*A*), only to see the huge obstructed loop suddenly become decompressed as barium passes the point of temporary obstruction and passed into the normal appearing loops beyond (Fig. 5*B*). In such cases the obstruction has sometimes appeared to be complete throughout a fluoroscopic examination during which several spot roentgenograms (Fig. 2, *B* and *C*) were obtained during compression and manipulation.

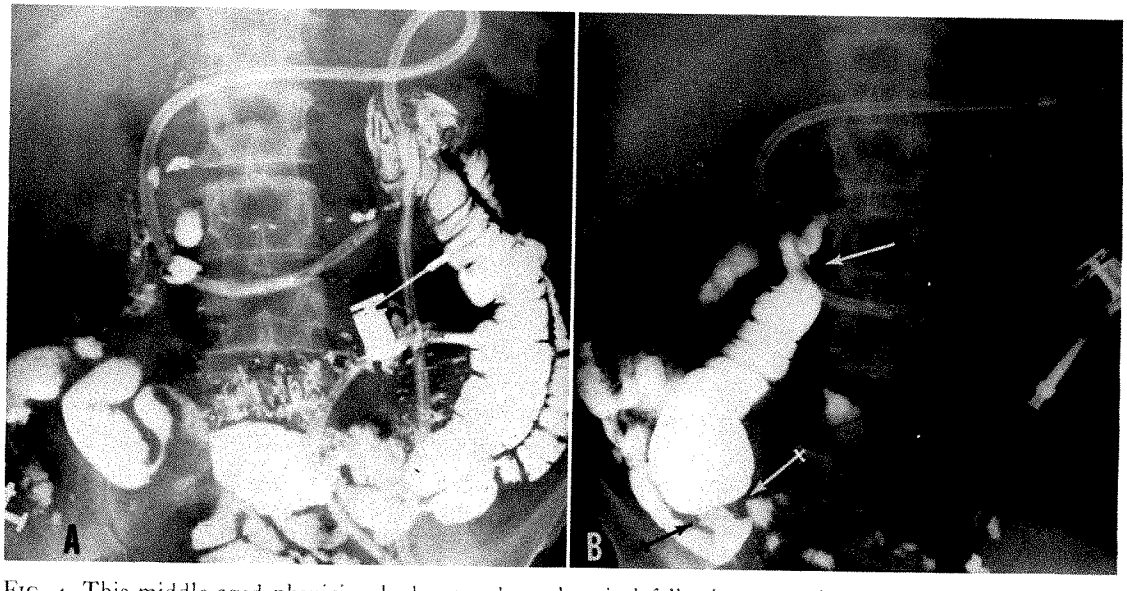



FIG. 4. This middle-aged physician had entered our hospital following operation at another institution at which a "small bowel neoplasm" had been encountered, but was not removed due to ventricular tachycardia which developed during the operation. Our first examination of the small intestine with an iodinated water soluble medium was completely nondiagnostic. Barium sulfate was then administered and roentgenograms obtained at intervals varying from 1 to 7½ hours thereafter. Periodic roentgenograms showed no lesions (*A*), until the roentgenogram made 6½ hours after the barium sulfate was given (*B*) showed the typical club-shaped rounded end of the obstructed loop due to an adhesion (upper arrow). Note typical extrinsic linear defect between opposing lower arrows. The terminal ileum distal to the obstruction is normal. All of the barium sulfate passed into the cecum.

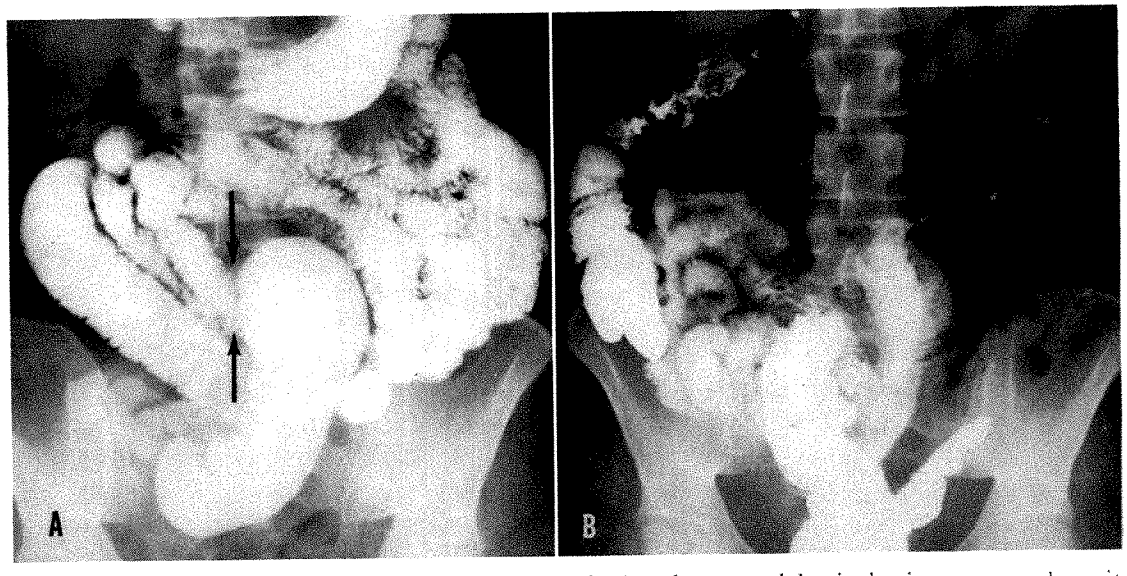



FIG. 5. A young female with a 3 year history of severe episodes of crampy abdominal pain, nausea and vomiting. Three previous hospitalizations at other hospitals had failed to clarify the etiology. Plain roentgenograms on admission to our institution were entirely normal. The roentgenogram made only 30 minutes following the oral administration of barium sulfate showed in the middle of the abdomen a club-shaped dilated loop typical of mechanical obstruction due to adhesion (between opposing arrows in *A*). Two subsequent roentgenograms showed marked diminution in size of this loop which, nevertheless, remained larger than normal. Films exposed later in the examination were entirely normal (*e.g.*, *B* was made 4 hours after *A*) and all of the barium sulfate passed the point of partial obstruction. At operation adhesions were found producing almost complete obstruction.

Thus, by studying obstructions in this way, we are able to obtain a graphic *in vivo* portrayal of the patho-physiologic nature of the clinical symptomatology which caused the patient to consult the physician. Such roentgenographic findings are all the more striking because they are so frequently seen in patients whose plain abdominal roentgenograms are entirely normal or equivocally abnormal.

The mechanism of how a temporarily complete obstruction is overcome is not well understood, but it may be related to peristaltic physiology; *i.e.*, each succeeding peristaltic wave probably becomes increasingly stronger, until one is finally able to overcome the resistance at the point of the obstruction. In other words, a spastic component of the obstruction may be overcome by the increasingly strong peristaltic waves, or possibly an obstruction may be released because of the breaking of small fibrinous adhesions which loosely bind contiguous walls of a kinked loop together. Whatever the cause or causes, this "release" of what for some time appears to be a complete obstruction of the small intestine *does* occur and is of interest because it teaches us that we may only have a fleeting opportunity to demonstrate a high grade obstruction which produces repeated and severe clinical symptoms (Fig. 5, *A* and *B*). We therefore recommend roentgenograms of the abdomen at planned intervals following the administration of barium sulfate for the study of a suspected obstruction of the small intestine.

It is usually not difficult to recognize obstructions due to adhesions because they often produce a typical extrinsic linear defect at the point of the obstruction (between opposing arrows in Fig. 2*B*; 4*B*; and 5*A*). When barium has passed such a point of partial obstruction, the small intestine distal to it will be seen to be normal in caliber, whereas the small intestine proximal to the point of obstruction varies from being markedly widened in caliber (Fig.

4*B*; and 5*A*) to normal in caliber (Fig. 2, *A–C*), the degree of widening depending upon the duration and the degree of the obstruction. It is interesting to note that the extrinsic linear defect of an adhesion which obstructs the small intestine is just as characteristic as is the extrinsic oblique linear defect on the posterior aspect of the barium-filled esophagus in a patient with an ectopic right subclavian artery.

Obstructing lesions of the small intestine due to inflammatory processes such as circumferential ulcers, neoplasms (Fig. 8, *A* and *B*), strictures due to regional enteritis or ectopic gastric mucosa (Fig. 7, *A* and *B*), etc., do not show the linear extrinsic defects so characteristic of adhesions. Obstructing or intussuscepting intraluminal polypoid neoplasms are often recognizable for what they are, and annular carcinomas of the small intestine usually have the same characteristic appearance as annular carcinomas of the stomach, esophagus, or colon. When such lesions produce obstructive symptomatology, a roentgenologic study using a barium sulfate suspension will frequently result in their recognition preoperatively, which is helpful to the surgeon.

### ROENTGEN SIGNS OF MILD TO MODERATE DEGREES OF LOCALIZED PARALYTIC ILEUS

In patients with mild to moderate degrees of localized paralytic ileus (*e.g.*, those with acute cholecystitis, pancreatitis, appendicitis, diverticulitis, etc.) the barium may pass through the small intestine at a very slow rate, but usually will reach the colon within 8–24 hours. Furthermore, the relatively small caliber of the characteristically localized segments of hypomotile bowel contiguous to the inflammatory

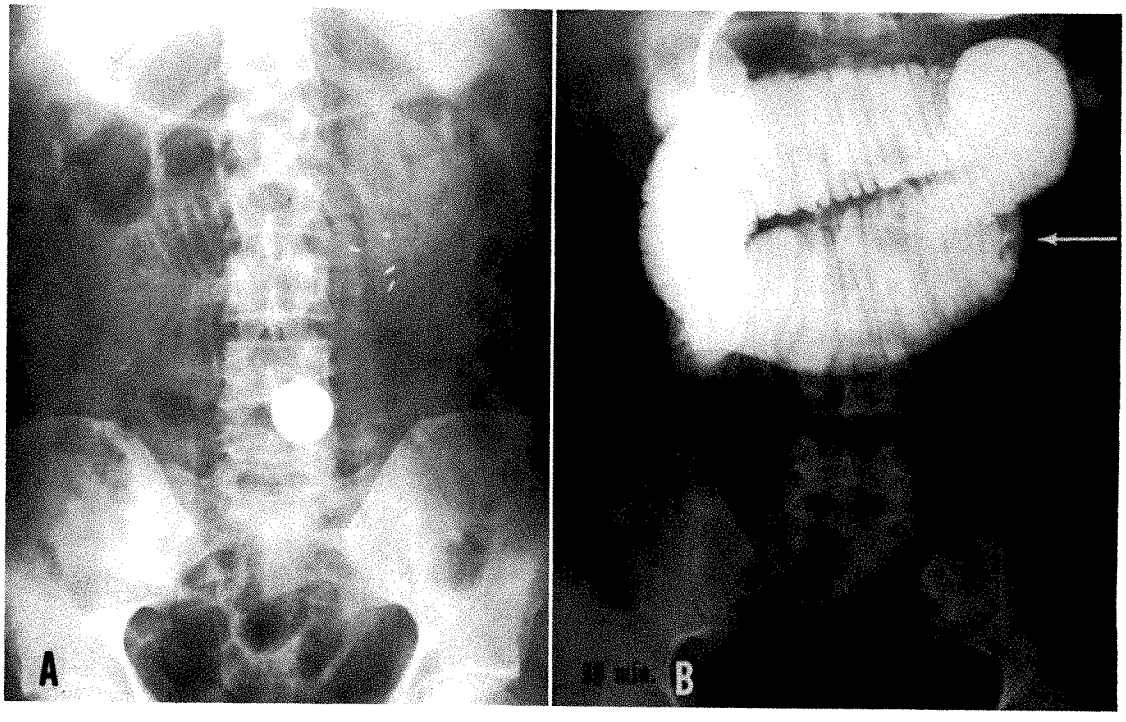



Fig. 6. This elderly male developed nausea and vomiting about 10 days following surgery for resection of an infarcted segment of jejunum due to a venous thrombosis. Plain abdominal roentgenograms showed a few short air-fluid levels in the middle of the abdomen, but were not diagnostic of mechanical obstruction or paralytic ileus. Supine roentgenogram (*A*) was not diagnostic of the obstruction. Barium sulfate was administered and all roentgenograms after the 30 minute roentgenogram (*A*) showed complete arrest of the barium sulfate in the proximal jejunum (arrow in *B*). At operation adhesions were found causing a complete mechanical obstruction in the proximal jejunum.

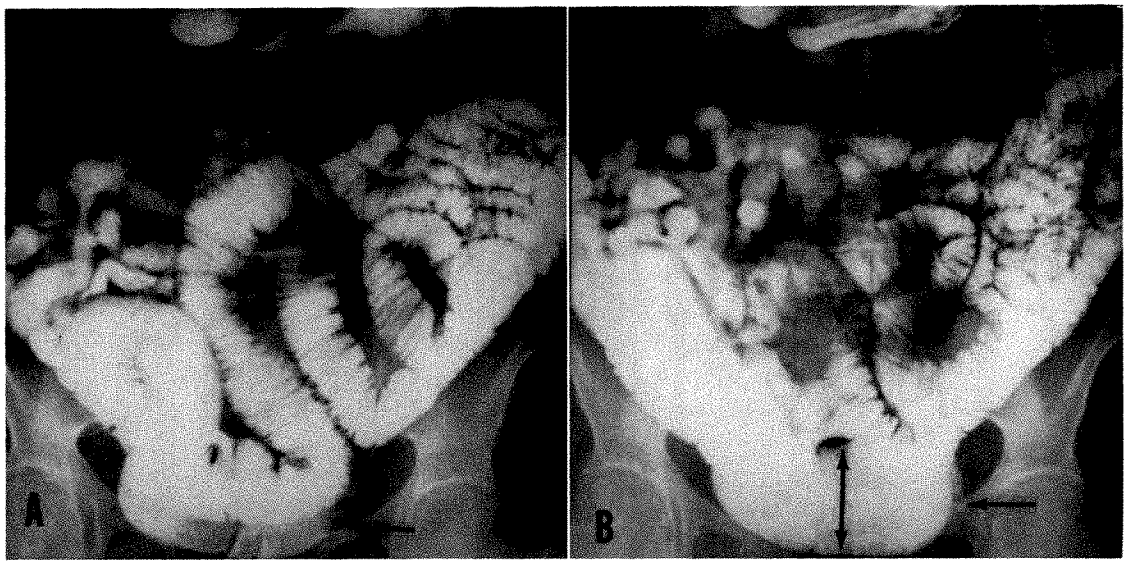



FIG. 7. An elderly white female patient with heart disease was admitted with the history of increasingly severe episodes of cramping pain, nausea, and vomiting, relieved after a few hours by the sudden passage of watery stools. She was normal between attacks. The trouble had begun 1 year earlier after having taken a diuretic pill for 3 months. We suspected circumferential small bowel ulcers due to KCl or thiazide diuretics. Barium sulfate was given orally and roentgenograms made 1½ hours later showed a dilated club-shaped loop deep in the pelvis (horizontal arrow in *A*). It could not be mobilized or profiled during fluoroscopy, and the appearance of this loop remained unchanged for several hours (horizontal arrow in *B*). Most of the barium sulfate passed beyond the partly obstructed point during the next 24 hours. The appearance was typical for a partial mechanical obstruction due to a benign lesion. At surgery a stenotic lesion was found, but it turned out to be due to ectopic gastric mucosa which had produced a stricture with a lumen only a few millimeters in diameter.

lesion (*e.g.*, inflamed gallbladder, pancreas, appendix, etc.) is in striking contrast to the wider and more numerous loops of bowel proximal to a long-standing mechanical obstruction or in patients with a strangulating obstruction.

### THE SIGNIFICANCE OF THE FAILURE OF THE BARIUM SULFATE SUSPENSION TO PROGRESS THROUGH THE SMALL INTESTINE

Whereas there can be no doubt about the presence of a mechanical obstruction when the point of obstruction is identified (Fig. 1–8), there is much doubt in our minds about our ability to determine the significance of the phenomenon in which barium sulfate enters the small intestine, becomes dispersed in several loops which contain large amounts of fluid, solids, and gas, but does not progress to a point of obvious obstruction. In some instances, most of the barium sulfate may remain in the stomach, and if so, it should at least alert the radiologist to the possibility that a strangulation may be present. This phenomenon of marked hypomotility which is seen in the presence of strangulation[19,20] is probably due to the severe systemic effects of the strangulated bowel (toxic, bacterial, etc.) and is, therefore, a type of severe paralytic ileus. We and others[4] have noted a marked diminution in peristaltic activity in the stomach and small intestine when strangulation has been present for more than 10 hours. However, prior to this time, the bowel proximal to a strangulated obstruction continues to have peristaltic activity which gradually diminishes and becomes minimal after 10 hours. Thus, the *presence* of peristalsis does not exclude strangulation, but the total *absence* of peristalsis should raise the possibility of a strangulation of at least 10 hours duration.

In addition to strangulating lesions,

there are other causes for the arrest of barium sulfate in the small intestine of patients in whom we cannot demonstrate a point of mechanical obstruction; *e.g.*, we have seen it in scleroderma and in some cases of moderate to severe paralytic ileus of various causes not related to strangulation. Furthermore, simple nonstrangulating mechanical obstructions of the small intestine which have been present for several days may become strangulated proximal to the obstruction because of the long-standing increase in intraluminal pressure, resultant compression of the blood vessels and eventual compromise of the circulation in the bowel wall. Furthermore, in the case of these mechanical obstructions of long duration, the bowel finally becomes so decompensated[15,16] (fatigued, atonic and dilated) that it is in effect acting like that of a patient with paralytic ileus. In fact, it is a bowel which is exhausted because of the long losing struggle to overcome a mechanical obstruction.

Thus, our as yet incomplete and continuing studies convince us that although stasis of the barium sulfate suspension in large fluid-filled atonic loops of small intestine should always raise the question of the presence of paralytic ileus due to strangulated bowel, it is not in our opinion diagnostic of it. However, if there is a complete failure of gastric emptying (gastric ileus) in the absence of an obstructing gastric or duodenal lesion, the large atonic fluid-filled loops are often due to stangulation, whether it is due to an intravascular obstructive lesion, volvulus, intussusception, or mechanical obstruction of long duration.

We have much more experimental and clinical work to evaluate before we can evaluate the significance of complete arrest of the opaque medium in the small

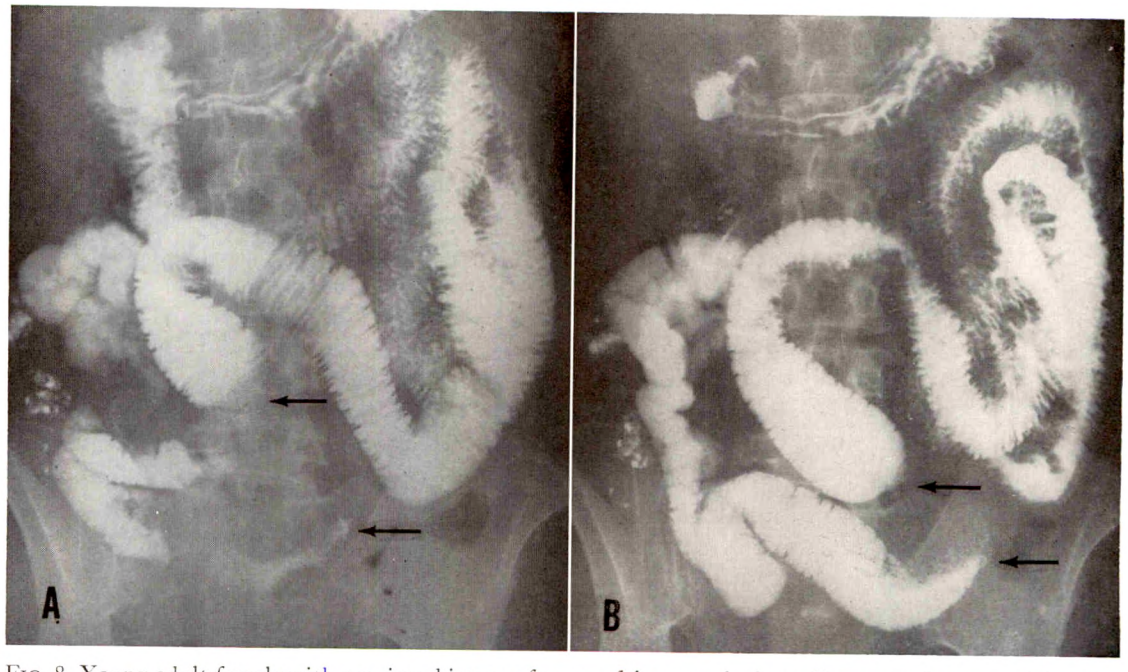



FIG. 8. Young adult female with previous history of successful removal of a malignant melanoma of the skin. She entered with crampy abdominal pain and vomiting. Plain roentgenograms were nondiagnostic, showing only a few tiny air-fluid levels in the right upper abdominal quadrant. One hour after barium sulfate was given orally there is a club-shaped loop of jejunum in the mid abdomen suggestive of a mechanical obstruction (upper arrow in *A*). Thirty minutes later the appearance is typical for a mechanical obstruction (upper arrow in *B*). Some barium passed distal to the obstruction. Lower arrows in *A* and *B* indicate the barium trickling into the small intestine distal to the obstructing lesion located between the upper and lower arrows. At surgery a metastatic melanoma was found.

intestine in the absence of a demonstrable mechanical point of obstruction. The analysis of this material will be the subject of future reports.

## TOLERANCE OF THE BARIUM SULFATE SUSPENSION

As learned in previous studies in animals,[12,13] and judging by our clinical experience to date, patients tolerate the barium sulfate suspension much better than they do the iodinated medium. There is less vomiting and less cramping following the administration of the barium sulfate than following the administration of the iodinated media. There has been no inspissation of the barium suspension proximal to the mechanical obstruction in any of our cases, the retained barium suspension being easily aspirated by enterotomy at the time of the operative correction of the obstruction. This confirmed our animal studies and those of Donato *et al.*[5]

## SUMMARY

We are now using a good commercial barium sulfate suspension with increasing frequency in patients suspected of having mechanical obstruction of the small intestine. The use of this heretofore feared and neglected method of studying this group of patients in whom the clinical findings and plain roentgenograms of the abdomen are nondiagnostic will do much to preclude undue procrastination in patients who have obstructions of the small intestine, and will also aid in clarifying the abdominal symptoms in those found to be free of obstruction.

This method is entirely safe, often results in an early objective diagnosis of a mechanical obstruction, and hopefully will result in earlier operative treatment of many cases of mechanical obstruction of the small intestine. It is likely that earlier operative treatment will further lower the mortality and morbidity in that group of patients who are difficult diagnostic problems.

Much remains to be learned, and it is hoped that the use of this method will continue to add to our knowledge of the roentgenologic manifestations of strangulating obstruction and paralytic ileus.

TABLE I

DIAGNOSTIC POSSIBILITIES FOLLOWING THE ADMINISTRATION OF $BaSO_4$ SUSPENSIONS ORALLY TO PATIENTS SUSPECTED OF HAVING MECHANICAL OBSTRUCTION OF THE SMALL INTESTINE

I. Normal Transit Time from Stomach to Cecum (approximately ½–8 hours)
  A. Normal morphologic study
  B. Nonobstructing lesion identified
  *C. Partial or temporary mechanical obstruction—point identified (Fig. 2, *A–C*; 4, *A* and *B*; and 5, *A* and *B*)
  D. Partial or temporary mechanical obstruction—point not identified
II. Slow Transit Time from Stomach to Cecum (more than 8 hours)
  A. Normal morphologic study
  B. Nonobstructing lesion identified
  *C. Partial mechanical obstruction—point identified (Fig. 7, *A* and *B*; and 8, *A* and *B*)
  D. Partial mechanical obstruction—point not identified
  E. Neuromuscular motility problems—normal morphology
    1. Scleroderma
    2. Aganglionosis
    3. Mild to moderate paralytic ileus
III. Complete Arrest of $BaSO_4$ in Small Intestine
  A. Mechanical obstruction—point identified
    *1. Simple mechanical (Fig. 3, *A* and *B*; and 6, *A* and *B*) (usually less than 3 days in duration)
    2. Early strangulated (usually less than 10 hours in duration)
  B. Mechanical obstruction—point not identified
    1. Simple mechanical (usually more than 3 days in duration)
    2. Strangulating (usually more than 10–12 hours in duration)
  C. Widespread paralytic ileus
    1. Nonmechanical strangulating obstruction (vascular)
    2. Postoperative ileus
    3. Postperforation

* Type of mechanical obstructions of the small intestine demonstrated by the method advocated in this communication.

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VOL. 101, NO. 2

# INTESTINAL EDEMA*

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DROPSY, or peripheral edema, is a clinical state whose earliest descriptions date to antiquity. With the exceptions of ascites and pulmonary edema, however, its central or internal accompaniments have been sufficiently subtle to escape ready recognition. Obviously, identifiable clinical consequences may follow the development of visceral edema but these have a nonspecific character and are often absent entirely. Only recently have sufficiently characteristic roentgen changes been described to permit establishment of objective diagnostic criteria for confirming the presence of edema of the bowel.[4,5,8,10]

By far the commonest cause of noninflammatory intestinal edema is hypoproteinemia, specifically hypoalbuminemia, resulting from excessive loss of protein from the intestinal tract, cirrhosis or nephrosis. The pathogenesis of the edema of the bowel seen in cirrhosis and nephrosis with hypoproteinemia is readily understandable, yet the roentgen findings of intestinal edema have not always been present. This discrepancy seems to be accounted for by the level of the serum albumin. When the serum albumin level is 2 grams per cent or lower, intestinal edema can be demonstrated roentgenologically. Protein loss from the gastrointestinal tract in normal situations is small. However, when the loss is excessive, hypoproteinemia occurs. Protein-losing enteropathy is not a single disease entity and has been demonstrated in association with a variety of gastrointestinal disorders,[6,9,11,12,17] as well as in apparently unrelated conditions such as congestive heart failure, constrictive pericarditis, exudative skin conditions, burns and allergy.[1,7,18] With recently developed techniques to detect protein loss via the gastrointestinal tract,[2,13,14,15] an increasing number of specific protein-losing enteropathies and generalized diseases associated with gastrointestinal protein loss are being discovered.[16] While these patients show intestinal mucosal edema, they may have no gastrointestinal symptoms. The symptoms then are those of the primary disorder or of peripheral edema resulting from the hypoproteinemia.

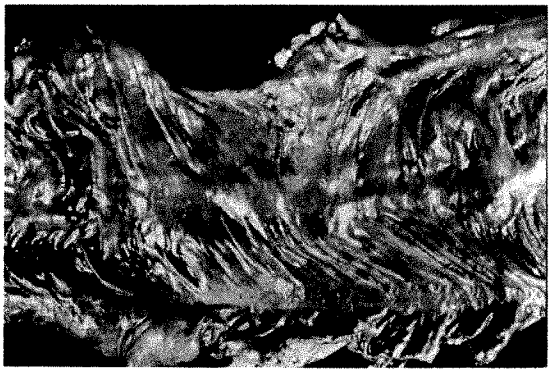


FIG. 1. Cirrhosis of the liver with hypoproteinemia; colonic segment showing thick, boggy, edematous folds.

Intestinal lymphangiectasia is a distinctive cause of protein loss from the gastrointestinal tract. Clinically, the condition manifests peripheral edema, serous cavity effusions and diarrhea. Laboratory studies demonstrate hypoproteinemia with excessive protein loss into the gastrointestinal tract.

Pathologically, hypoalbuminemia causes an accumulation of cell free, noninflammatory fluid within the wall of the intestinal tract producing uniform thickening of the mucosal folds (Fig. 1, 2 and 3). The bowel lumen may be slightly dilated. Secondary to the fluid accumulation, there are anatomic and motor alterations in the intestine which are reflected in the roentgenogram. In intestinal lymphangiectasia, the small bowel appears edematous and its

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FIG. 2. Cirrhosis of the liver with hypoproteinemia; edema of the small intestine.

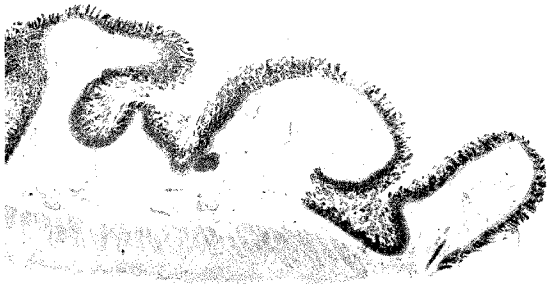


FIG. 3. Section of the small intestine showing marked cell-free accumulation of fluid in the submucosa in a patient with cirrhosis of the liver.

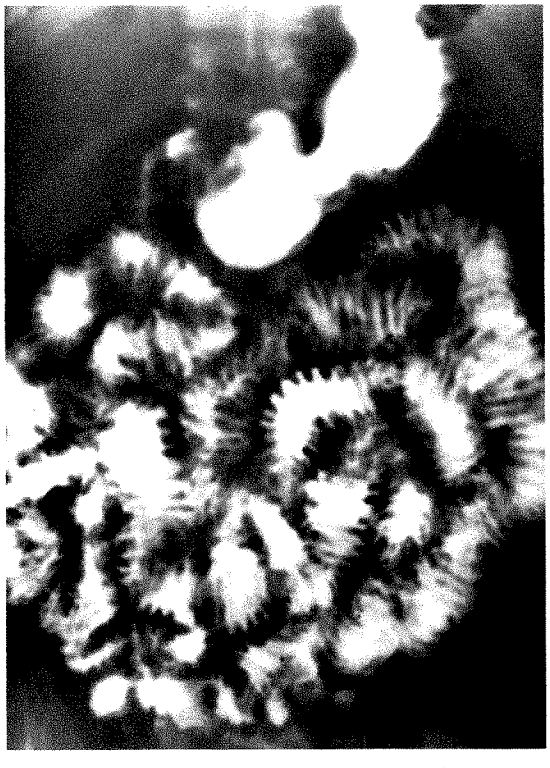


FIG. 4. Cirrhosis of the liver. Serum albumin 2 grams per cent. Minimal dilatation of the lumen with symmetric thickened folds suggesting a stacked coin configuration is seen. Contractions are present but incomplete. The loops are separated and uncoiled due to bowel wall and intervening soft tissue edema.

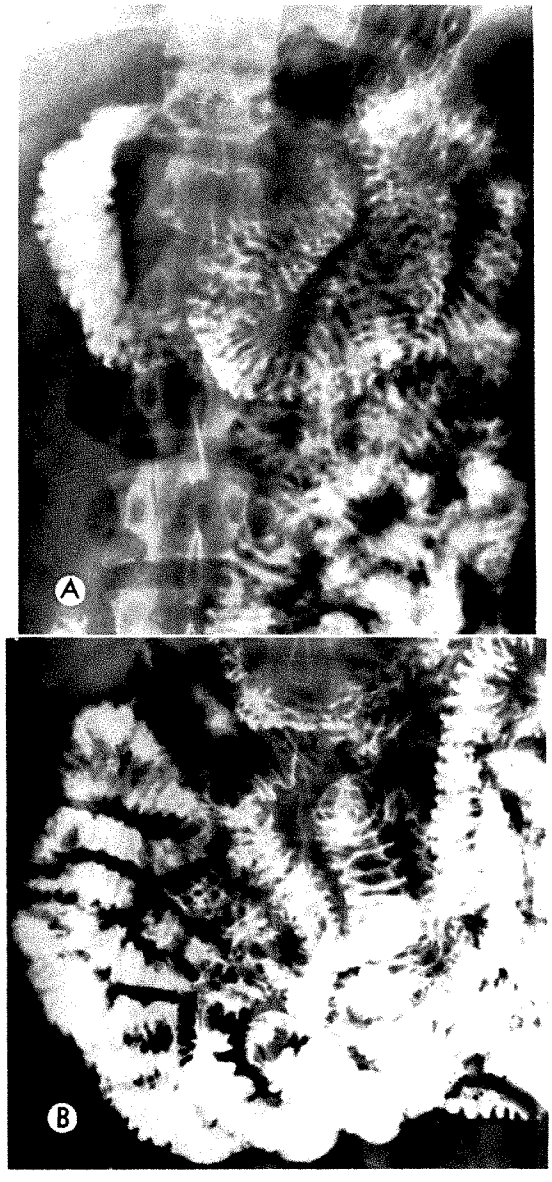



FIG. 5. (*A*) Cirrhosis of the liver. Serum albumin 1.8 grams per cent. There is symmetric thickening of the folds associated with slight dilatation. The changes are universal. (*B*) Two months later. The changes persist but to a lesser degree. Many of the individual folds show a biconvex appearance.

serosal surface pigmented. The hallmark lesion is demonstrated microscopically and consists of dilated mucosal and submucosal lymphatics. Within the mucosa are abundant foamy lipophages.

It is not clear how protein is lost into the gastrointestinal tract in protein-losing enteropathies. Waldmann,[13] who first described intestinal lymphangiectasia in 1961, has suggested that the dilated lymphatic vessels noted in this condition rupture and discharge their contents into the lumen of the gut. In some patients, with the onset of symptoms in infancy and childhood, the condition appears to be congenital. However, it has been described late in life, probably as an acquired abnormality, perhaps occasioned by lymphatic obstruction.

### ROENTGEN FINDINGS IN EDEMA

The changes are often present throughout the gastrointestinal tract but are best appreciated in the jejunum. The colonic alterations have been confined to the right colon.

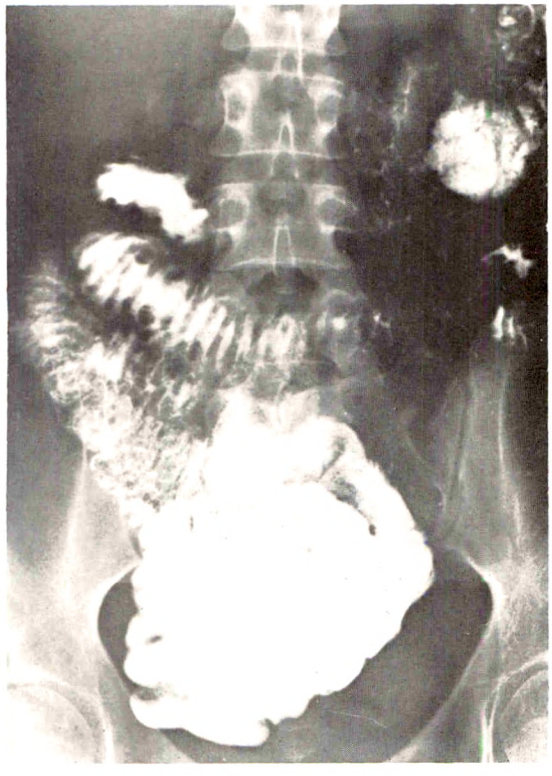


Fig. 6. Ulcerative colitis with hypoproteinemia. Serum albumin 1.7 grams per cent. The typical thickening of the folds of hypoproteinemia are seen.

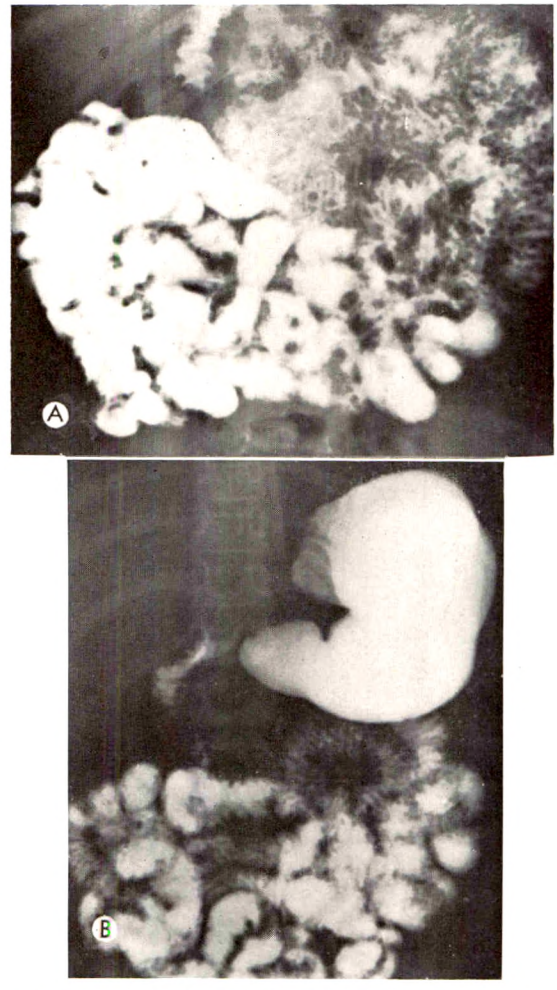



Fig. 7. (*A*) Nephrosis. The folds are thickened and hazy. Abnormal motor activity is demonstrated. (*B*) Two months previously the small bowel was normal.

The entire small intestine is usually slightly dilated. The loops of bowel are slightly separated and uncoiled due to edema of the bowel wall and intervening soft tissues (Fig. 4). The mucosal folds are uniformly and symmetrically thickened (Fig. 4; 5, *A* and *B*; 6; 7, *A* and *B*; and 8, *A* and *B*). Occasionally the thickening may be so pronounced that the appearance simulates a stack of coins as has been described in intramural hemorrhage.[3] The stacked coin appearance is a feature of

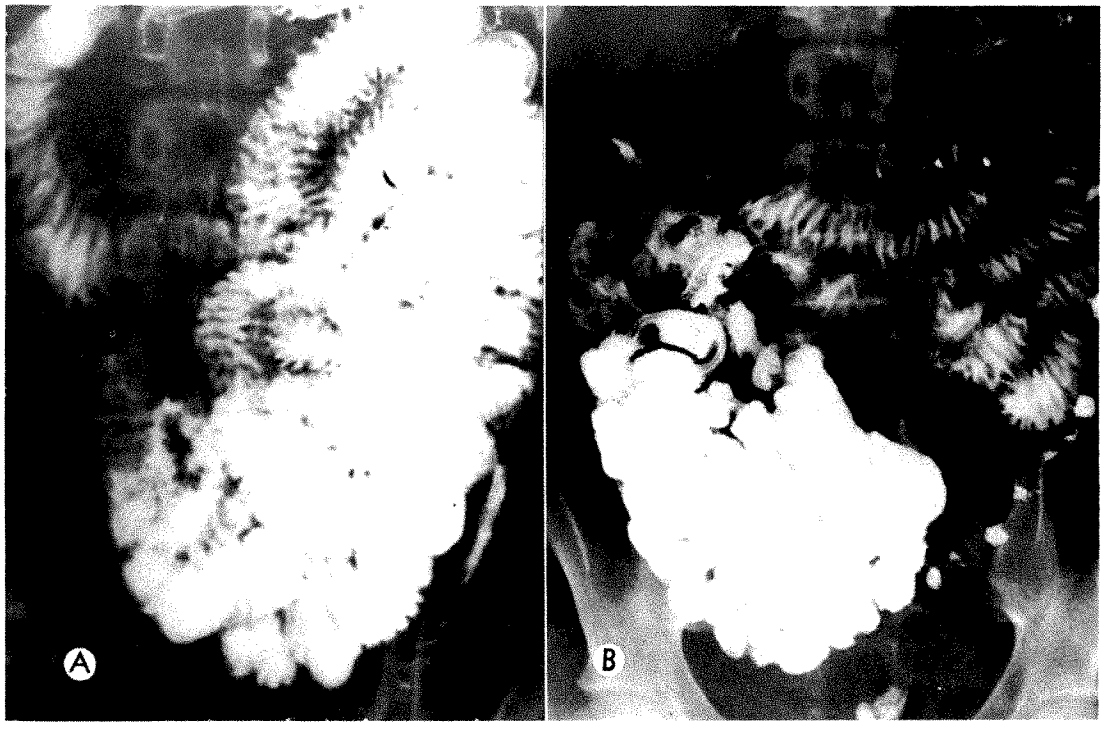



FIG. 8. (*A*) Nephrosis. The serum albumin is 1 gram per cent. Intestinal edema. (*B*) Two hours later; the changes appear to be limited to the jejunum. The ileum is normal.

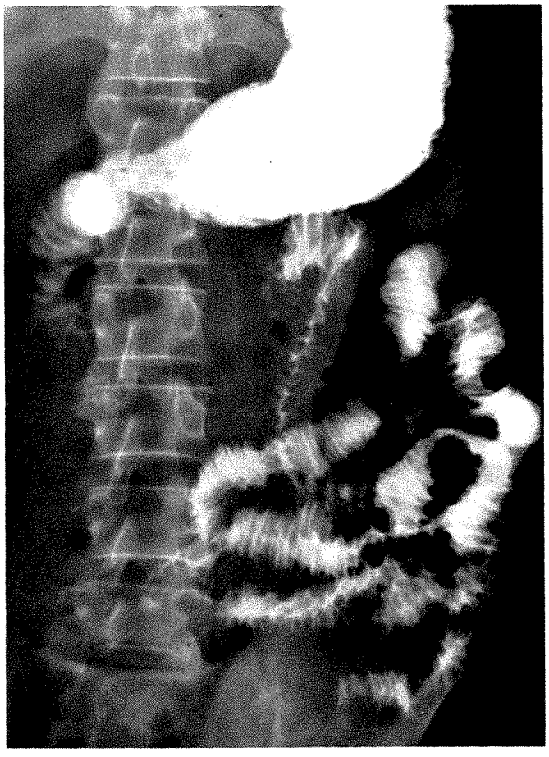


FIG. 9. Nephrosis. Irregular, incomplete contractions are prominent in the proximal small intestine. The appearance is transient. Thickened, regular, edematous folds are seen throughout. The stomach shows changes of edema.

several entities and is presumably the result of intramural fluid accumulation. The intramural fluid alters the motor activity of the intestine and contractions are irregular and incomplete, producing a bizarre pattern which can mimic inflammatory disease or lymphoma (Fig. 9; 10, *A* and *B*; and 11). However, there is no evidence of mucosal ulcerations or nodules. The barium column is continuous with neither segmentation nor flocculation. In many of these cases, ascites is present. Ascites itself due to causes other than cirrhosis does not produce thickening of the mucosal folds. However, in liver cirrhosis with ascites and without marked hypoproteinemia, there may be some old thickening. In this instance, portal hypertension with resultant venous stasis may be responsible for the roentgen change (Fig. 12).

In our cases, involvement only of the right colon has been seen. The reason for this localization is obscure. There is some limitation of distensibility of the bowel wall and evacuation is usually incomplete because of the thickened walls and altered

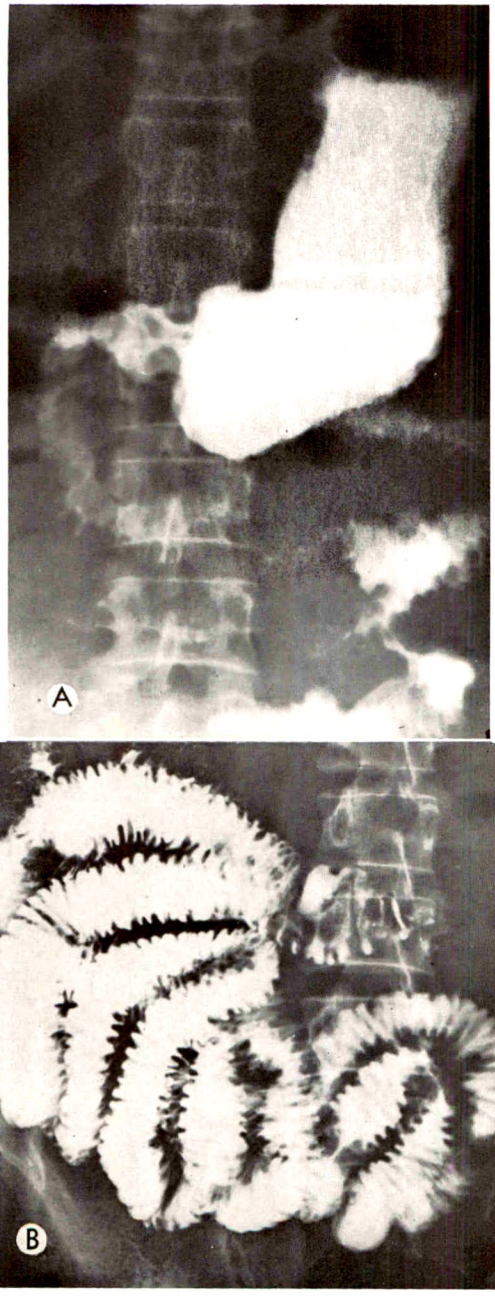



FIG. 10. (*A*) Nephrosis. Serum albumin 1 gram per cent. The stomach is slightly dilated and atonic. The folds along the greater curvature are flattened, irregular and indistinct. These changes are due to edema of the gastric wall. Abnormal motor activity and thickened mucosal folds are seen in the jejunum. (*B*) Later study. Typical changes of intestinal edema are present.

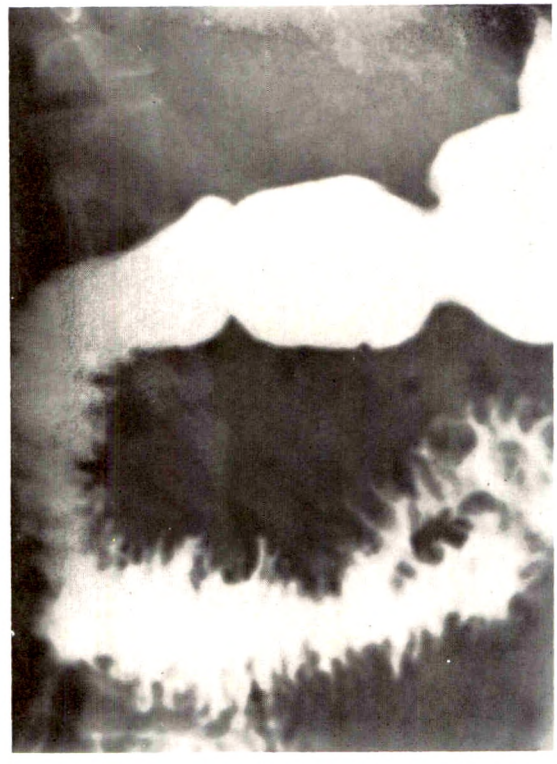


FIG. 11. Cirrhosis of the liver. Serum albumin 2 grams per cent. The duodenal loop is dilated and the folds are thickened. The irregularity of the folds and nodular defects along the contour were transient and due to abnormal motor activity.

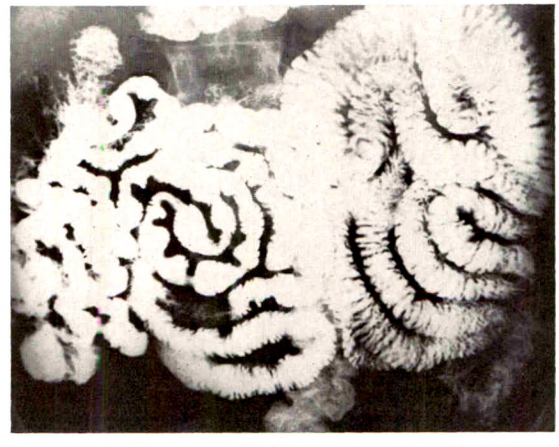


FIG. 12. Cirrhosis of the liver associated with ascites. The serum albumin is not significantly lowered. There is central location of the intestinal loops due to ascites. Slight thickening of the jejunal folds is presumably due to portal hypertension.

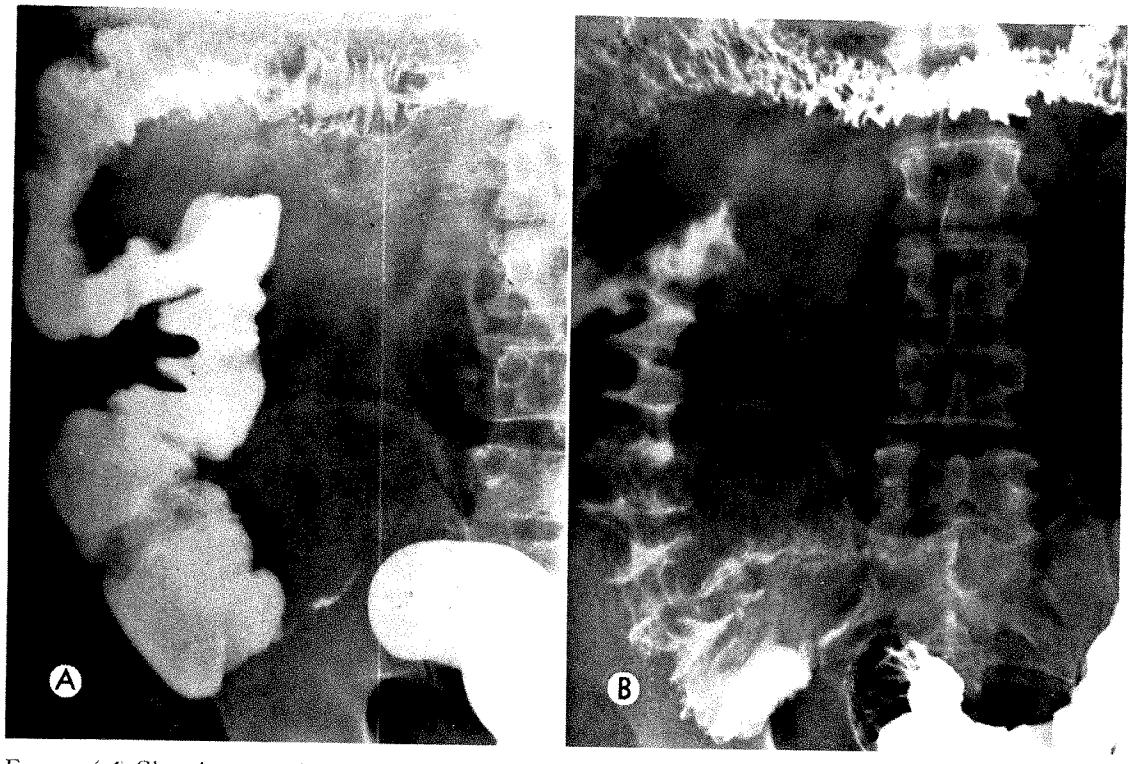



FIG. 13. (*A*) Chronic congestive heart failure. Hypoalbuminemia. The right side of the colon has an irregular caliber and haustrations. Despite filling, the folds are still visible and appear thickened. (*B*) Following evacuation, the arrangement of the boggy, thickened folds is maintained. This is due to incomplete contractility secondary to intramural edema. The normal longitudinal shortening is absent and the right side of the colon retains the same anatomic configuration as seen in *A*. Repeat examination 3 years later showed no abnormality.

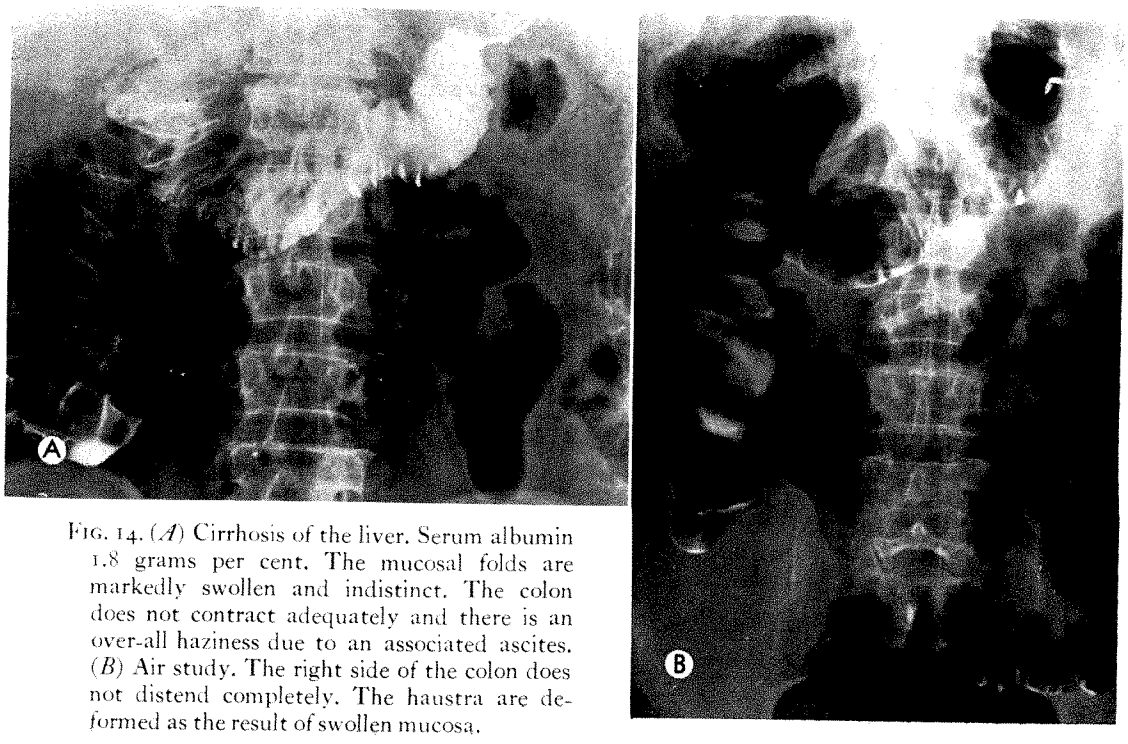



FIG. 14. (*A*) Cirrhosis of the liver. Serum albumin 1.8 grams per cent. The mucosal folds are markedly swollen and indistinct. The colon does not contract adequately and there is an over-all haziness due to an associated ascites. (*B*) Air study. The right side of the colon does not distend completely. The haustra are deformed as the result of swollen mucosa.

motor function. A conspicuous feature is absence of normal longitudinal shortening following evacuation (Fig. 13, *A* and *B*). The anatomic configuration of the colon remains the same on both the barium-filled and postevacuation roentgenograms. Multiple irregular contractions are seen which may be persistent. If the edema is marked, the haustra may be deformed and irregular (Fig. 14, *A* and *B*; 15; and 16). The mucosal folds appear thickened, somewhat indistinct and distorted. There is no ulceration or nodulation.

In intestinal lymphangiectasia (Fig. 17, *A* and *B*; and 18, *A* and *B*), the roentgen



FIG. 15. Cirrhosis of the liver with ascites. Hypoalbuminemia. A striking demonstration of multiple nodular defects in the transverse colon as a result of edema of the bowel. The colon is visualized following barium meal examination. Two months earlier, the colon was normal.

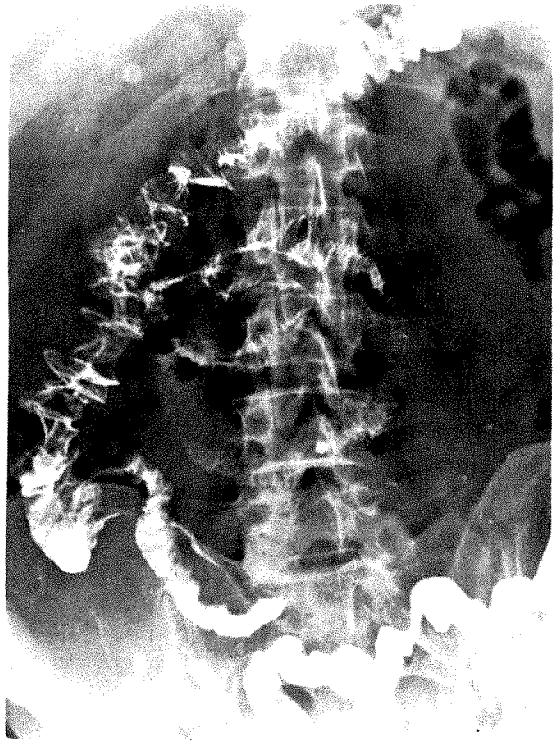


FIG. 16. Budd-Chiari syndrome. The serum albumin is 2 grams per cent. Thickening of the mucosa involves the distal ileum and the right side of the colon. Ascites and a calcified gallstone are present.

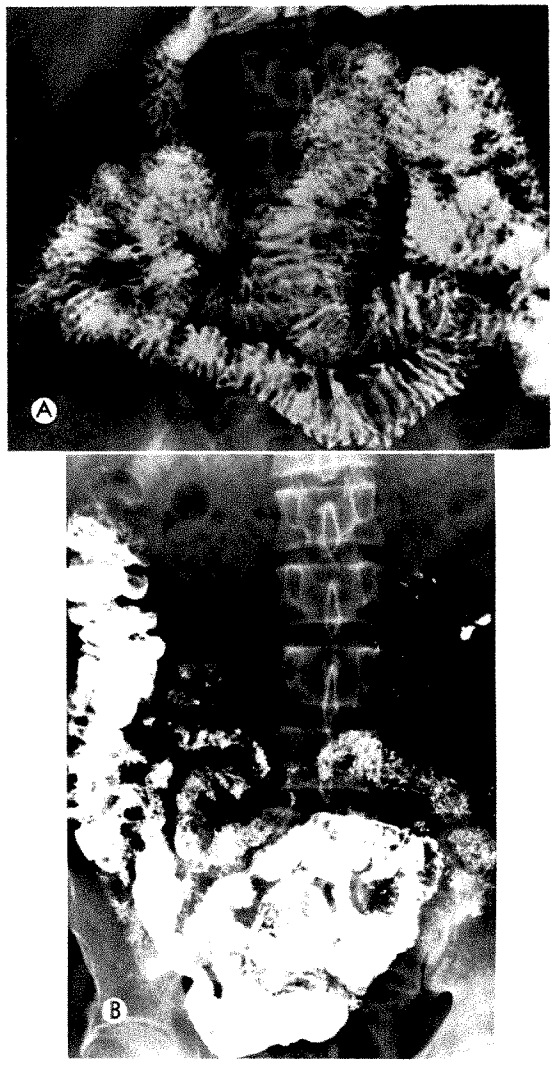



FIG. 17. (*A*) Intestinal lymphangiectasia. The serum albumin is 1.5 grams per cent. The jejunum is dilated, the folds are thickened and there are increased secretions. (*B*) The barium has a granular appearance due to excessive secretions. There is no segmentation.

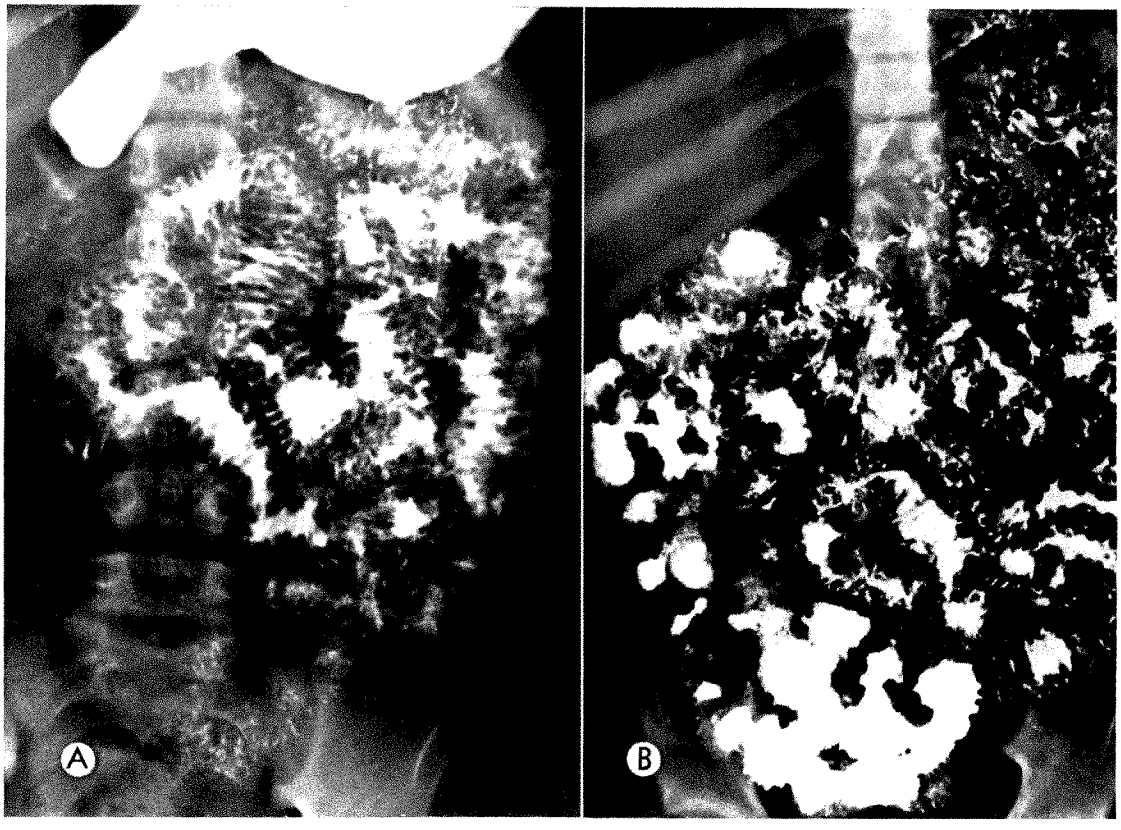



FIG. 18. (*A* and *B*) Intestinal lymphangiectasia. The serum albumin is 1 gram per cent. There is diffuse thickening of the valvulae conniventes associated with excessive secretions and slight dilatation. Blunting of the folds, fragmentation of the barium column and minimal segmentation are noted.

findings are similar to other hypoalbuminemic states except for the presence of excessive intestinal secretions which may alter the small intestinal pattern considerably. The folds are thickened and, depending on the amount of secretions, they may appear blunted, incomplete, and irregular. The barium column is fragmented and segmentation is seen. Occasionally the appearance resembles sprue. In sprue, however, the folds are usually thin and dilatation is a significant feature. Whipple's disease may also mimic intestinal lymphangiectasia on occasion. The folds in Whipple's disease are thickened, irregular and redundant. Since hypoproteinemia is frequent in many of the malabsorptive disorders it is possible that the same roentgen findings seen in these conditions may be a reflection of low serum proteins.

SUMMARY

The commonest cause of noninflammatory edema is hypoalbuminemia resulting from nephrosis, cirrhosis and intestinal lymphangiectasia. When the serum albumin level is 2 grams per cent or less, intestinal edema can be demonstrated roentgenologically. The roentgen findings in these conditions are similar and characteristic. The small intestine is slightly dilated, separated and uncoiled due to edema of the bowel wall and the intervening soft tissues. The mucosal folds are uniformly and symmetrically thickened. The intramural fluid alters the motor activity and the contractions are irregular. The right side of the colon may also be involved with similar findings. Ascites is frequent. The findings are most marked in intestinal lymphangiec-

tasia, where slight fragmentation and segmentation may also be seen.

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# THE ROENTGENOGRAPHIC EVALUATION OF ASCITES*

*By* EUGENE J. KEEFFE, M.D., RAYMOND A. GAGLIARDI, M.D., *and* RICHARD C. PFISTER, M.D.†
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FOR many years, radiologists have been taught and then taught others that the roentgenographic diagnosis of ascites was made on the basis of the following signs: (1) Separation and floating of bowel loops; (2) fuzziness or obliteration of the psoas muscle shadows; (3) over-all abdominal haziness; (4) increased density shifting to the pelvis on upright roentgenograms; and (5) poor definition of major abdominal organs.[5]

Two roentgenographic findings, one old and one new, should be added. The older sign has been referred to by Frimann-Dahl[1] under the eponym of Hellmer's sign. This is described as a relatively radiolucent shadow between the lateral wall of the liver and the abdominal wall. The radiolucency is said to be on the basis of differential density between the liver parenchyma and the fluid within the peritoneal cavity. We have reviewed all of our material with proved ascites of varying degrees and have been unable to corroborate this sign. For this reason, it has not been included in our present evaluation. More recently Margulies and Stoane[3] have described a sign in which the lower and lateral angle of the liver serves as a reference point for the diagnosis of ascites. This *hepatic angle* has been demonstrated by them to be obliterated in the presence of even quite small amounts of intra-abdominal fluid.

An important sign which we have used with success is what we refer to as the "McCort sign" based on a recent article of McCort[4] which dealt primarily with post-traumatic collections of intra-abdominal blood. His work was based largely on earlier fundamental observations of Frimann-Dahl and Laurell and has become a valuable aid in the diagnosis of intra-abdominal blood. This requires a knowledge of the roentgenographic anatomy of the flanks. The fact that ascitic fluid can displace the bowel in a predictable manner can be better appreciated if one is familiar with the anatomical landmarks in the flanks. The inner wall of the abdominal cavity is formed by the transverse abdominal muscles which are separated from the peritoneal lining by a layer of fat referred to generally as extraperitoneal or properitoneal fat. The wall of the colon can be distinguished because of the contrast provided by the presence of feces or gas within the colon. The filled colon should normally lie tight to the fat line. This very narrow division consists of the lateral wall of the colon and peritoneum. In our experience in a normal subject, this has been approximately 1–2 mm. in width and almost never more than 2 mm. (Case 1). If blood or ascitic fluid should collect in the flanks, there will be medial displacement of the colon. The very narrow *flank stripe* of increased density is then increased from its normal range of 1–2 mm. to a greater width depending on the amount of accumulated fluid.

Because of new interest in this area, we have evaluated the many roentgenographic signs to determine which of these were of real value. Recently, Hoffmann, Wankmuller and Rigler[2] have demonstrated the unreliability of the sign of separation and floating of bowel loops. They have actually called this "a fallacious sign of intra-peri-

* Presented as a Scientific Exhibit at the Sixty-eighth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., September, 26–29, 1967.
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Table I

PROVEN ASCITES

| Hepatic Angle Sign | Flank Stripe Sign | Combination |
|---|---|---|
| positive 78<br>indeterminate 22 | positive 91<br>indeterminate 9 | positive 97<br>indeterminate 3 |

toneal fluid." Fuzziness or obliteration of the psoas muscle shadow has been demonstrated by McCort[4] and by others to be a sign of retroperitoneal fluid and not intraperitoneal fluid. Sharply defined psoas muscle shadows are usually present even with quite large collections of intraperitoneal fluid. Other signs relating to over-all abdominal haziness, increased pelvic density or poor definition of major abdominal organs are known, without further documentation, to vary greatly with different degrees of obesity, minimal movement, bowel distention, or minor variations in technique. In effect, we are usually unable to make an unequivocal statement about ascites using the cardinal signs unless it is quite massive or is a confirmation of what is already clinically obvious. For this reason, we limited our survey of proven material to those cases in which evaluation was made of the *hepatic angle, the flank stripe* or *both.*

The clinician has a problem in the diagnosis of ascites similar to that of the radiologist. Although it is generally agreed that a good clinical examination will detect as little as 800 or 1,000 cc. of ascitic fluid, there is great difficulty in determining the presence of these small amounts. Wherever the ascites is not readily apparent in large quantities, extremes of disagreement are often found in different examiners.

## EXPERIMENTAL CONSIDERATIONS

Because our interest as regards this subject was in relatively small amounts of fluid, we tried to determine the area in which there would be sufficient fluid to provide a positive sign. This was done by the use of a fresh cadaver of a patient who had died of unrelated illness. Serial roentgenograms were made following the injection of 100 cc. increments of contrast material. Displacement of the bowel was minimal at about 800 cc. and by the time 1,000 cc. had been injected, the displacement of the bowel was readily apparent to all observers. It was our conclusion, therefore, that the sign of colon displacement could appear with as little as 800 cc. and become definite with 1,000 cc. (Fig. 1, *A–D*). Later clinical experience has confirmed this observation.

## RESULTS AND CLINICAL CONSIDERATIONS

We have reviewed 500 abdominal studies in adult patients; 100 of these were of cases with known ascites or ascites which was verified following roentgenographic examination. The other 400 were examinations of the abdomen or portions of spine examinations in patients with no suggestive symptoms or any clinical signs of abdominal fluid. From the figures in Table 1, it can be seen that the use of the flank stripe as a diagnostic sign was possible in 91 of 100 cases without introduction of contrast material and was possible in 78 of the 100 cases using only the hepatic angle. The use of the two signs together raised the accuracy to 97 per cent for all cases of ascites of any degree.

On the basis of these findings we have come to some conclusions regarding the relative value of these signs. We feel that the flank stripe and the hepatic angle together are the cardinal signs of abdominal ascites since they represent exact criteria. The sources of error in the McCort sign arise when the signs are not sufficiently defined for accurate diagnosis. If the colon is filled with fluid and fecal material, it is difficult to demonstrate the actual thickness

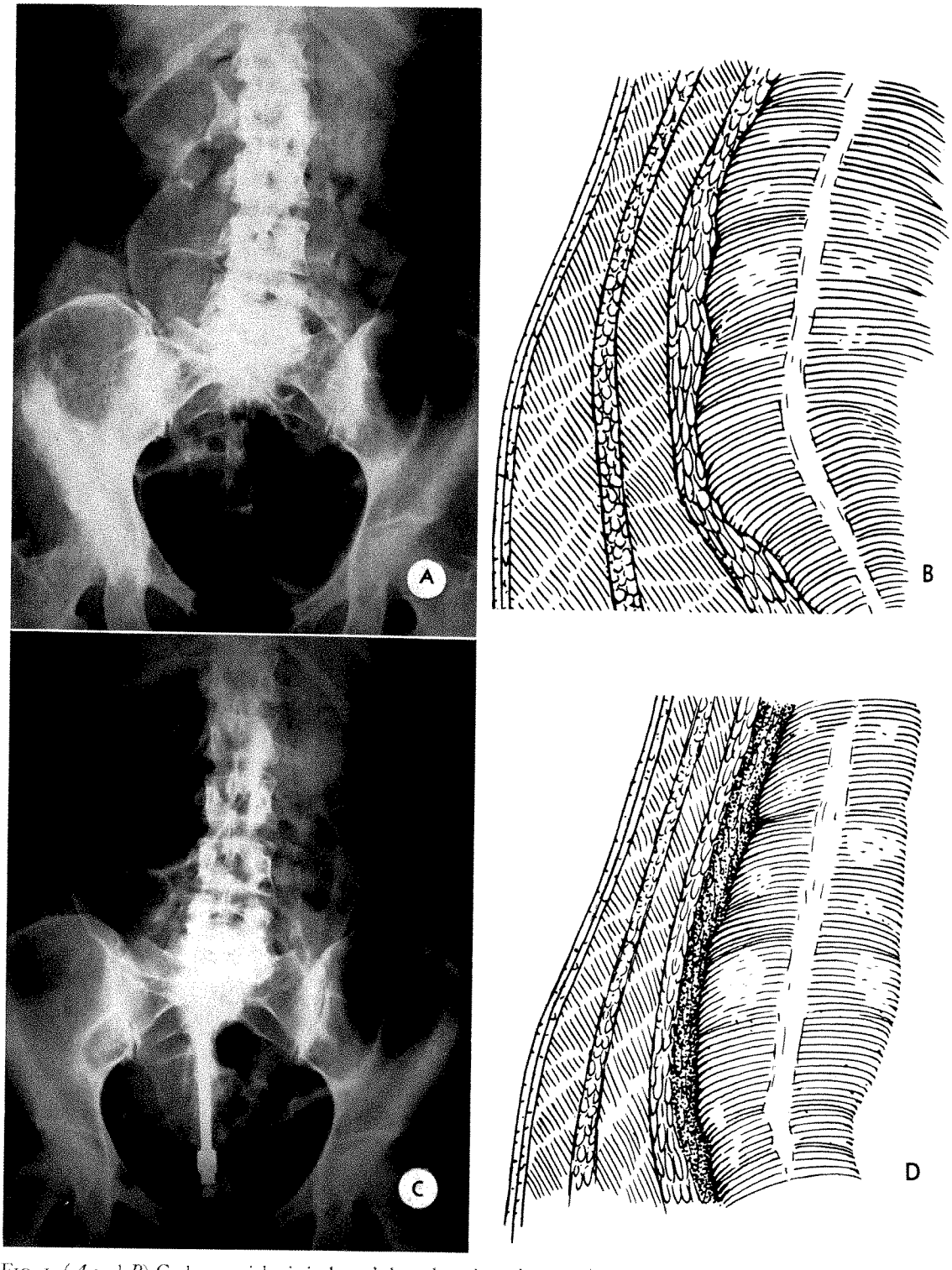



FIG. 1. (*A* and *B*) Cadaver with air in bowel, but there is no intraperitoneal fluid. (*C* and *D*) 1,000 cc. of contrast medium introduced via trochar. Note separation of bowel wall by fluid contrast material.

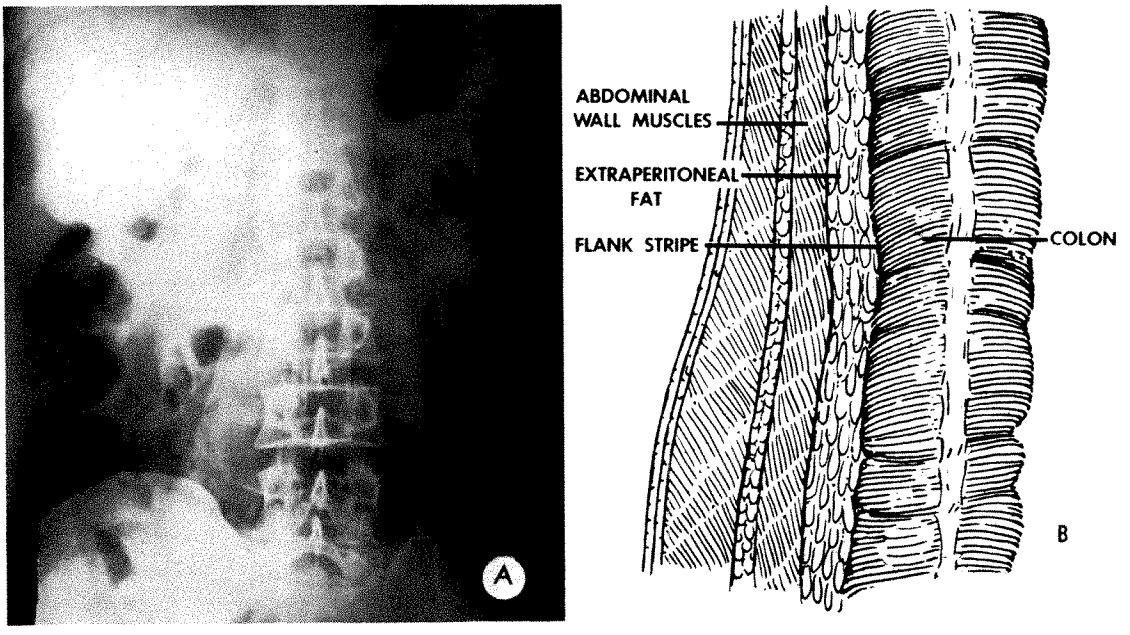



Fig. 2. Case 1. (*A* and *B*) Normal relationships.

of the bowel wall and the possibility of a false positive sign is present. On the left side the same rules of the flank stripe apply, but are somewhat less exact because fluid-filled small bowel may interpose between the descending colon and the abdominal wall giving the impression of a dense area between. We have found that awareness of this possibility in itself will usually aid in avoiding error, but both findings can be obviated by the introduction of a small amount of air or barium in the colon whenever the finding is in doubt. The obliteration of the hepatic angle is subject to the same pitfalls as the flank stripe sign. In reviewing the normal cases for determination of whether either sign could be used, it was found that the value of the hepatic angle was limited by fecal material in the ascending and transverse colon (Table II). Although there was no attempt to make a quantitative evaluation of the two signs, it was our general impression that the hepatic angle sign worked to advantage with larger amounts of fluid, but was less useful with small amounts of fluid, whereas the flank stripe sign was of greater value when small amounts of fluid were present. When neither sign can be used, we now try the introduction of air or barium to produce controlled distention of the colon and evaluate the flank stripe sign. It would appear obvious, however, that the optimum diagnostic conditions are present with use of both signs as corroborating findings.

## REPORT OF CASES

Case 1. (Fig. 2, *A* and *B*). R. J. was a normal healthy 44 year old male. The examination was a preliminary roentgenogram for cholecystography.

*Comment.* This is an example of the normal relationships one should expect to find showing the appearance of the fat and muscle layers and bowel wall in a normal subject. It should also be noted that the lateral inferior margin of the liver or hepatic angle is sharply defined.

Case II. (Fig. 3, *A–D*). J.M., a 31 year old female, was admitted with acute abdominal pain and a clinical diagnosis of possible rup-

Table II

CLEARLY DEFINED NORMAL SIGNS

| Hepatic Angle Sign | Flank Stripe Sign | Combination |
|---|---|---|
| 324 | 368 | 376 |

Total 400 cases.

tured ectopic pregnancy. The roentgenographic diagnosis of intraperitoneal blood was made and exploratory surgery confirmed the presence of a ruptured ectopic pregnancy with a massive amount of intraperitoneal blood.

*Comment.* Although the fluid is blood rather than ascitic fluid, it reveals the massive shift of the colon away from the radiolucent fat line with a dense stripe separating these two structures. This finding was of definite value because, as a coincidence, previous roentgenograms were available on this patient which demonstrated normal relationships. As a further point of interest, it should be noted that in spite of the massive amount of intraperitoneal bleeding, the psoas muscle shadows were

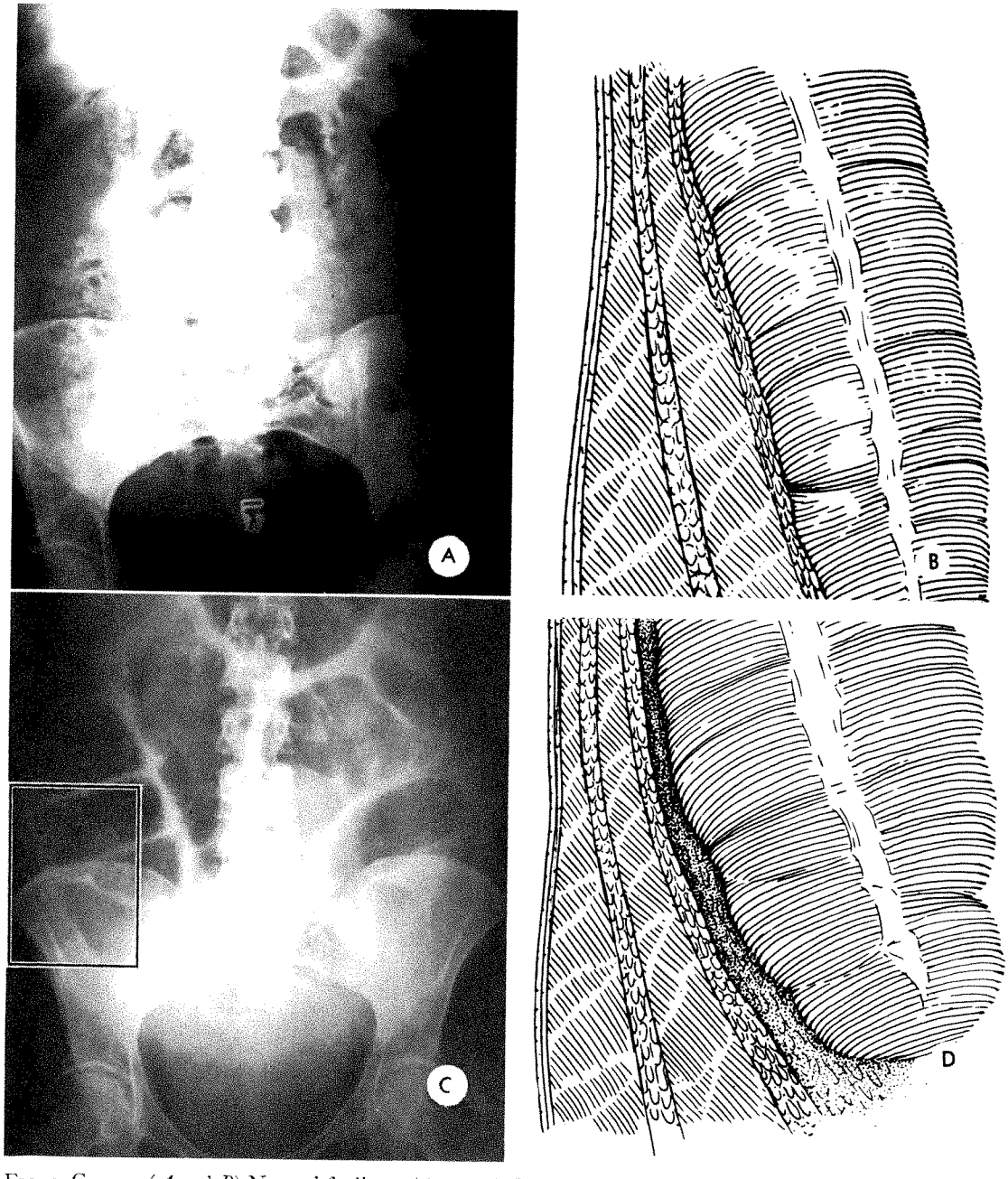



Fig. 3. Case 11. (*A* and *B*) Normal findings. (*C* and *D*) Change from normal to displacement with massive intraperitoneal bleeding. Psoas shadows remain well defined.

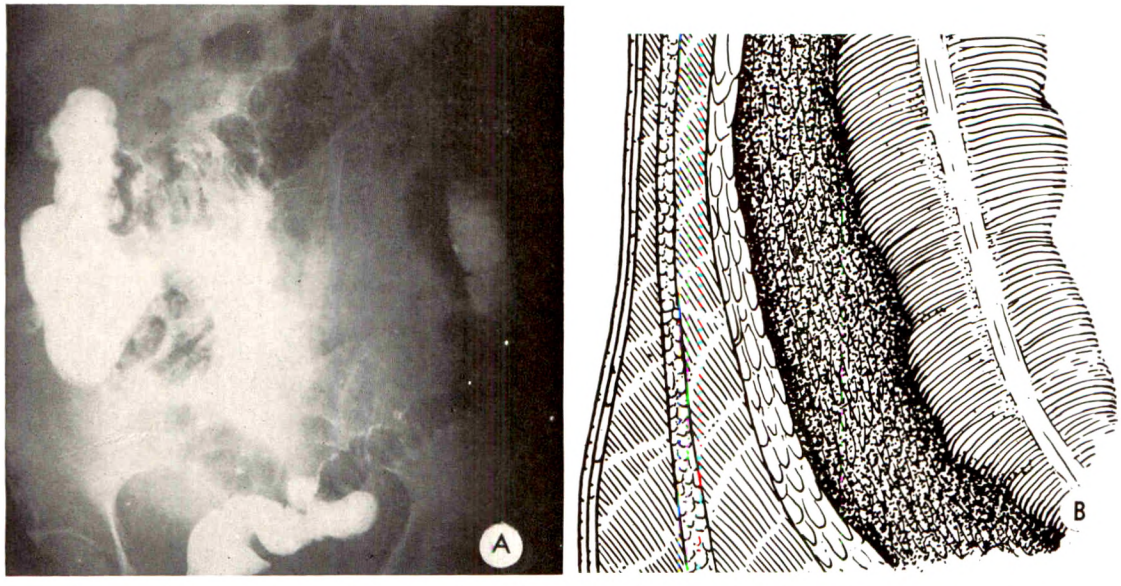



Fig. 4. Case III. (*A* and *B*) Positive hepatic angle and flank stripe signs with massive ascites.

sharply defined, since there was no retroperitoneal bleeding to produce obliteration of these structures.

Case III. (Fig. 4, *A* and *B*). M.J., a 73 year old white female, had a long history of recurrent heart failure. At the time of examination the patient had obvious clinical signs of congestive heart failure with enlargement of the liver and abdominal ascites as well as peripheral edema.

*Comment.* This is a case in which both the hepatic angle sign and the flank stripe sign are positive in the presence of massive ascites. This demonstrates that any intra-abdominal fluid can produce the changes seen with blood in the previous case.

Case IV. (Fig. 5, *A* and *B*). S.H., a 50 year old female, was a clinically and chemically confirmed case of cirrhosis of the liver with no definite evidence of abdominal ascites by clinical examination. Aspiration of the abdominal

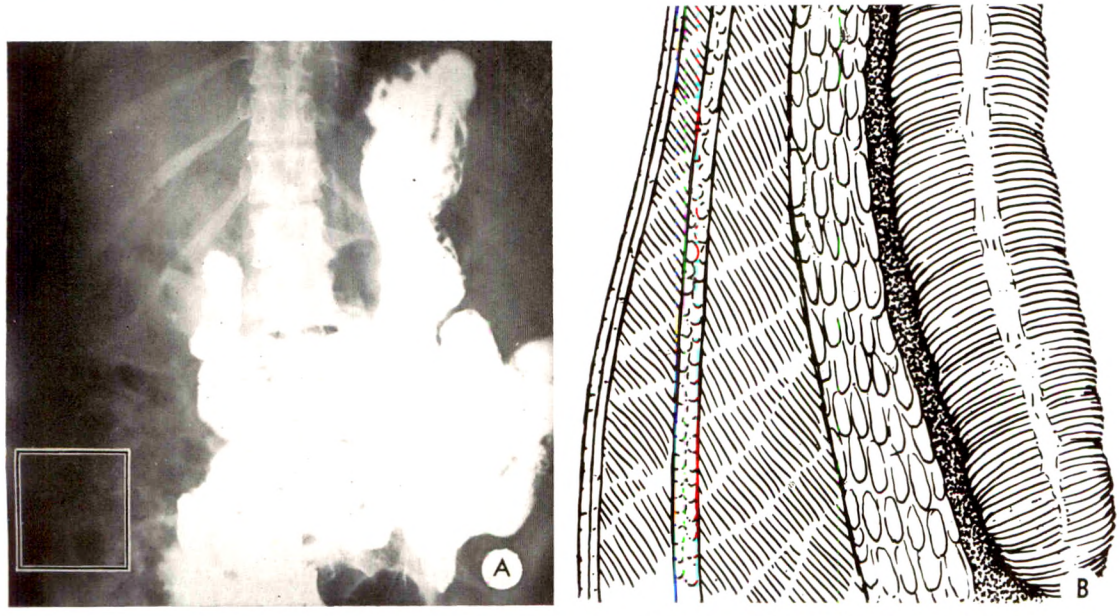



Fig. 5. Case IV. (*A* and *B*) Flank stripe is only 4 mm., but there is sufficient widening to indicate ascites.

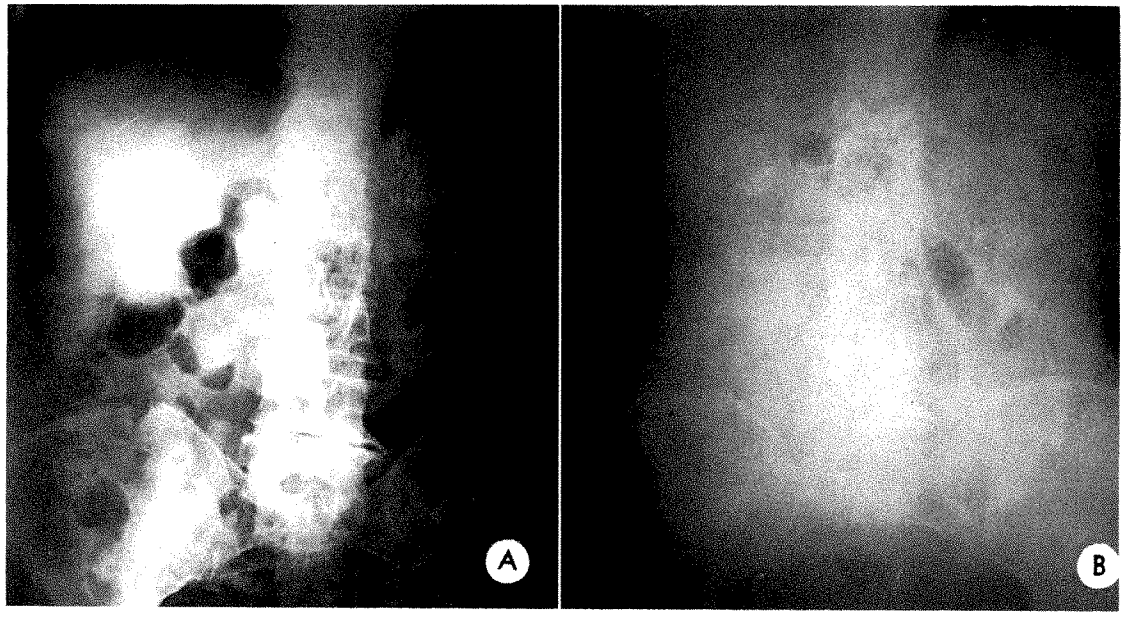



Fig. 6. Case v. (*A*) 1964: Normal finding. (*B*) 1966: Hepatic angle sign is of greater aid in massive (and clinically evident) ascites.

cavity demonstrated an excess of 1,000 cc. of fluid.

*Comment.* This case demonstrates the value of the positive flank stripe in the presence of a relatively small amount of intra-abdominal fluid. The conventional roentgenographic signs could not be elicited and clinical determination was uncertain, in spite of the known cirrhosis. The flank stripe was definitely positive, although the change was a small increase in the dense line of demarcation.

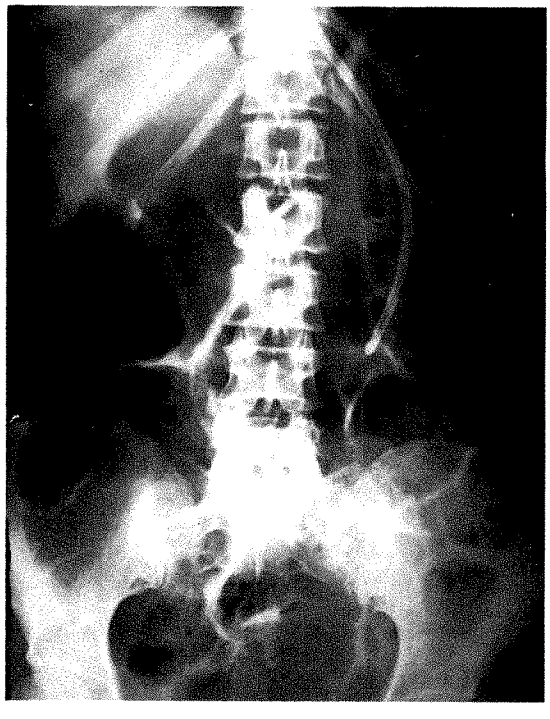


Fig. 7. Case vi. Normal flank stripe, but hepatic angle sign is of no aid.

Case v. (Fig. 6, *A* and *B*). A.B., a 76 year old woman, was first seen with abdominal discomfort. Barium enema examination demonstrated a carcinoma of the large bowel. At surgery the carcinoma was removed, but peritoneal seeding was already present. The patient was seen 1 year later with abdominal distention and with clinically obvious ascites.

*Comment.* Because of the distention and the large amount of ascitic fluid, the flank stripe sign was indefinite and of limited aid. This is a case in which the hepatic angle was of definite value since it showed the positive sign of intra-abdominal ascites. This case promoted the impression that the flank stripe sign is of greater value with small amounts of fluid, while the hepatic angle sign becomes of increasing value with larger amounts of fluid.

Case vi. (Fig. 7). J.D., a 40 year old woman, presented with symptoms suggestive of a urinary calculus. The examination was the preliminary roentgenogram for intravenous pyelo-



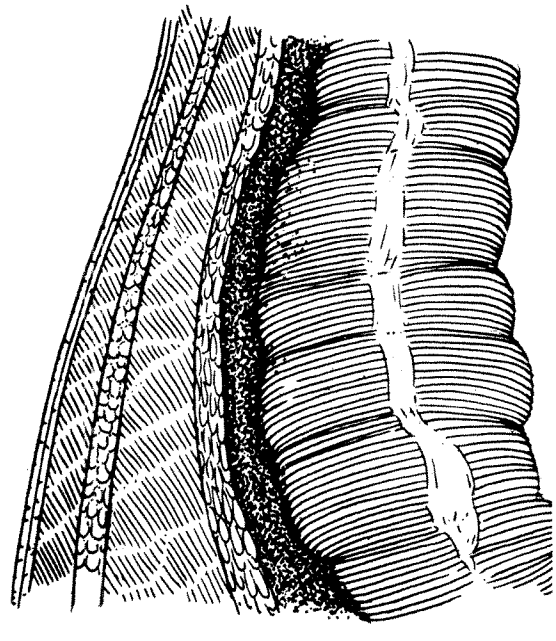




FIG. 8. Case VII. (*A*) Indefinite because of failure to identify bowel wall. (*B* and *C*) Widening of flank stripe becomes more definite when the bowel wall is determined with certainty.

graphy which was normal. The roentgenographic evaluation definitely excludes ascites.

*Comment.* This is a case in which the flank stripe sign was definitely normal, but the hepatic angle sign could not be utilized because of bowel distention.

CASE VII. (Fig. 8, *A–C*). F.K., a 76 year old woman, had a known ovarian carcinoma with peritoneal seeding and clinical findings suggesting the presence of ascites. Plain roentgenograms were indeterminate, but barium in the colon made the diagnosis of ascites definite. Autopsy confirmed the presence of fluid in excess of 1,000 cc.

*Comment.* This is an example in which there was poor contrast and in which the flank stripe sign could not be properly evaluated because of

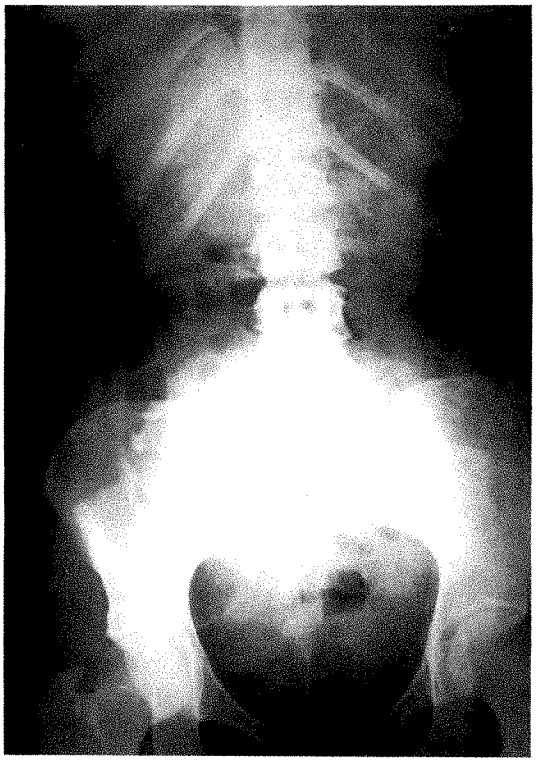


FIG. 9. Case VIII. This is a false positive. A diagnosis of intra-abdominal fluid was made, an error which should have been prevented by introduction of contrast material.

bowel distention and considerable fecal material in the colon. The use of barium was of definite value in delineating the relationship between the distended colon and the flank.

CASE VIII. (Fig. 9). R. H., a 25 year old woman, was admitted with severe abdominal pain and with a questionable pelvic inflammatory disease. The flank stripe sign was thought to be positive and a diagnosis of intra-abdominal fluid was made which, together with the clinical symptoms, suggested the presence of a ruptured ectopic pregnancy. Exploratory operation demonstrated pelvic inflammatory disease with no evidence of intra-abdominal fluid.

*Comment.* This case represents a false positive, diagnosed early in our utilization of this sign when we were not aware of all the pitfalls. The bowel was filled with some feces and a large amount of fluid material, but it was not distended with gas. Because of this, the fluid between the small amount of gas and the bowel wall was interpreted as additional widening of the flank stripe and was therefore in error. It should be pointed out incidentally that for this same reason the hepatic angle sign also would have pointed to the presence of ascites.

## SUMMARY AND CONCLUSIONS

The principles underlying the roentgenographic diagnosis of abdominal ascites are considered.

Evaluation of the hepatic angle sign and the flank stripe sign has been found to be the most useful and reproducible criterion for this diagnosis.

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# ROENTGENOLOGIC DIAGNOSIS OF RECTUS SHEATH HEMATOMA*

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THE anterior abdominal wall is of interest to the surgeon almost as frequently as any other region of the body. Rupture of the epigastric vessels within the rectus muscle is a definite surgical condition which is quite uncommon. The aim of this study is to establish the roentgenologic diagnostic signs to aid in the preoperative diagnosis.

A search of the world literature on the subject revealed sporadic reports on more than 500 patients. One of the first cases described in the United States was in the article of Richardson,[22] which appeared in 1857. An important report on this subject was that by Cullen and Brödel,[5] whose classic paper written in 1937 was an excellent clinical and anatomic study. Very few roentgenologic observations are available concerning this syndrome. Backwinkel[2] suggested preoperative cystography as a roentgenologic diagnostic aid. If the hematoma involves the lower portion of the rectus, it may cause extrinsic pressure on the bladder. This, however, is not specific, for similar deformity of the bladder may be caused by other pelvic space occupying lesions. Selective catheterization of the inferior epigastric artery is under investigation at this hospital.

Interest in the study of this syndrome was aroused in connection with a case seen in 1955. My preliminary observations were presented at the XIth International Congress of Radiology held in Rome in 1965.[9] Since then, 7 more cases have been observed and the roentgenologic features of this condition have been established.

## ETIOLOGY

The etiology of the rectus sheath hematoma may be (1) traumatic or (2) spontaneous.

The traumatic etiology includes blows or falls, violent muscular effort by heavy lifting, leaping of parachute jumpers, shock therapy, horseback riding, etc.

In the spontaneous group belong blood dyscrasias, anticoagulant therapy, infectious diseases, epigastric artery aneurysm, obesity, pregnancy, arteriosclerosis, chronic respiratory disease with severe cough, and hypertensive cardiovascular disease.

## CLINICAL FEATURES

The physical signs and symptoms are misleading and may suggest serious acute intra-abdominal pathology. The patient usually complains of sudden, severe abdominal pain and an extremely tender abdominal mass of variable size is present. In the majority of cases, no preoperative clinical or roentgenologic diagnosis is made. The anteroposterior supine or upright roentgenogram and lateral kidney, ureter and bladder roentgenograms are noncontributory.

Usually a variety of acute intra-abdominal conditons is suspected. If the mass is situated in the right lower abdomen, the case may be diagnosed as one of acute appendicitis, ovarian cyst with a twisted pedicle, ruptured tubal pregnancy, etc. When rupture has occurred low down in the left rectus sheath, the lesion has been mistaken for acute diverticulitis. When the rupture of the rectus sheath has occurred above the umbilicus on the right side, acute gallbladder disease has been diagnosed. The hemorrhage may be due to rupture of the muscle or to tearing of the deep epigastric artery or vein or to the simultaneous injury of both structures; it usually occurs below the umbilicus and is nearly always limited to one side.

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Wohlgemuth[24] observed that in about 53 per cent of his cases, the hemorrhage was on the right side, in about 33 per cent of the cases on the left side, and only 14 per cent above the umbilicus. Small hematomas may resolve without surgery. Chronic hematomas may show calcification and can be palpated as a hard tender mass.

## ANATOMY

Cullen and Brödel[6] found that the adult rectus muscles are 35 cm. long, 7 cm. wide and 0.8–1.0 cm. thick. The thickness of the muscles varies with the patient's age, sex and general condition. The muscles are divided by 3 or 4 strong transverse tendons into 4 or 5 independent muscular masses, each of which has its own motor nerve supply. They have, however, only one common vascular system fed from above and below. The muscles arise from the crest of the os pubis and from the ligaments covering the ventral surface of the symphysis pubis. The muscles are inserted into the cartilages of the fifth, sixth and seventh ribs. The rectus muscles are encased in a strong sheath formed through fusion of the flat tendons of the three broad abdominal muscles, the external oblique, the internal oblique and the transversus abdominis. The anterior leaf of the rectus sheath covers the entire ventral surface of the rectus. It is adherent to all of the transverse tendinous intersections and also to the lateral and medial margins of the muscle. The posterior leaf consists of the flat tendons of the transversus muscle and of the posterior half of the internal oblique tendon. The posterior leaf is not adherent to the rectus at any point. The lower tapering part of the rectus which is the strongest portion of the muscle is separated from the abdominal viscera by only the thin peritoneum. Consequently, if the hematoma lies between the muscle and the peritoneum, it produces severe peritoneal irritation.

The muscles are supplied by two arteries, the inferior and superior epigastric arteries. The deep epigastric artery arises from the external iliac artery. The superior epigastric artery is the direct continuation of the internal mammary artery. The deep inferior epigastric artery lies on the posterior aspect of the rectus muscle separated from the abdominal viscera only by the peritoneum and a thin fascia. The superior epigastric artery enters the upper medial portion of the muscle. There are no main arterial branches in the mid point of the muscles, for the two systems meet in an undifferentiated plexus of small vessels. The veins of the upper rectus drain upward into the subclavian and superior vena cava; in the lower 2/3 of the muscle the venous drainage is downward into the external iliac and inferior vena cava.

The healthy muscle can be stretched enormously without injury as demonstrated by acrobats and dancers. Pregnancy, ascites and excessive obesity can easily elongate the rectus to twice its length and almost twice its width. Frequently, hyaline or waxy degeneration of the muscle after typhoid fever, influenza or infectious diseases is a predisposing factor to rupture of the blood vessels with hematoma formation.

## ROENTGENOGRAPHY OF THE RECTUS MUSCLE

The rectus muscle is not visible in the anteroposterior roentgenogram of the abdomen or of the lower chest. Its upper segment and attachment to the thorax is almost always visible on the lateral roentgenogram (Fig. 1). Its mid portion and origin from the symphysis are recognized on the lateral roentgenogram of the abdomen or pelvis when made with soft tissue technique (Fig. 2 and 3). The superimposed rectus muscles are forming the rectus shadow. The rectus shadow which is the dividing landmark between the anterior abdominal wall and the intra-abdominal structures is better visualized in obese patients. Localized widening of the rectus shadow or calcification in it, indicates acute or chronic hematoma. The hematoma is usually confined to the rectus sheath, which

I was able to confirm by injecting contrast material into the rectus sheath of a cadaver (Fig. 4 and 5).

The examination is performed with the patient lying on the left or right side on the table with the roentgen-ray beam centered on the anterior abdominal wall. If the patient is unable to lie on his side, a dorsal decubitus roentgenogram can be taken, the so-called supine transabdominal view. Soft tissue or low penetration technique should be used.

## REPORT OF CASES

Case 1. A 41 year old obese white female was admitted to the Cambridge City Hospital with right lower quadrant pain. Her present illness began the same morning, soon after she had slipped and fallen on the ice and landed on her buttocks. Immediately after falling, she noticed severe pain in the right lower quadrant. The pain gradually increased and became excruciating. It interfered with her raising herself up or moving around or lifting her right leg. After a few hours, she became nauseated and vomited several times. On palpation, a large football sized, exquisitely tender, movable mass was present in the right lower quadrant. There was spasm and rebound tenderness around the

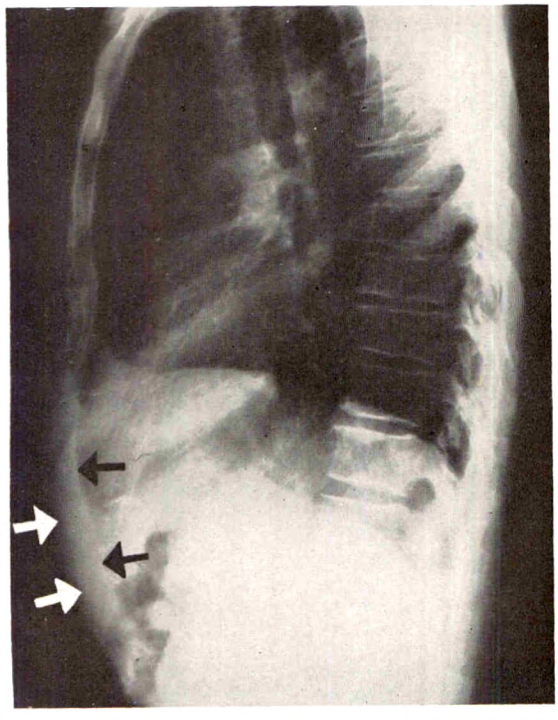


Fig. 1. Lateral roentgenogram of a severe asthmatic patient showing hypertrophy of the rectus muscle due to forced abdominal breathing.

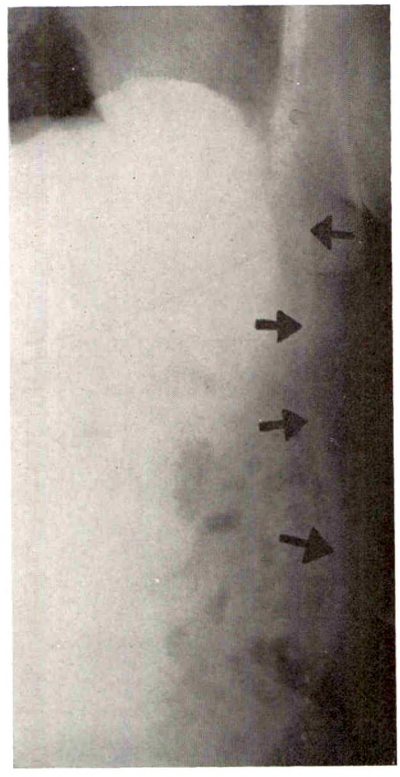


Fig. 2. Obese patient with good visualization of nearly the entire rectus muscle.

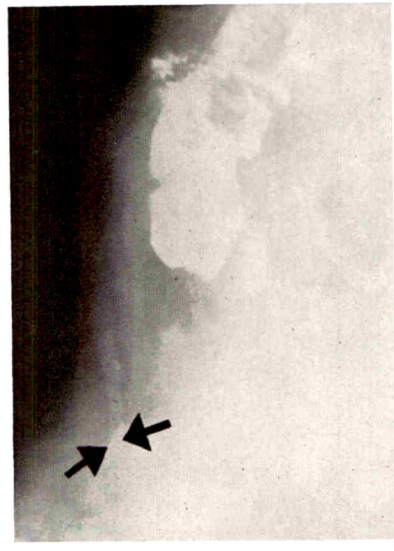


Fig. 3. Origin of the lower rectus from the pubic bones.



Fig. 4. Injection of 30 cc. of hypaque into the anterior rectus sheath of a cadaver.

mass. The anteroposterior roentgenogram of the abdomen was noncontributory. The lateral roentgenogram revealed a placenta like, spindle shaped mass in the right lower anterior quadrant and gas filled small bowel loops in its vicinity indicating possible intestinal strangulation (Fig. 6). At surgery a long rectus muscle retracting incision was employed. When the rectus sheath was reached, there was noted to be a bluish discoloration and bulging of the sheath. This was opened and a large hematoma was evacuated from beneath the right rectus muscle. Further exploration revealed a ruptured and bleeding inferior epigastric artery. Because of the roentgenologic report of dilated small bowel loops, the peritoneum was opened but no intra-abdominal pathology was found. The cause of the gas filled small bowel was probably a mild secondary paralytic ileus. Her recovery was uneventful.

Case II. A 58 year old white female with arteriosclerotic heart disease and congestive heart failure was admitted to Cambridge City Hospital with right lower quadrant abdominal pain. Physical examination revealed a 10 cm. rounded tender right lower quadrant mass. Conventional anteroposterior and upright roentgenograms of the abdomen were noncontributory. The lateral roentgenogram of the

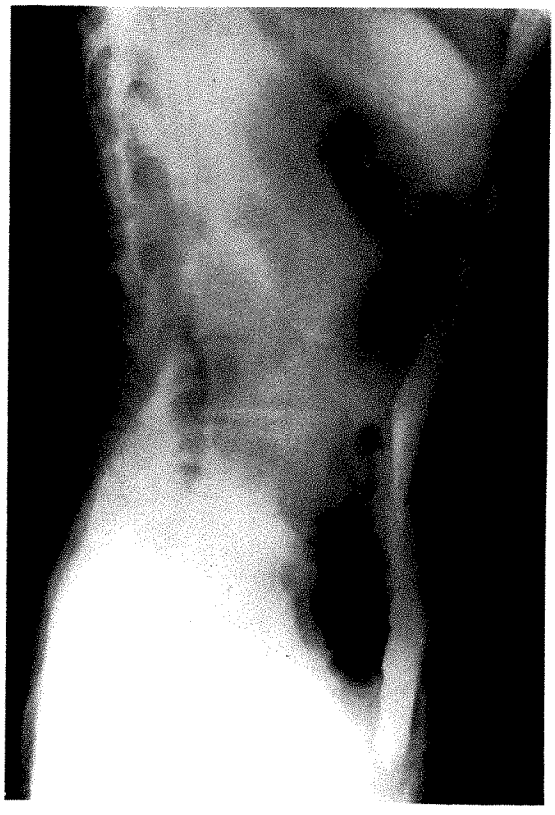


Fig. 5. Contrast material filling of the entire posterior rectus sheath of a cadaver.

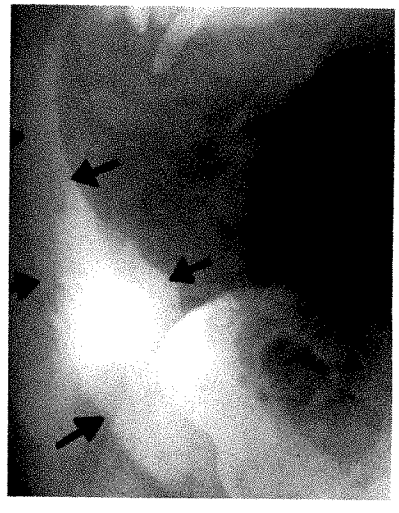


Fig. 6. Case I. Acute massive rectus hematoma. Spindle shaped widening of the rectus shadow.

abdomen, however, with soft tissue technique revealed localized widening of the rectus shadow (Fig. 7). At surgery hematoma of the rectus sheath was found, drained and the patient was discharged 10 days later.

Case III. A 30 year old male was playing with his child who jumped on the patient's lower abdomen. Following this incident, he experienced pain in the right lower quadrant and developed a fever up to 101° F. Physical examination revealed a small tender mass in the right lower abdomen. The lateral roentgenogram of the abdomen with soft tissue technique showed widening of the rectus shadow in its right lower portion, indicating the presence of a small rectus sheath hematoma. He was treated conservatively without operation (Fig. 8).



Fig. 7. Case II. Acute rectus hematoma. Widening of the rectus shadow is less marked than in Case I.

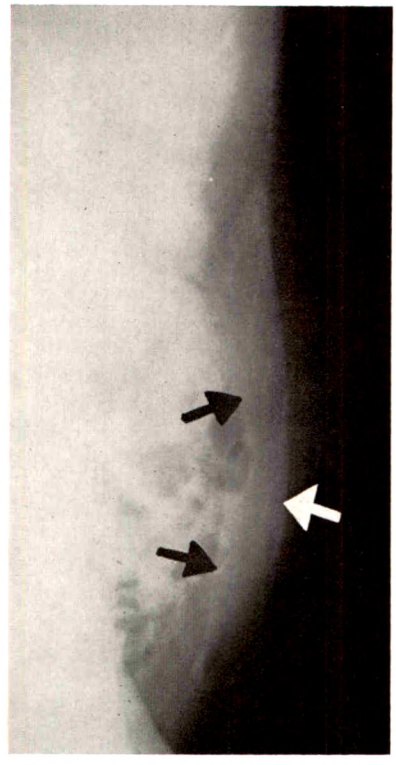


Fig. 8. Case III. Widening of the lower rectus shadow, posterior rectus sheath hematoma. Similar to Case II.

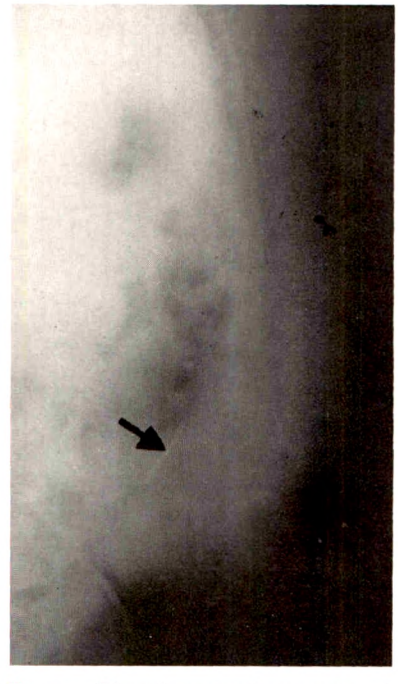


Fig. 9. Case IV. Chronic rectus hematoma with calcification. Widened rectus sheath with thin plate like calcification.

Case IV. A 42 year old obese female was admitted with essential hypertension and a painful lump in the right lower quadrant. Physical examination revealed a firm exquisitely tender mass in the right lower quadrant with rebound tenderness. The patient was referred for roentgenographic examination with the diagnosis of right lower quadrant abdominal mass (Fig. 9). The lateral roentgenogram of the abdomen demonstrated widening of the rectus shadow with a thin walled shell like calcification and the diagnosis of chronic hematoma of the rectus sheath was made and confirmed by surgery. The calcification was excised, the hematoma removed and the patient recovered. Roentgenograms of the specimen showed this to be bone (Fig. 10, *A* and *B*).

## DIFFERENTIAL DIAGNOSIS

Chronic rectus hematoma with calcification should be differentiated from ossifications of abdominal scars following gastric surgery. The ossifications of scars are asymptomatic and therefore are often overlooked. Their location is supraumbilical and in the mid line. Ossifications of surgical scars can also follow operations in the lower abdominal region. The ossification may involve the entire length of the scar or only a part of it. Ossification of the scar following gastric surgery was described in 1965 by Reboul *et al.*[21] These authors collected 15 cases from their own

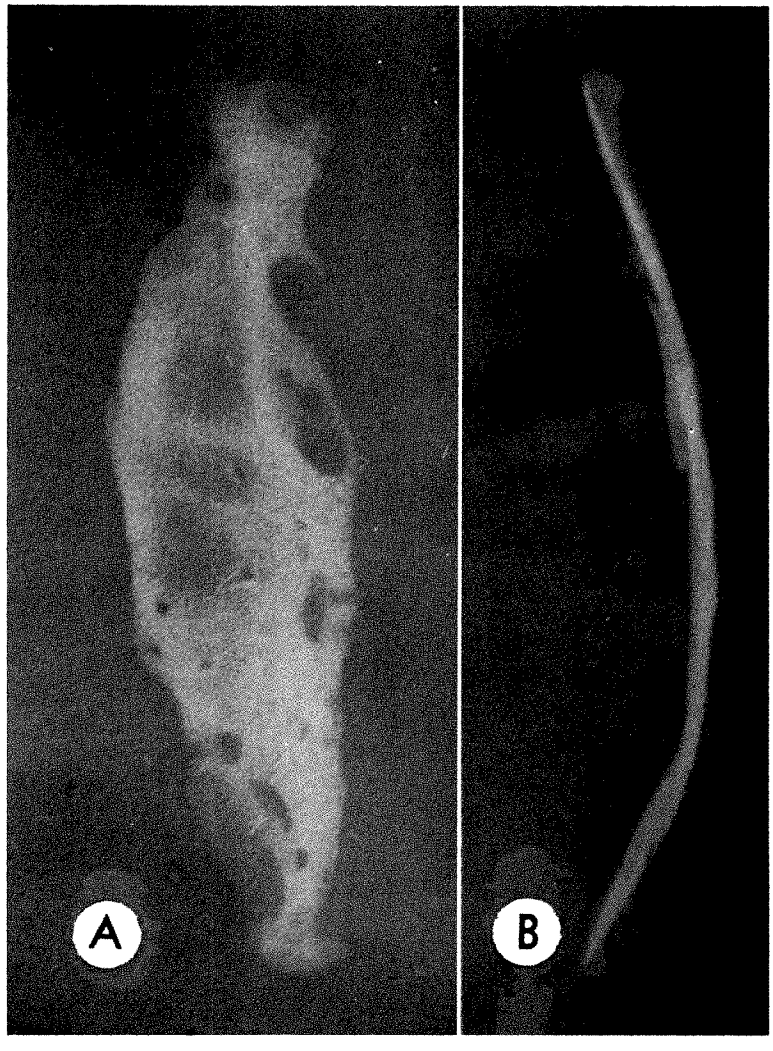



Fig. 10. Case IV. (*A*) Anteroposterior and (*B*) lateral views of the specimen.

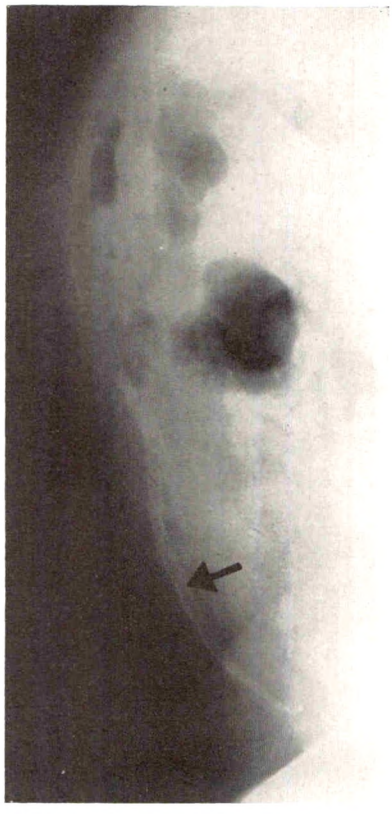


Fig. 11. Case v. Harpoon shaped ossification in surgical scar following sigmoid resection.

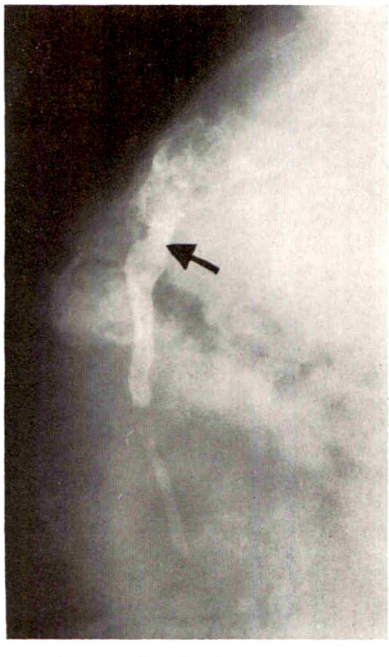


Fig. 12. Case vi. Large elongated ossified scar beneath the xiphoid.

material and found 104 additional cases in the literature.

The ossification occurs almost exclusively in males, whose age is over 50 years, in a mid line supraumbilical scar. It generally appears in approximately 1 or 2 years after the operation, but it may also occur much earlier. The shortest interval that has been observed was 1 month. The ossified mass has the appearance of an elongated hard nontender plaque below the xiphoid. In some cases, it may cause mild discomfort if the patient leans forward. The histologic structure is that of bone. It can be easily demonstrated by a roentgenogram taken in the lateral or oblique position with soft tissue technique. In the material at this hospital, 2 cases of ossified scars have been found following gastric surgery and 1 following resection of the sigmoid colon.

## Report of Additional Cases

Case v. A 74 year old white male, in whom a sigmoid adenocarcinoma had been resected 5 years previously, was admitted with left lower quadrant pain. Physical examination revealed a hard nontender mass in the left lower quadrant. Lateral roentgenogram of the abdomen showed an elongated, ossified, curved density in the anterior abdominal wall (Fig. 11). Laparotomy was performed which disclosed no intra-abdominal pathology, but did show a calcified mass in the anterior abdominal wall, measuring 7 cm. × 4 cm. × 1 cm. The mass was biopsied and the wound was closed. Microscopic examination of the biopsy specimen showed skin and old scar tissue with foci of old hemorrhage and metaplastic bone with marrow.

Case vi. An 84 year old white male was admitted February 28, 1964, because of repeated tarry stools. Roentgenologic study of the gastrointestinal tract revealed a large benign ulcer of the antrum of the stomach and a small hiatus hernia. The patient underwent surgery March 26, 1964. A mid line incision was made starting from the xiphoid and terminating near the umbilicus and a pyloroplasty was performed. A year later, during a follow-up gastrointestinal examination an ossified mass was visualized below the xiphoid process (Fig. 12).

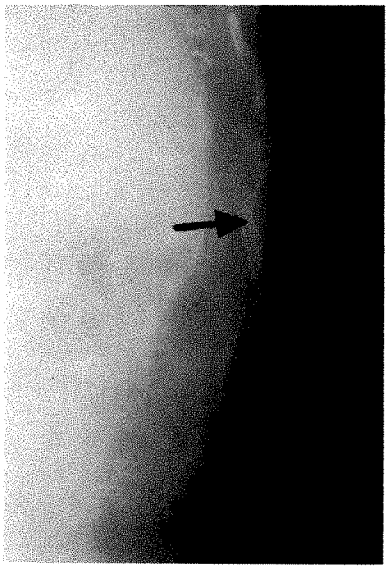


Fig. 13. Case vii. Another case of ossified surgical scar beneath the xiphoid.

Case vii. A 73 year old white male had a repair of an hiatus hernia through an upper midline incision in 1964. His hospital course was uneventful and he was discharged, symptom free. A year later, he was readmitted with hematemesis. A routine upper gastrointestinal series revealed an ossified mass in the surgical scar which had not been present the previous year (Fig. 13).

Case viii. A 79 year old white male was operated on in June, 1956 with a diagnosis of cholelithiasis. A high, right upper quadrant incision was made. In February, 1967 during a routine gastrointestinal study, an ossified scar was demonstrated.

In the opinion of Reboul *et al.*[21] in all probability, a process of heterotopic ossification is involved in such cases under the influence of enzymatic reaction and of local factors in the linea alba.

## SUMMARY

Acute hematoma in the rectus sheath is manifested on the lateral roentgenogram as a placenta shaped or ovoid spindle shaped mass in the anterior abdominal wall, or as widening of the rectus shadow.

Chronic hematoma with ossification shows a thin walled, shell like, broad ossified mass in the widened rectus shadow, since it is confined in the tight compartment of the rectus sheath.

A postoperative scar with ossification is finger thick, curved, elongated, pencil shaped with a diameter about 2×2 cm. or more. Its location in the anterior abdominal wall is more superficial, separated from the rectus. It is embedded in the scar tissue and follows its course.

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# ROENTGEN VISUALIZATION OF PHLEBOLITHS IN HEMANGIOMA OF THE GASTRO-INTESTINAL TRACT*

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ROENTGEN visualization of a cluster of phleboliths in the abdomen in an atypical location should suggest a diagnosis of hemangioma. If the entire group of phleboliths shifts position but maintains a constant relationship to each other, then the hemangioma is probably in the gastrointestinal tract or mesentery. Contrast studies should establish the diagnosis by demonstrating a lesion in the alimentary tract intimately associated with the phleboliths.

The symptoms of hemangioma of the gastrointestinal tract are those referable to bleeding, obstruction and intussusception.

It is interesting to note that as early as 1919, Ruggles[7] showed examples of phleboliths in hemangioma of the soft tissues of the jaw, arm, forearm and thumb, and stated that "this type of growth also occurs in the liver and intestine, so that it is possible that similar shadows may be observed in those regions." Thus he predicted that roentgen diagnosis of hemangioma of the bowel could be made.

Preoperative diagnosis of hemangiomas has been made on the basis of the roentgen findings in the stomach,[2] jejunum,[6] ileum,[5] colon,[1] sigmoid,[4] and rectum.[3]

In a case to be reported here, this sign of atypically located intra-abdominal phleboliths, which should have been obvious, unfortunately was overlooked, not only at our hospital but at 2 other hospitals to which the patient had been admitted for gastrointestinal bleeding. Certainly in retrospect, this roentgen finding was plain and clear. The patient had been and continued falsely to be labelled as a case of bleeding duodenal ulcer which befogged the obvious. Therefore, we feel that this roentgen sign merits re-emphasis.

## REPORT OF A CASE

A 40 year old Negro male was admitted to the Methodist Hospital because of severe anemia and weakness. There were two admissions, 2 years and 5 years previously, to two other hospitals for gastrointestinal bleeding. He was discharged from the second hospital after 7 weeks with a diagnosis of bleeding duodenal ulcer.

At the present admission, his symptoms included periodic epigastric and periumbilical pain when hungry, with relief after eating. The stool examination was slightly positive for occult blood. The hemoglobin was 5.4 gm. per cent and the hematocrit 21 per cent. There was moderate anisocytosis, mild poikilocytosis, mild polychromatophilia, many microcytes, several macrocytes, marked hypochromia and a few target cells. The white blood cells were normal in appearance with a normal differential and normal total count. No evidence of blood dyscrasia was found.

The upper gastrointestinal series showed equivocal mild deformity of the duodenal bulb. A group of multiple, small, discrete, round, calcified bodies were present in the mid abdomen which shifted easily and they had the appearance of phleboliths (Fig. 1, 2 and 3).

The preoperative clinical diagnosis was bleeding duodenal ulcer. The patient was given 3 transfusions of 500 cc. each.

At laparotomy there was a vascular tumor in the jejunum about 30 cm. distal to the ligament of Treitz. A segment of jejunum containing the tumor was resected. A gastrotomy was also done and there was no evidence of gastric or duodenal ulcer. All other organs appeared normal.

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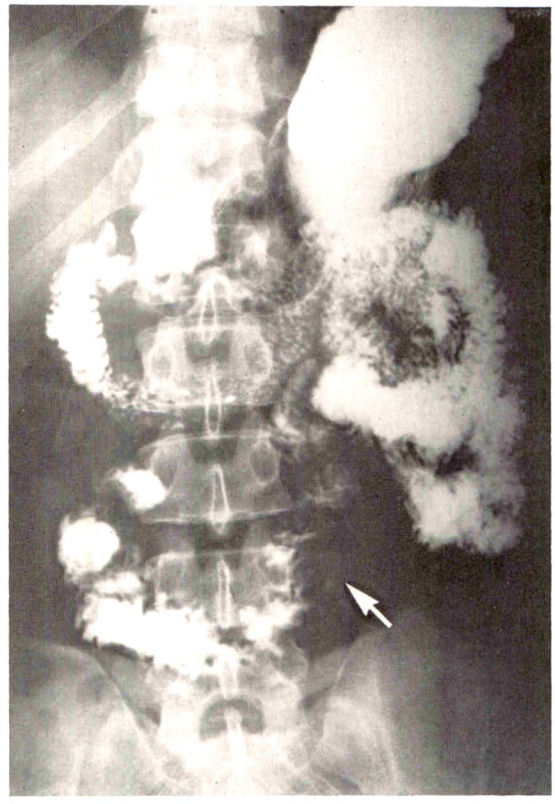


Fig. 2. Arrow points to phleboliths and the adjacent slightly narrowed segment of jejunum.

The specimen showed an oval mass measuring approximately 8×5 cm. which appeared mottled with light and dark granularity and nodularity. The long axis was parallel with that of the bowel. The mass contained closely placed minute vascular channels engorged with blood and it extended through the bowel wall to the mucosa.

The microscopic examination showed that the hemangioma extended through the wall of the small bowel involving the muscularis, submucosa and mucosa. The hemangioma was plexiform and composed of intercommunicating dilated channels partly enclosed by smooth muscle walls. Several channels were thrombosed. There were a few foci of calcification. The diagnosis was hemangioma of the venous cavernous type.

The patient was in good condition when discharged.

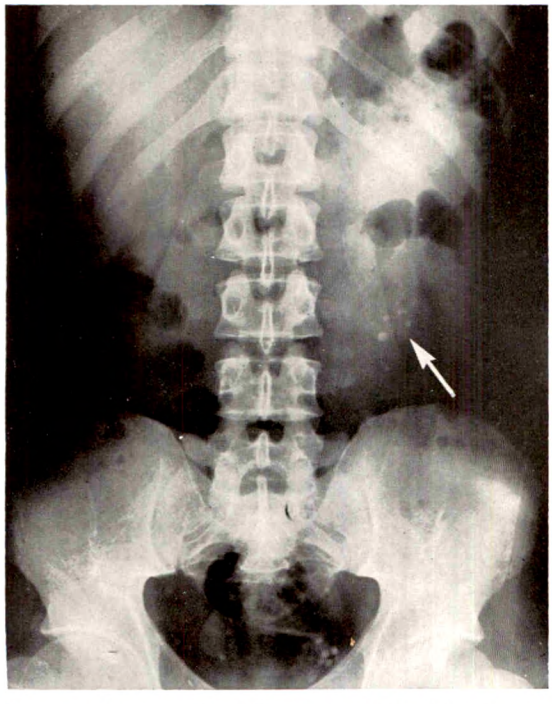


Fig. 1. Cluster of abdominal phleboliths in an atypical position. Note the typical location of pelvic phleboliths.

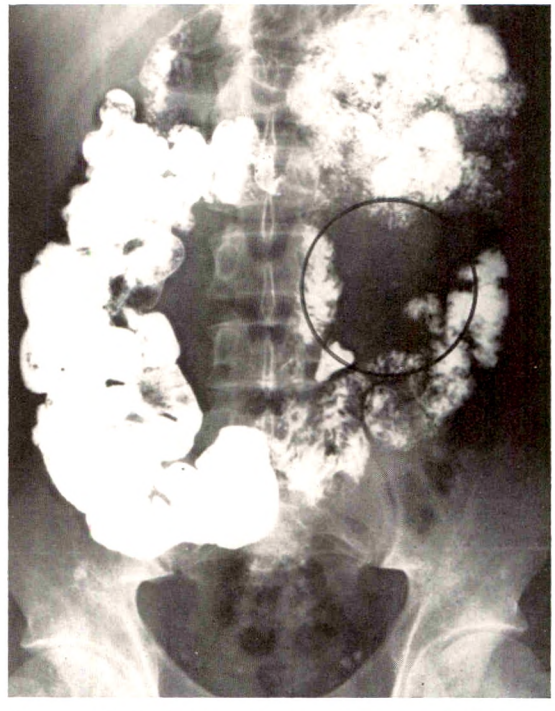


Fig. 3. Circle indicates area of separated loops of bowel at the site of the hemangioma, which measured 8×5 cm. on the gross specimen.

SUMMARY

A case of hemangioma of the jejunum is reported.

The presence of a cluster of mobile, intra-abdominal phleboliths in an atypical location is a reliable roentgen sign of hemangioma somewhere in the gastrointestinal tract or the mesentery. The diagnosis is established when barium studies reveal a lesion intimately associated with phleboliths.

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# TUBERCULOUS PERITONITIS*

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TUBERCULOUS peritonitis has a relatively constant incidence.[4] At the New York Hospital—Cornell Medical Center 4 cases with roentgenologic documentation have been seen in the past year. This report is to review the subject and state the findings which have not appeared in the radiologic literature in 15 years.

Tuberculous peritonitis is to be distinguished from tuberculous enteritis with its ulcerative and scarring characteristics. The involvement of the serous cavity can be by direct extension, or by hematogenous or lymphatic routes. However, in 90 cases Auerbach[1] did not find a single case of rupture of a mesenteric node. In over 50 per cent the primary site is not demonstrable clinically or roentgenographically.[2,5] Three forms are generally recognized. There is the wet type, with ascites or pockets of loculated fluid; a dry form with caseous nodules and adhesions creating a "plastic" abdomen; and a third form in which the fibrotic omentum appears as an abdominal mass.[1,2,5]

Clinically the onset can be rapid, or present with symptoms persisting over the course of years. The association of cirrhosis with ascites complicated by peritonitis is well known.[3] These patients were in high number in the study of Burack and Hollister[4] at the Boston City Hospital. The doughy abdomen is characteristically not present, occurring in less than 10 per cent of the cases. Abdominal distention, chronic or acute abdominal pain and fever are usual. Further history may reveal weight loss and night sweats. Laboratory data often include secondary anemia and hypoproteinemia. The white blood cell count is rarely over 12,500. Paracentesis yields a nonbloody clear fluid with a protein over 2.5 gm. per cent and a leukocytosis of over 250 cells/cu. mm. Nearly one half of the cultures are negative, and guinea pig inoculations are positive in only 60 per cent of the determinations.[3] The association of skeletal and genitourinary tuberculosis is high and should be considered.[1,5]

Roentgenologically, the small bowel will show accelerated or delayed motility, fixation of bowel loops, slight separation of adjacent segments and an elevation of the bowel out of the pelvis. Rigidity of the walls, spiculation and some narrowing may be encountered.[1,6] Stierlin's sign of spasm of the cecum is characteristic of tuberculous enteritis as is ulceration. The colon often shows fixation, manifested by the failure of the usual descent of the transverse colon after evacuation. Adhesive bands may distort the normal configuration of the colon. Plain roentgenograms show nonspecific ileus with or without ascites.

## REPORT OF CASES

Case 1. 1028180. A 31 year old Negro female was admitted in October, 1965 with chronic renal disease and uremia. Hepatomegaly and splenomegaly were noted. She had a hematocrit of 15 per cent and a positive tuberculin skin test. A urine culture was negative for tuberculosis. After some improvement in her condition, she was discharged. Two months later she presented with a history of increasing abdominal girth associated with fever of 2 weeks' duration. She was anemic, had ascites and ankle edema. Her chest roentgenogram was normal. A roentgenogram of her abdomen (Fig. 1) showed bulging flanks, and separation of the loops compatible with ascites and ileus. On paracentesis, the fluid showed 4.5 gm. per cent protein. Cultures (the results of which were returned later) of the bone marrow, peritoneal fluid and blood were negative for tuberculosis. On the second hospital day she expired. Autopsy revealed caseating granuloma in the peritoneum, studding the peritoneum, and involving the mesenteric lymph nodes as well as the hilar lymph nodes. Dense adhesions produced fixation of

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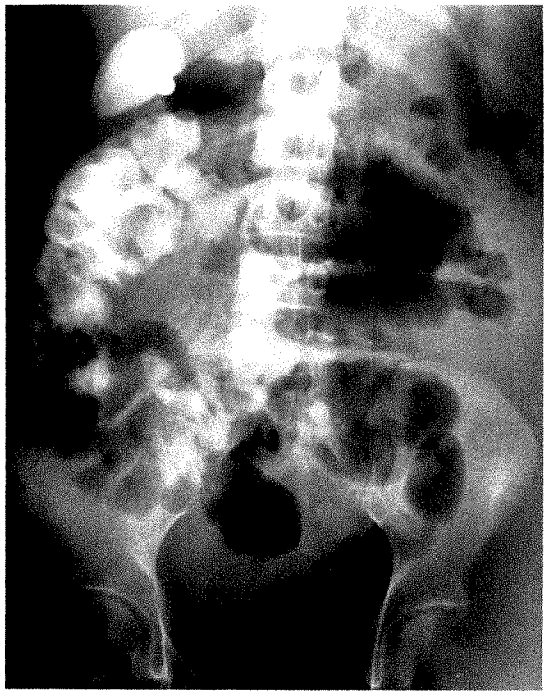


FIG. 1. Opaque material in the gallbladder and colon is secondary to a previous cholecystography. There is ileus associated with ascites.

the omentum to the parietal peritoneum. Acid fast stains revealed numerous bacteria.

CASE II. 1012756. A 49 year old Negro male presented with a history of chronic alcoholism, fever of 3 weeks' duration, and abdominal soreness and night sweats of 3–4 days' duration associated with abdominal swelling. A paracentesis showed fluid which contained 2,500 white blood cells per cu. mm. (80 per cent lymphocytes) and protein of 6.0 gm. per cent. His hematocrit was 33 per cent and the skin test was positive. Cultures of sputum, peritoneal fluid and gastric washings were negative. A urine culture which was obtained at the time was positive for tuberculosis after 1 month's growth. A small bowel series showed separation of loops and a diffusely coarse pattern with fixation (Fig. 2, *A* and *B*). Laparotomy revealed the omentum stuck to the peritoneum with pockets of ascitic fluid. The cecum was entered because of the massive adhesions. An omental biopsy showed proliferating fibrous connective tissue with caseating granuloma. Cultures and smears were negative, but the slides were considered most compatible with tuberculosis. He was treated with INH but had

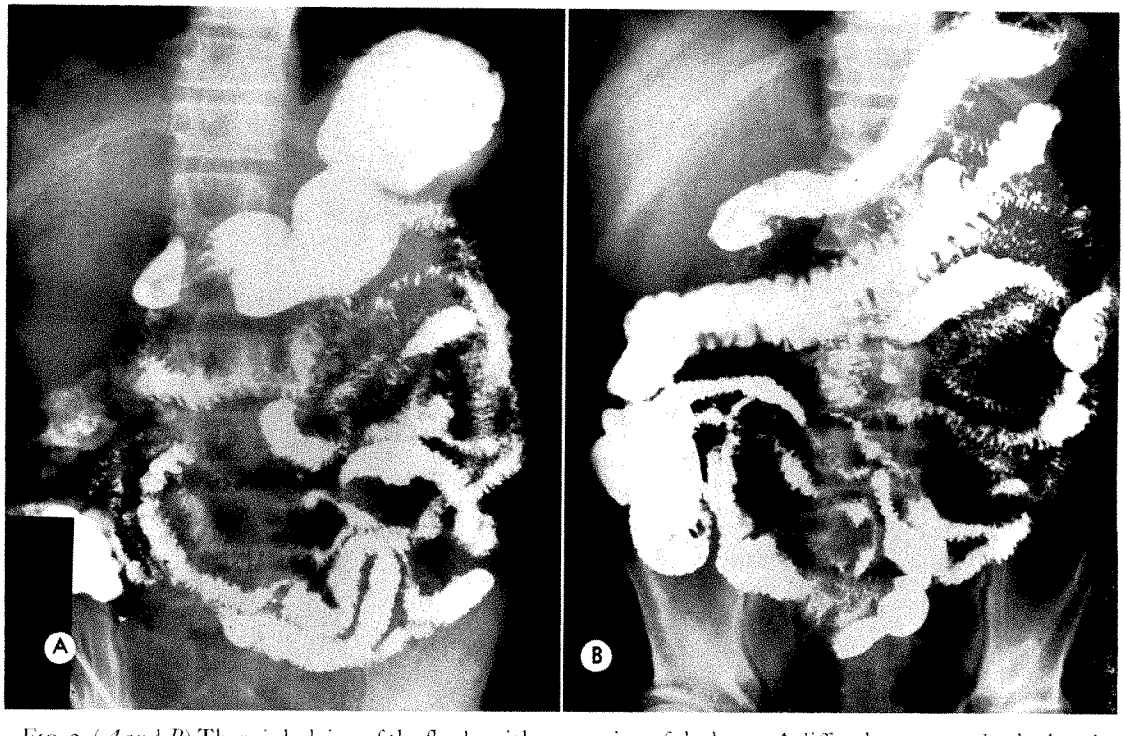



FIG. 2. (*A* and *B*) There is bulging of the flanks with separation of the loops. A diffusely coarsened spiculated mucosal pattern is seen with crowding of the small bowel into the center of the abdomen.

alteration of his liver function. Secondary antituberculous drugs were used with gradual improvement. The chest roentgenogram and intravenous pyelograms were normal.

Case III. 917887. This 23 year old Negro male had rheumatic fever and subsequent flexion contracture of his left elbow in 1956. An elbow biopsy because of pain and swelling in 1959 revealed tuberculosis. In 1960 an operation for tuberculous osteomyelitis was done. An abdominal skin graft to the site followed the original procedure. In 1964 the grafted elbow broke down. He was lost to follow-up when he was told of a possible re-operation. In November 1965 he presented with left chest and diffuse abdominal pain. Roentgenograms of the elbow showed no change. His gastric and sputum cultures were negative. His hematocrit was 49 per cent and his white blood cell count was 7,000. In February, 1966 he was seen again. He had not followed his drug program. His history was 2 months of low grade fever, diarrhea, anorexia and a 24 pound weight loss. A questionable fluid wave was noted in a tender abdomen. His hematocrit was 36 per cent and the white blood cell count was 10,000. The plain roentgenogram (Fig. 3) showed ascites and nonspecific ileus. The skin test was positive. Smears of the fluid were negative. A small intestinal series (Fig. 4, *A–C*) showed central location of the small bowel. There was spiculation and slight separation of loops associated with fixation and elevation out of the pelvis. The flanks were bulging. A chest roentgenogram was normal. An exploratory laparotomy revealed adhesions with miliary nodules scattered over the abdomen and serosal surfaces. Microscopically there were caseating granulomas and positive acid fast stains for bacteria. Langhans' giant cells were a conspicuous feature. Cultures of the omental tissue and peritoneal fluid were positive for tuberculosis. His subsequent course has been a lysis of the fever on antituberculous medications.

Case IV. 253608. This 60 year old Negro woman had a past history of an appendectomy associated with peritonitis with gram negative bacteria secondary to a perforation in 1940. She had had right pleurisy in 1936 and an episode of hemoptysis in 1939. In June, 1965 she presented with right upper quadrant pain of 2 weeks' duration. Her chest roentgenogram

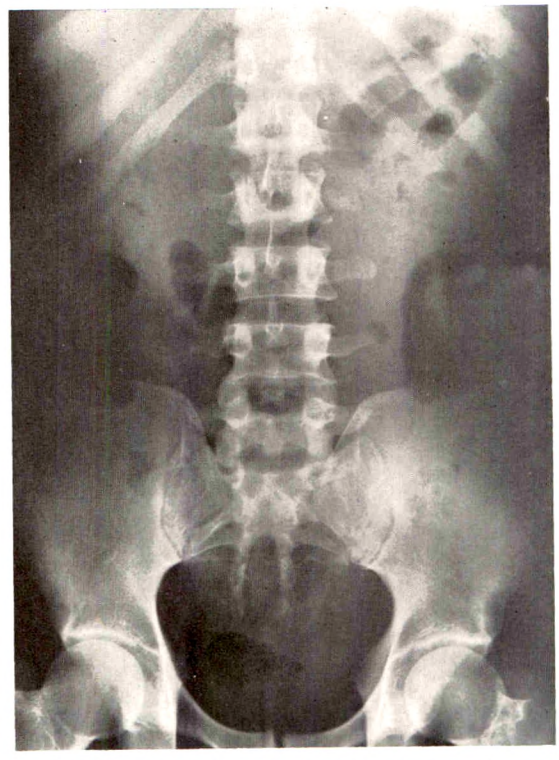


Fig. 3. There is ascites. An irregular isolated loop of small bowel appears in the left mid abdomen.

showed right hilar calcification and slight cardiomegaly. The abdominal roentgenogram demonstrated a right upper quadrant calculus (which was later placed in the gallbladder on an intravenous cholangiogram) and ileus. At operation, implants were scattered over the peritoneal surface and small bowel. Adhesions were noted between several loops. A narrowed segment of ileum without obstruction but associated with many more nodules was seen. Several nodes were biopsied. A cholecystostomy with drainage was performed. Microscopically she had chronic cholecystitis and cholelithiasis. Granulomas with central necrosis were found in the lymph nodes and the implants. Acid fast stains were positive. She has subsequently done well on antituberculous therapy after an initial postoperative course of prolonged ileus without obstruction.

## DIFFERENTIAL DIAGNOSIS

The principal differential diagnosis of tuberculous peritonitis includes nontuberculous peritonitis, regional enteritis, lymphoma, metastasis to the small bowel and




FIG. 4. (*A–C*) There is centralization of the small bowel. Coarsening, separation and diffuse spiculation are present. There are no loops in the pelvis.

mesentery as well as ascites associated with hypoproteinemia. Fixation of the loops militates against uncomplicated ascites or pyogenic peritonitis. Mass formation, localized dilatation and segmental narrowing with effacement and ulceration of bowel favors lymphoma. Narrowing of segments, particularly the terminal ileum with skip areas, and diarrhea suggest regional enteritis. Separation of loops, fixation and spiculation are common to all. The characteristic ascites with its high protein and leukocytosis should suggest a diagnosis of tuberculosis as would associated skeletal and genitourinary disease.

## CONCLUSION

It is clear that there are no pathognomonic signs or symptoms. However, the usual history and roentgenographic signs of tuberculous peritonitis might suggest the diagnosis or indicate the appropriate laboratory studies before the return of positive cultures or exploratory biopsy. An erect roentgenogram during the small intestinal examination might show fixation with lack of descent of the bowel into the pelvis as a further differential point.

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# TUBERCULOSIS OF THE PYLORO-DUODENAL AREA*

## REPORT OF FOUR CASES

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TUBERCULOSIS of the stomach and duodenum is rare even in patients with pulmonary or intestinal tuberculosis. Its preoperative diagnosis has infrequently been made and still remains a challenging problem to the clinician and the radiologist.

The incidence of gastric tuberculosis in specimens examined following gastric resection varies between 1 in 75,000 (Sullivan *et al.*[26]) and 3 in 1,568 cases (Demel[10]). The incidence of gastric tuberculosis at autopsy, in patients with pulmonary tuberculosis, has been 0.36–2.3 per cent (Gains *et al.*[14]), and duodenal tuberculosis occurred in only 0.5 per cent of the cases of tuberculosis autopsied (Feldman[12]). The investigations of Ricard and Chevrier[22] revealed that about 10 per cent of gastric tuberculosis cases had duodenal involvement, but of the 16 cases of pyloric tuberculosis the duodenum was involved in 9 cases.

The roentgenologic studies of pyloro-duodenal tuberculosis are few. Gains *et al.*,[14] in 1952, could collect only 4 cases of tuberculosis of both the stomach and the duodenum that had roentgenologic examinations prior to pathologic study. Since then, no report has appeared in the English language literature showing simultaneous involvement of the stomach and duodenum. Although much has been written on the pathology of gastric tuberculosis and some points for roentgenologic diagnosis are suggested on the few cases reported,[1,5,8,14,19,20] yet the detailed roentgenologic features of tuberculosis at the pyloro-duodenal area have not been well illustrated.

The purpose of the authors is to present the clinical and detailed roentgenologic findings of 4 cases of tuberculosis of the stomach and duodenum localized primarily at the pyloro-duodenal area.

### REPORT OF CASES

CASE 1. M.K., a 45 year old male, was admitted to the hospital with the complaint of progressively increasing postprandial epigastric pain and vomiting of 1 year's duration. He was afebrile with a mildly emphysematous chest but the rest of the physical examination was essentially negative. An upper gastrointestinal series revealed a diffuse irregularity of the wall of the duodenum with slight dilatation of the lumen. A small ulcer was suspected at the junction of the descending and transverse part of the duodenum, and few small nodular defects were seen in the descending duodenum (Fig. 1). Antacids and antispasmodics were given with no improvement of his symptoms.

The hematocrit was 30 per cent, white blood cell count 12,800 with normal differential, and his blood chemistry was within normal limits. A chest roentgenogram revealed fibrotic changes of reinfection tuberculosis in the right upper lobe but no soft infiltrates.

At operation, the first, second and third portions of the duodenum were found to be involved in an infiltrative process invading the entire wall of the duodenum with large nodule formation on the serosa. The terminal jejunum contained within its wall a nodular mass, 2×1 cm., and there were several enlarged lymph nodes in the mesentery of the small intestine. Retrocolic gastrojejunostomy was performed. Biopsy from the mesenteric lymph nodes revealed hyperplasia and tubercles with caseation surrounded by epithelioid cells and some Langhans giant cells.

Following antituberculous treatment, there was slight improvement in his symptoms but a large mass was felt in the epigastrium and a

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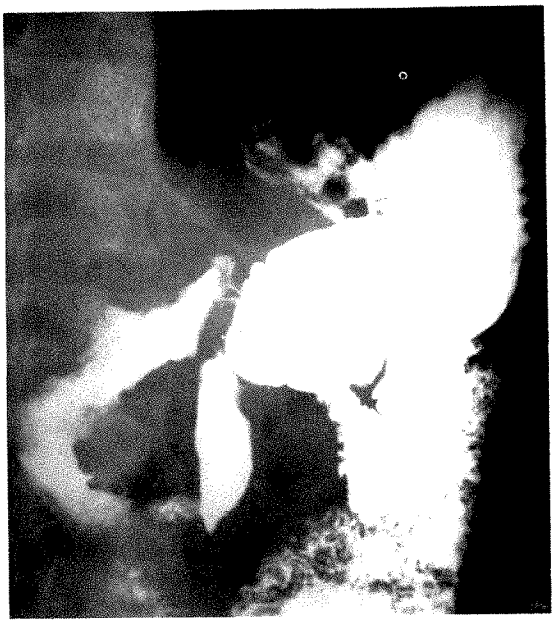


FIG. 1. Case I. Roentgenogram showing slight dilatation and diffuse irregularity of the wall of the duodenum (proved infiltrative lesion at operation).

follow-up upper gastrointestinal series, 7 months after the operation, showed large nodular lesions with smooth and well demarcated margins almost completely filling the lumen of the descending part of the duodenum (Fig. 2*A*). There was complete effacement of the gastric antral mucosa by a large constricting lesion with considerable pyloric stenosis; the gastroenterostomy was functioning well (Fig. 2*B*). Ten days later, he was seen with symptoms of small bowel obstruction, was advised hospitalization but was lost to follow-up.

CASE II. M.A.H., a 37 year old male was admitted with the complaint of burning epigastric pain of 3–4 years' duration, coming before

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A small ulcer is suggested at the junction of the descending and transverse part of duodenum, and few small nodular defects in the descending duodenum (nodular lesions were felt at operation). The pyloric canal and gastric antrum are uninvolved.

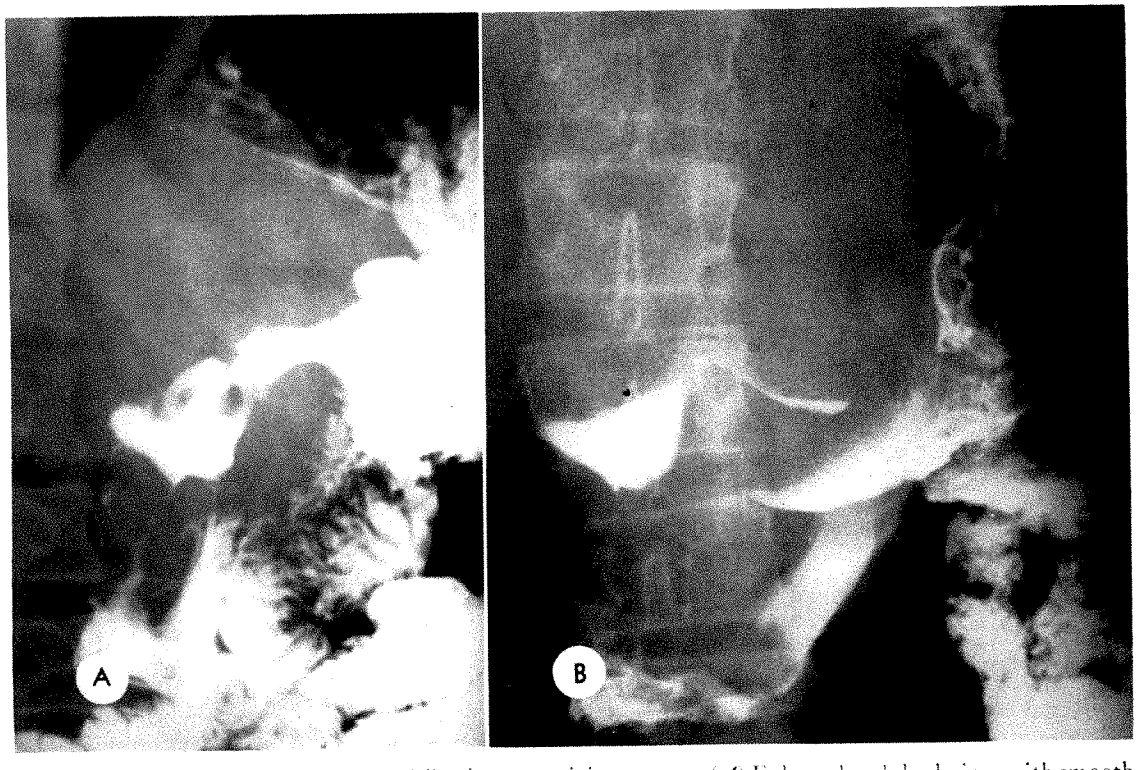



FIG. 2. Case I. Seven months later and following gastrojejunostomy. (*A*) Enlarged nodular lesions with smooth and well demarcated margins are seen almost completely filling the lumen of the descending part of the duodenum. (*B*) Complete effacement of the gastric antral mucosa by a large constricting lesion with considerable pyloric stenosis. The gastroenterostomy is functioning well.

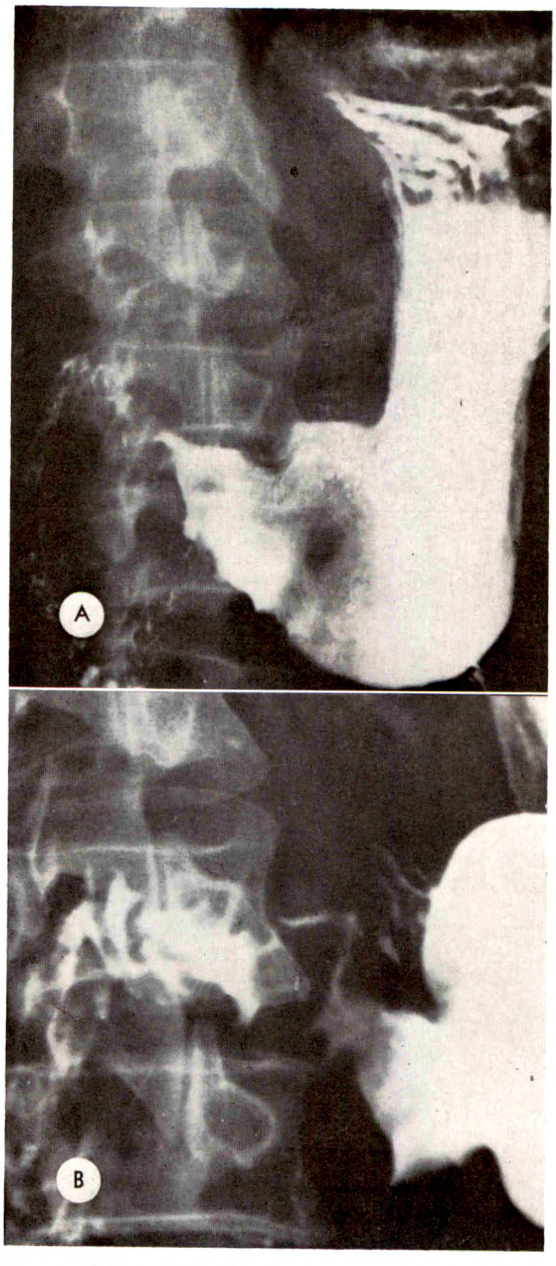



FIG. 3. Case II. (*A*) Roentgenogram demonstrating irregular tapering of pyloric antrum with poor filling of the duodenum. (*B*) Spot roentgenogram showing a short narrowing with nodularity of the wall of the pyloro-duodenal area. The duodenal bulb is irregularly deformed with no evidence of ulcer crater.

meals and relieved by food intake. About 8 months prior to admission, he started having postprandial vomiting, one-half hour after ingestion, and on one occasion he had hematemesis. For 2 months prior to admission, he was treated for duodenal ulcer.

On admission, he was afebrile, his liver was 4 cm. below the costal margin and he had a fixed hard mass, measuring about 6 cm. in diameter in the epigastric region. The rest of the physical examination was essentially negative. His hemogram and blood chemistry were normal. The sedimentation rate was 24 mm. per hour, his P.P.D. ++, and the gastric juice analysis showed normal acidity.

The chest roentgenogram was negative. An upper gastrointestinal series revealed irregular tapering of the pyloric antrum with delayed gastric emptying (Fig. 3*A*). There was a short narrowing with nodularity of the wall of the pyloro-duodenal area. The duodenal bulb was irregularly deformed with no evidence of crater or any cicatricial contracture suggestive of a healing peptic ulcer deformity (Fig. 3*B*). The preoperative diagnosis was lymphoma involving the pyloro-duodenal area.

At operation, there was a big irregular hard mass at the pylorus and there were many enlarged lymph nodes throughout the mesentery of the intestines. One of the lymph nodes contained caseous material. In the liver, there were many hard nodules. A gastroenterostomy and liver biopsy were performed. The liver biopsy revealed granulomatous inflammation and tubercles with central necrosis, abundant epithelioid cells and few giant cells of the Langhans type. He was given antituberculous therapy.

CASE III. S.H.A., a 25 year old male was admitted with the complaint of mild epigastric pain, occasional postprandial vomiting of 3 years' duration and severe epigastric burning of 1 year's duration. A month prior to admission, an upper gastrointestinal series done here had revealed a markedly dilated stomach with retention of barium and marked decrease of peristalsis (Fig. 4*A*). The spot roentgenogram showed narrowing, irregularity and elongation of the pyloric canal with gradual tapering of the pyloric antrum. The duodenal bulb showed deformity and a penetrating ulcer tract (Fig. 4, *B* and *C*). The chest roentgenogram was negative.

Physical examination revealed an emaciated, ill looking man with a tympanitic fullness in the epigastrium that was tender on pressure. His spleen was 4 cm. below the costal margin. The hemogram and blood chemistry were normal. The gastric juice analysis showed normal acidity. The preoperative diagnosis was posterior perforating peptic ulcer.

At operation, the stomach was markedly

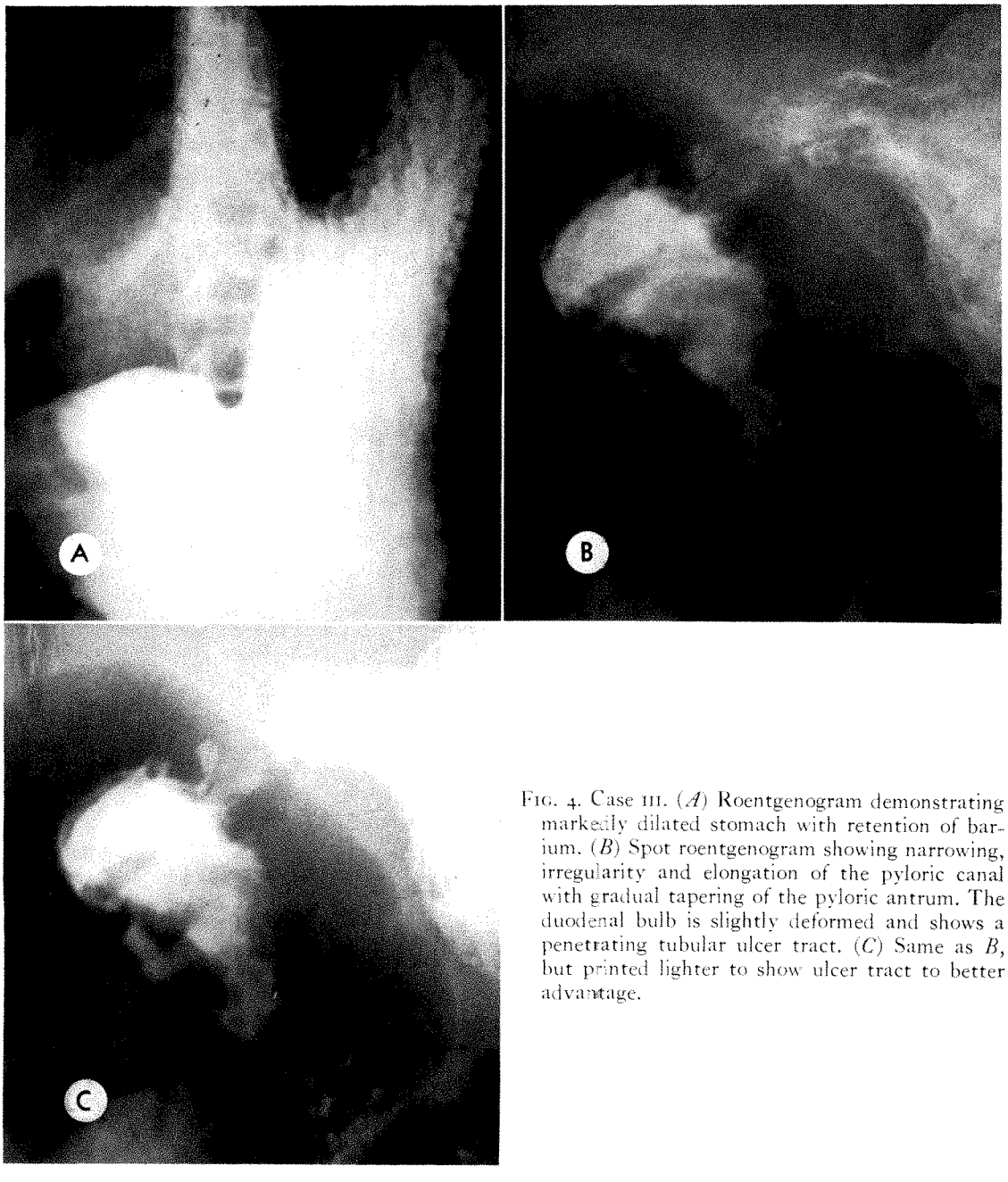



Fig. 4. Case III. (*A*) Roentgenogram demonstrating markedly dilated stomach with retention of barium. (*B*) Spot roentgenogram showing narrowing, irregularity and elongation of the pyloric canal with gradual tapering of the pyloric antrum. The duodenal bulb is slightly deformed and shows a penetrating tubular ulcer tract. (*C*) Same as *B*, but printed lighter to show ulcer tract to better advantage.

dilated. At the pyloric region, a firm scarred mass was palpable around the pylorus, and the first portion of the duodenum was adherent to the pancreas. The spleen was three times the normal size. A partial gastrectomy and gastrojejunostomy were performed. Histologic examination of the pylorus revealed conglomerate tubercles with epithelioid cells, Langhans giant cells and foci of necrosis. The tubercles extended to the submucosa and the muscularis. The lymph nodes contained numerous tubercles. He was given antituberculous therapy.

Case IV. F.Kh., a 51 year old male was admitted with the complaint of dull epigastric pain of 1 year's duration which was not relieved by food intake. Two months prior to admission, he had repeated attacks of vomiting, hematemesis and melena. On the day of admission, he was having cough, productive of bloody spu-



FIG. 5. Case IV. Roentgenogram showing deformity and stenosis of the pyloro-duodenal area and a saccular ulcer crater that retained the barium from a previous barium swallow study (made a day earlier). A constricting lesion in the lower esophagus exhibited chronic inflammation and failed to show tubercles on a biopsy taken at esophagoscopy.

tum. His blood pressure was 90/60 mm. of mercury. He was emaciated and pale and had mild epigastric tenderness but no hepatosplenomegaly.

The hemoglobin was 4.5 gm. per cent, hematocrit 14 per cent, white blood cell count 8,100 with normal differential, and the stools guaiac 4+. Total serum protein was 5.8 gm. per cent, albumin 2.4 gm. per cent and globulin 3.4 per cent. The rest of the blood chemistry was normal.

A chest roentgenogram showed irregular infiltrates in both apices highly suggestive of active reinfection phase of tuberculosis. An upper gastrointestinal series revealed a constricting lesion of the esophagus, deformity and stenosis of the pyloro-duodenal area and a penetrating ulcer crater that retained the barium from a previous barium swallow study made a day earlier (Fig. 5).

At operation, the pyloro-duodenal area was completely involved with edema, adhesions, scarring and an active mucosal ulceration. The lymph nodes along the distal end of the greater curvature were enlarged and matted together. Subtotal gastrectomy and gastrojejunostomy were performed. Histologic examination of the pyloric end of the stomach and the lymph nodes revealed an ulcer and an inflammatory process of the stomach involving the mucosa, submucosa and the muscularis. There were discrete and coalescing tubercular structures composed of epithelioid cells and occasional Langhans giant cells and central necrosis. A similar process was present in the lymph nodes. Biopsy of the lower esophagus showed inflammation, multiple foci of ulceration but no tubercle formation. He was given antituberculous therapy.

## DISCUSSION

### A. GENERAL PATHOLOGIC CONSIDERATIONS

The extreme rarity of gastric and duodenal tuberculosis in patients with pulmonary or intestinal tuberculosis led many authors to postulate several factors for this rare *localization*. These have been: sparsity of lymphoid structures in the gastric wall, intact gastric mucosa, acidity of the stomach, and rapid passage of the ingested organism through the stomach.[3,9,20] The possible *routes of infection* that have been considered include: (a) direct infection through the mucosa, (b) hematogenous spread, (c) lymphatic spread, and (d) spread from the serosa by continuity from adjacent structures, especially the lymph nodes.[1,5] The *pathologic types* of gastric tuberculosis have been classified into ulcerative, hypertrophic, solitary or miliary tubercles, larger nodular foci, and diffuse tuberculous gastritis.[1,4,6,11,16,17,20] Combinations of these lesions do frequently occur.[19,21] The ulcerative and hypertrophic types are the commonest.[20] The duodenal lesions are more recently classified as ulcerative, hyperplastic, infiltrative, and enteroperitoneal.[2,12,13,15] There is usually extensive lymph node involvement in the surrounding areas.[1,20]

### B. CLINICAL CONSIDERATIONS

The clinical picture of gastric tuberculosis is known to mimic gastritis, peptic ulcer and carcinoma. Pain after meals, epigastric

discomfort, vomiting and loss of weight may occur.[8] Duodenal tuberculosis may be symptomless, or give symptoms and signs identical with those of the usual form of duodenal peptic ulcer with obstruction.[2,18] In 50 per cent, there is a palpable mass and in about 50 per cent there is evidence of tuberculosis elsewhere in the body.[7,14,24,26] Three of our 4 cases had a palpable epigastric mass (Case I, II and III); 1 case had old residues of pulmonary tuberculosis (Case I) and another had active pulmonary lesions (Case IV). Hypochlorhydria and achlorhydria are said to be most frequent abnormalities.[21] In 2 of our patients in whom the test was done it showed normal gastric acidity.

### C. ROENTGENOLOGIC CONSIDERATIONS

The roentgenologic appearances are more conveniently classified as of predominantly ulcerative and predominantly hyperplastic type.[14,20] Three of our 4 cases presented some element of hypertrophy and 3 of them had ulcers. There was a small cuff-like constricting lesion at the pylorus in 1 case (Case III, Fig. 4*B*), a short area of narrowing with nodularity of the wall at the pyloro-duodenal area in a second case (Case II, Fig. 3*B*) and a large annular lesion involving almost the entire pylorus in another case (Case I, Fig. 2*B*). One of our cases first showed a diffuse infiltrative lesion of the entire duodenal loop with normal antrum and pyloric canal (Case I, Fig. 1) that later extended to involve the pylorus with flattening of the gastric mucosa (Case I, Fig. 2*B*). We have not encountered a similar retrograde extension reported in the literature.

Nodular lesions of the duodenum seen at operation were followed up by upper gastrointestinal series and were demonstrated to have grown in size, manifested smooth borders and obstructed the descending duodenum (Case I, Fig. 2*A*).

Ulcerative lesions were less commonly demonstrated roentgenologically than hypertrophic lesions, in the 49 cases reviewed by Gaines *et al.*[14] Two of our cases showed large ulcers at the pyloro-duodenal area, one of them retaining the barium for 24 hours (Case III, Fig. 4*B*; Case IV, Fig. 5), and in a third case there was suggestion of a small ulcer in the descending duodenum (Case I, Fig. 1).

Ackermann[1] suggested that if a fistulous tract could be demonstrated by roentgen examination, it would assist in the diagnosis of this entity, and he reported one such case. Since then, no similar case has been reported. We have demonstrated a penetrating tubular ulcer tract in a deformed duodenal bulb that, at operation, had extensive adhesions with the surrounding pancreas (Case III, Fig. 4*B*).

Ackermann[1] also believed that simultaneous involvement of the stomach and duodenum increases the possibility of a tuberculous etiology as compared to carcinoma which stops at the pyloric ring. Gaines *et al.*[14] raised the question of difficulty of differentiating it from a benign peptic ulcer deformity. None of our cases showed any of the typical pictures of chronic peptic ulcer deformity. We found it difficult in 1 case to differentiate from a lymphoma (Case II, Fig. 3*B*) which does not stop at the pyloric ring. Whereas tuberculosis shows a tendency to localize at the pyloric end of the stomach, in lymphomas the process tends to be more diffuse.

Pop and Hanganut[22] thought that smooth rounded appearance of the stomach at the site of pyloric obstruction, rather than irregular tapering, was the most characteristic feature in the differential diagnosis between gastric tuberculosis and carcinoma. However, in 1 of our cases (Case II, Fig. 3*A*) we found irregular tapering, a prominent roentgenologic feature.

### SUMMARY

1. Tuberculosis of the stomach and duodenum is a rare disease entity even in patients with pulmonary or intestinal tuberculosis.

2. Four cases of tuberculosis, primarily localized at the pyloro-duodenal area, are reported.

3. The clinical and detailed roentgenologic manifestations of these cases are discussed. The observed roentgenologic features include retrograde involvement of the pylorus from an infiltrative duodenal tuberculosis, a penetrating narrow tubular ulcer tract, chronic saccular ulcer crater, and the various findings of hypertrophic tuberculosis of the pyloro-duodenal area.

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# GASTRIC ULCERS SIMULATING INTRAMURAL, EXTRAMUCOSAL TUMORS

## REPORT OF THREE CASES

*By* JOSEPH F. LINSMAN, M.D.*
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THE well established roentgenographic criteria for diagnosing intramural, extramucosal gastric tumors may occasionally be misleading. While certain roentgen features are more or less typical of these lesions, other possibilities in the differential diagnosis should not be overlooked. Usually, the following group of roentgenographic findings are considered characteristic:[2,3,4] (1) a clearly circumscribed filling defect with a smooth outline which, if located on either curvature, often assumes a crescentic shape; (2) an abrupt transition between the lesion and the uninvolved portion of the stomach wall, usually forming an acute angle at the junction; (3) normal mucosa surrounding the lesion; (4) normal pliability of the gastric walls with normal peristalsis; and (5) barium trapped within a central umbilication or ulcer crater. In the 3 cases reported here, preoperative roentgenographic studies showed most of these characteristics, yet subsequent histopathologic examination revealed benign gastric ulcers.

### REPORT OF CASES

CASE I. A.P., male, age 55, gave a long history of excessive belching and discomfort in the left subcostal region brought on by eating or drinking. Intermittent headaches and nausea were relieved by induced vomiting. There were occasional episodes of bright red rectal bleeding but no melena, constipation or hematemesis. The patient smoked about 40 cigarettes a day. Roentgenograms of the upper gastrointestinal tract 10 years previously were normal. Physical and laboratory findings were noncontributory.

Roentgen examination (Fig. 1) demonstrated a moderately large crescentic contour defect in the prepyloric region of the lesser curvature of the stomach. A small collection of barium was present in the center of this defect. The lesion was located in the immediate prepyloric region and extended 3 cm. proximally from the pylorus. The margin formed an acute angle with the adjacent normal mucosa. Peristaltic waves passed freely through the involved zone.



Fig. 1. Case 1. Spot compression roentgenogram demonstrates a moderately large prepyloric, lesser curvature, crescentic contour defect with a small, central collection of barium. The proximal border between the normal mucosa and the lesion presents an acute angle. Peristalsis traversed the area of the lesion.

*Preoperative diagnosis:* Intramural, extramucosal tumor of the stomach.

Exploration revealed a firm nodule, 1 cm. × 1 cm. × 2 cm., at the posterosuperior aspect of the lesser curvature about 1 cm. from the pylorus. An ulcer was observed in the center of the nodule. The lesion was excised and pyloroplasty was performed.

Inspection of the gross specimen revealed an ulcer 0.8 cm. across and 0.1 to 0.2 cm. deep. Microscopic examination showed superficial ulceration with submucosal inflammation showing marked eosinophilia. The inflammation extended into the muscularis.

*Pathologic diagnosis:* Benign gastric ulcer.

CASE II. M.F., male, age 70, gave a history of gastric ulcer at age 34. He currently complained of a burning sensation in the epigastrium of

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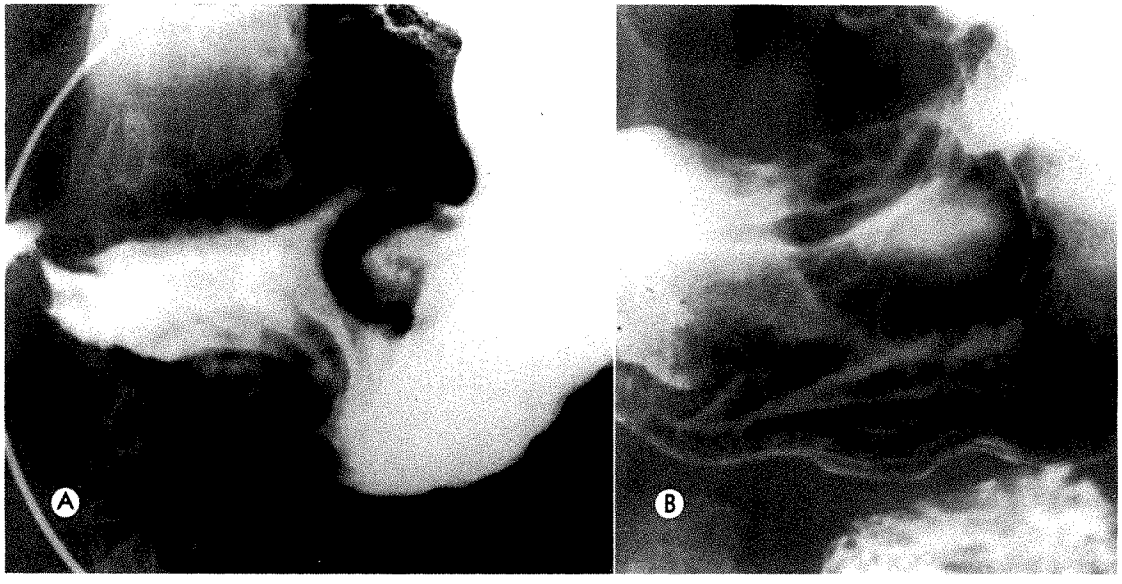



Fig. 2. Case II. The roentgenograms demonstrate a large, circumscribed, rounded, smoothly marginated filling defect in the body of the stomach. A large ovoid collection of barium is present centrally. The mucosa surrounding the defect is normal, and there was normal pliability of the stomach with peristalsis traversing the area of the lesion. (*A*) Prone position with compression. (*B*) Air contrast demonstration of the lesion with the patient supine.

several weeks duration. The pain was not constant and did not radiate. It occurred mainly at night and was severe enough to awaken the patient. The discomfort could be relieved by eating or passing flatus. There was no nausea or vomiting. Physical and laboratory findings were noncontributory.

Roentgen examination (Fig. 2, *A* and *B*) of the upper gastrointestinal tract revealed a large, circumscribed, smoothly rounded filling defect in the body of the stomach. A large ovoid pool of barium collected in its center. The mucosa surrounding the defect appeared intact. The stomach was pliable, and peristalsis was entirely normal.

*Preoperative diagnosis:* Intramural, extramucosal tumor of the stomach.

Surgical exploration revealed a thickened, indurated mass on the posterior wall of the body of the stomach extending toward the lesser curvature. It was situated approximately 8 cm. proximal to the pyloric canal. Subtotal gastrectomy was performed.

Pathologic examination disclosed an ulcer, 2.5 cm. in diameter. The edges were raised and the bed indurated. Microscopic examination revealed typical benign mucosal ulceration with regional antral gastritis. Eosinophils were numerous.

*Pathologic diagnosis:* Benign gastric ulcer.

Case III. I.B., male, age 62, had been hospitalized 6 years earlier with severe melena requiring transfusions. He gave a history of longstanding dyspepsia, especially at night. His symptoms were heartburn and postprandial distress relieved by eating. Roentgenographic study at that time (Fig. 3*A*) revealed a sliding, nonreducible hiatal hernia about 5 cm. in diameter. Coarse folds were demonstrated in the prepyloric antrum together with a 1 cm. rounded filling defect on the lesser curvature. There was no evidence of ulceration or umbilication. Minimal duodenal cap deformity was present.

The bleeding was attributed to the hiatus hernia by the attending physician.

Roentgenograms 2 years later (Fig. 3*B*) were identical to the earlier studies with the additional finding of a tiny collection of barium in the center of the prepyloric filling defect.

After a lapse of 4 more years, the patient was seen for a third time, still voicing the same complaints. Roentgenographic study at that time (Fig. 3*C*) revealed that the diameter of the intramural defect had increased from 1.0 to 2.5 cm. A small, but larger, collection of barium was again demonstrable in the center of the defect.

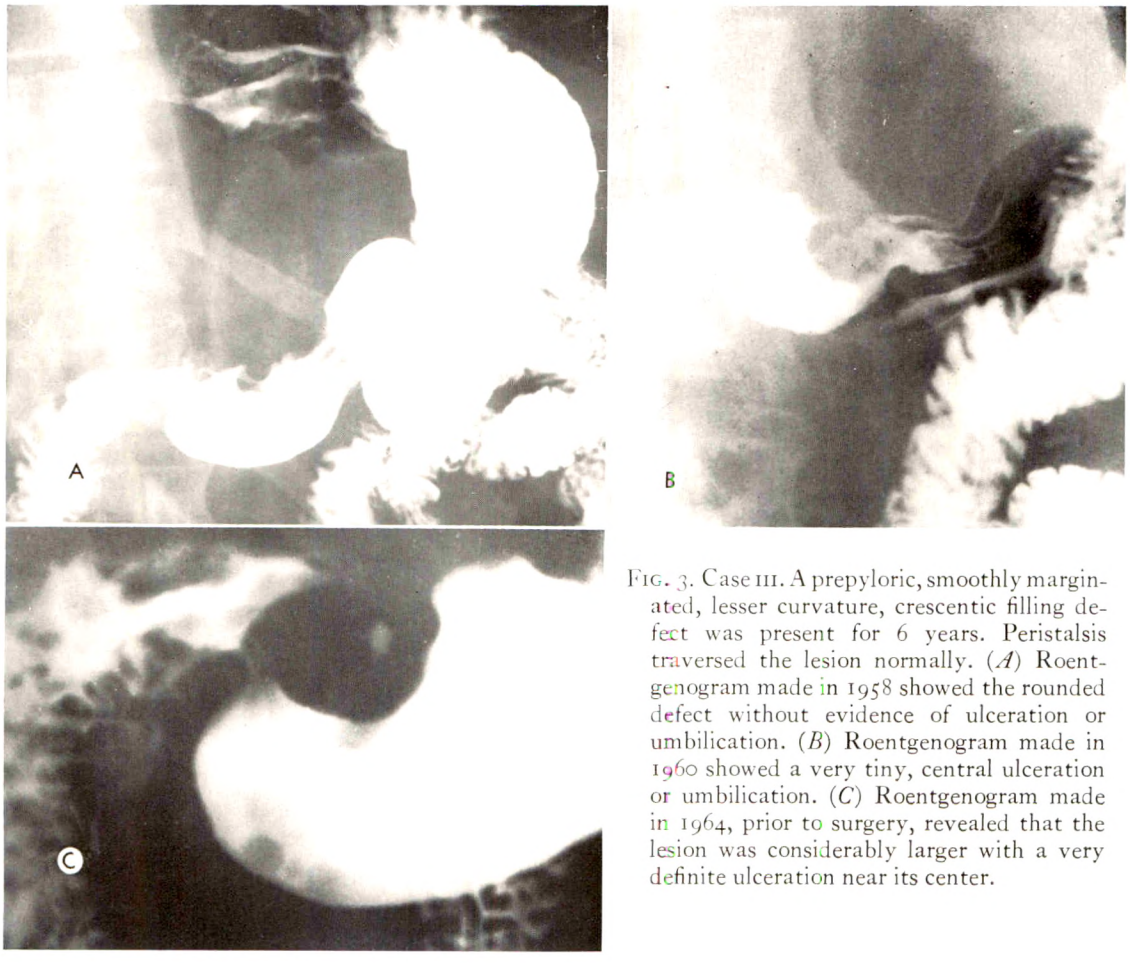



Fig. 3. Case III. A prepyloric, smoothly marginated, lesser curvature, crescentic filling defect was present for 6 years. Peristalsis traversed the lesion normally. (*A*) Roentgenogram made in 1958 showed the rounded defect without evidence of ulceration or umbilication. (*B*) Roentgenogram made in 1960 showed a very tiny, central ulceration or umbilication. (*C*) Roentgenogram made in 1964, prior to surgery, revealed that the lesion was considerably larger with a very definite ulceration near its center.

All other findings were unchanged. Physical and laboratory examinations, as in the first 2 cases, added nothing further.

*Preoperative diagnosis:* Intramural, extramucosal tumor of the stomach.

At surgery, marked thickening of the gastric wall in the prepyloric area was noted, but no definite tumor was palpable. The distal half of the stomach was resected.

Pathologic examination revealed a thickened segment of stomach which included an ill-defined, prepyloric, mound-like elevation in the center of which a discrete 0.3 cm. ulceration filled with exudate was present. In the antrum, the mucosal folds were thickened and focally superficially ulcerated. Microscopic examination confirmed the diagnosis of benign gastric ulcer with regional gastritis, both acute and chronic. Focal superficial erosions and marked mucosal edema were present.

*Pathologic diagnosis:* Benign gastric ulcer.

## DISCUSSION

The roentgenographic and pathologic findings in these 3 patients demonstrate that the long established roentgenographic criteria for intramural, extramucosal tumors are not infallible. Roentgen studies in all 3 patients amply fulfilled the diagnostic criteria, yet pathologically each proved to be a benign gastric ulcer accompanied by marked inflammatory infiltration.

Not only may the roentgenographic appearance be deceiving, but the symptoms, too, can be misleading. Ulcerating, intramural, extramucosal tumors often produce distress indistinguishable from ulcer pain. As with gastric ulcers, hemorrhage may also occur.

Lowman and his co-workers[1] reported 3 patients with adenocarcinomas which had

invaded the gastric wall producing an endogastric mass. Roentgenographically, these lesions simulated intramural, extramucosal tumors, except that in one instance peristaltic waves did not pass through the lesion. The findings regarding peristalsis were inconclusive in the second case and not noted in the third. Nevertheless, the authors emphasized that the accepted criteria for benign intramural tumors are not singly, or even collectively, pathognomonic and do not permit differentiation from certain epithelial neoplasms of the stomach. In the light of the 3 examples presented here, this is evidently true also for benign gastric ulcers.

Under these circumstances, perhaps a clinical test might prove useful in differentiating these conditions. For example, trial on a rigid peptic ulcer diet could prove a simple and useful method of distinguishing between benign gastric ulcer and intramural, extramucosal tumor. If roentgen examination after four weeks on such a regimen were to show regression or disappearance of the ulcer crater, with diminution or disappearance of the wall of inflammation about it, there would be little doubt of the diagnosis.

## SUMMARY AND CONCLUSIONS

1. Three cases in which the established roentgen criteria for the diagnosis of ulcerating intramural, extramucosal tumor of the stomach were satisfied, proved to be benign gastric ulcers.

2. Since benign gastric ulcers may occasionally simulate ulcerating intramural, extramucosal tumors, a limited trial on a rigid peptic ulcer regimen may clarify the diagnosis in some cases.

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# GASTRIC INVOLVEMENT IN CROHN'S DISEASE*

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REPORTS of gastric involvement in regional "enteritis" have been sporadic and few. Six cases are presented in which this diagnosis has been made. Pathologic material was obtained from the stomach in two of these, and more distal areas of disease were demonstrated in all. The literature is reviewed with regard to the roentgenographic findings of the previously reported cases.

## REPORT OF CASES

CASE I. M.F., a 19 year old male, had gastric distress for 5 months prior to his initial visit to our institution. This consisted of food not digesting and pain in the epigastrium after eating. Occasionally he would vomit, but denied diarrhea and melena. He had noted increased fatigability and had lost 14 pounds in weight during the previous year. The patient appeared pale and slender and had general abdominal tenderness. His hemoglobin was 10.2 gm. per cent, white blood cell count 10,000 per cubic millimeter, and a normal differential count. There was 1+ occult blood in the stool.

An upper gastrointestinal examination was performed in which gastric peristalsis was normal. Multiple punctate ulcers involved the gastric antrum, and there was a "cobblestone" appearance of the proximal antral mucosa (Fig. 1, *A*, *B* and *C*). The proximal duodenum and terminal ileum were irregular and narrowed. The intervening portions of small bowel and the colon appeared normal.

At gastroscopy multiple ulcerations were visualized in the antrum, and a biopsy was interpreted as a focal granulomatous reaction consistent with the diagnosis of Crohn's disease. On a follow-up radiographic examination the ulcerations had apparently healed with residual constriction of the gastric antrum.

CASE II. S.I., a 22 year old male, had a 3 year history of anorexia, loose stools, and weight loss. He was hospitalized on several occasions because of vomiting secondary to duodenal disease with pyloric obstruction. A diffuse ulcerative process was also found in his colon which was diagnosed as regional enteritis. The patient was referred to our hospital for management of a gastrocolic fistula which had developed.

On physical examination he was quite emaciated, but without abdominal tenderness or palpable mass lesions. His hemoglobin was 14.5 gm. per cent and white blood cell count 12,400 per cubic millimeter, with a normal differential. The gastrocolic fistula was demonstrated on the colon examination, but not on the antegrade studies. A diffuse ulcerative process involved the distal ileum and proximal colon to the descending portion. A constricting and ulcerative lesion was also present in the gastric antrum extending into the base of the duodenal bulb. The descending portion of the duodenum was dilated (Fig. 2, *A*, *B* and *C*). At gastroscopy, a normal mucosal pattern was seen, but it is doubtful that the antral area was visualized. Gastrojejunostomy, vagotomy, and a transverse colostomy were done. The pathologic material obtained from the stomach at surgery showed an inflammatory process with several poorly defined granulomatous areas, compatible with Crohn's disease.

Postoperatively he rapidly regained his weight. A roentgenographic examination in the fifth postoperative month showed a functioning gastroenterostomy, but no significant change in the morphologic appearance of the stomach.

CASE III. K.G., a 20 year old female, had cramping abdominal pain for 3 years prior to admission. In addition, there was mild intermittent swelling of her ankles, general weakness, and occasional mild bouts of loose stools. She had lost 20 pounds during this period of time. Her physical findings at the time af admission were those of a pale, thin, young woman with a mildly distended, nontender abdomen. Her hemoglobin was 11 gm. per cent and her serum albumin 2.2 gm. per cent, respectively. The white blood cell count and differential count were normal.

Roentgenographic examinations of her gas-

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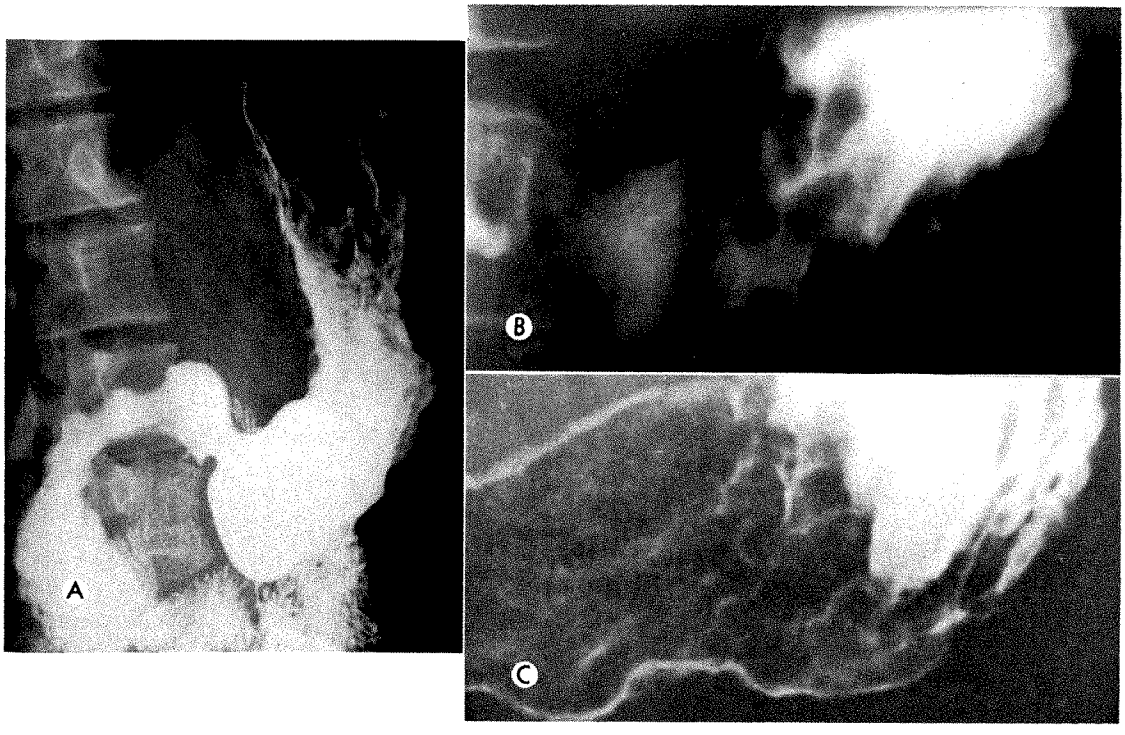



Fig. 1. Case 1. (*A*) Prone-oblique projection. The barium filled antrum is distensible. A fine, serrated margin could be observed on the original roentgenogram. There is a tubular narrowing of the distal bulb and proximal descending duodenum with irregular margins. (*B*) Pressure spot roentgenogram of the proximal antrum. A "cobblestone" mucosal surface pattern is apparent. (*C*) Air contrast spot roentgenogram of the gastric antrum. Multiple, fine punctate ulcers retain the barium contrast. The "cobblestone" appearance can again be seen proximally. An irregular, finely ulcerated terminal ileum was also present. On follow-up examination the gastric ulcers could not be identified, but the antrum was mildly contracted.

trointestinal tract demonstrated extensive ulcerative and stenotic disease extending from the gastric antrum to the ileocecal valve (Fig. 3). The duodenal biopsy was indeterminate. The clinical diagnosis was regional enteritis and protein losing enteropathy.

Case iv. J.T., a 37 year old male, had a history of chronic diarrhea without mucus or blood observed in the stool. Ulcerative lesions were observed at sigmoidoscopy. The patient was thin and had no positive abdominal findings. His hemoglobin was 13.4 gm. per cent; white blood cell count and differential count were normal.

Roentgenographically the patient had a diffuse ulcerative process involving the colon and distal ileum with a skip area of involvement in the mid ileum. In addition, there were stiff rugal folds and multiple pin-point irregularities in the gastric antrum (Fig. 4). A biopsy of the rectum was interpreted as a nonspecific inflammatory change without evidence of lymphoma. The clinical diagnosis was regional enteritis.

Case v. M.C., a 17 year old male had persistent abdominal cramps for 3 years prior to admission. He also had lost 23 pounds during that time. At 11 years of age he had had an appendectomy. On physical examination the patient was alert but poorly nourished. There was general abdominal tenderness without a palpable mass. The sigmoidoscopy was negative. His hemoglobin was 12 gm. per cent, white blood cell count and differential count were normal.

Roentgenographically, the gastric antrum and duodenal bulb were contracted and deformed (Fig. 5). Approximately 20 cm. of distal ileum was fixed and irregular with a pseudodiverticulum at the proximal portion. The clinical diagnosis was regional enteritis.

Case vi. C.A., a 22 year old female, had anal puritus and rectal bleeding 4 years prior to

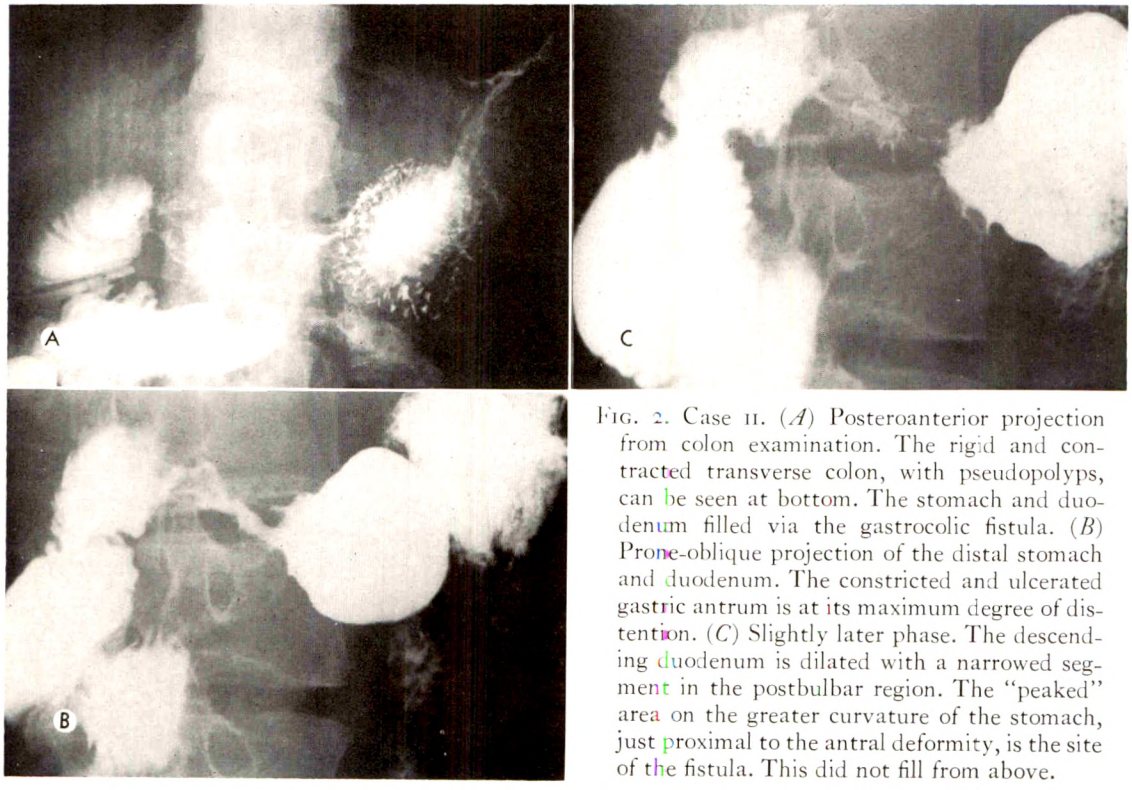



Fig. 2. Case II. (*A*) Posteroanterior projection from colon examination. The rigid and contracted transverse colon, with pseudopolyps, can be seen at bottom. The stomach and duodenum filled via the gastrocolic fistula. (*B*) Prone-oblique projection of the distal stomach and duodenum. The constricted and ulcerated gastric antrum is at its maximum degree of distention. (*C*) Slightly later phase. The descending duodenum is dilated with a narrowed segment in the postbulbar region. The "peaked" area on the greater curvature of the stomach, just proximal to the antral deformity, is the site of the fistula. This did not fill from above.

being seen at our institution. At that time an anterior tissue tag was removed and a hemorrhoidectomy performed. One year later an anal fissure developed. She had increasing bouts of loose stools and an occasional gnawing sensation in her abdomen which did not awaken her from her sleep and was not located in the epigastrium. On physical examination the patient had a mild pallor, but appeared well nourished. Her abdomen was soft and nontender. There was a tender indurated area at the anterior anal margin. Her hemoglobin was 11.6 gm. per cent

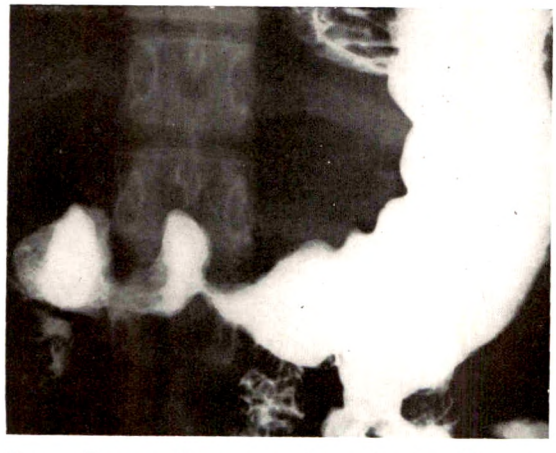


Fig. 3. Case III. Posteroanterior projection. The concentric narrowing of the distal antrum and the deformity of the duodenal bulb persisted. This patient also had extensive small bowel disease with several stenotic areas and rigid, nodular segments.

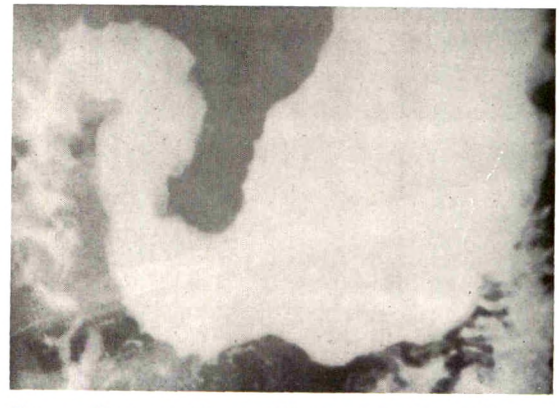


Fig. 4. Case IV. Prone-oblique projection. The antrum is slightly narrowed with irregular margins that suggest multiple superficial ulcerations. Thickened mucosal folds are present in the proximal duodenum. A diffuse ulcerative process involved the distal ileum and colon as well.



FIG. 5. Case v. Posteroanterior projection. The gastric antrum and duodenal bulb are contracted and deformed. Also, a long segment of ileum was fixed, irregular and nodular.

and 10.8 gm. per cent on succeeding days. The white blood cell count was 12,000 per cubic millimeter with a slight shift to the left. An upper gastrointestinal examination demonstrated irregularity and diminished distensibility of the distal antrum and proximal duodenum (Fig. 6). The distal segment of ileum was narrowed, fixed and had an irregular surface.

The perianal lesion was excised and reported as a granulomatous lymphangitis consistent with Crohn's disease.

## DISCUSSION

The roentgenographic findings of the gastric disease in the cases presented may be characterized in the following manner:

(1) Abnormality confined to the antrum of the stomach—all cases
(2) Contiguous disease in the duodenum —all cases
(3) "Cobblestone" mucosal relief; thickened mucosal folds—Case I and IV
(4) Diminished distensibility or contracture—all cases (Case I on follow-up)
(5) Multiple pin point or shallow ulcers —identified, Case I, II and IV

Case II is unique in this group because of the gastrocolic fistula. It is probable that the fistula originated from the colon with secondary development of the gastric lesions. In the remaining cases the disease has apparently developed *de novo* in the antroduodenal region. Despite the duodenal abnormality only 1 (Case II) of these patients had symptoms clinically diagnosed as duodenal peptic disease. Distal small bowel and/or colonic abnormalities with the features attributed to regional enteritis were demonstrated in all.

The roentgenographic findings of the previously reported cases are listed in Table I. This information has been extracted from the corresponding texts and roentgenographic reproductions. Two patterns may be deduced from this material. The first is that of delayed gastric emptying, atony, and dilatation; the second, gastric antral and duodenal morphologic alterations. The former was not a significant finding in our series, but might represent an occurrence during early phases of the disease. The latter is in accord with our observations.

Differential considerations of the roentgenographic manifestations in themselves would include the following: corrosive gastritis, chronic acid peptic disease, antral gastritis, scirrhus carcinoma, eosinophilic



FIG. 6. Case VI. Prone-oblique projection. Concentric constriction of the distal gastric antrum and proximal duodenum is evident with considerable deformity of the duodenal bulb. There was a similar morphology in the terminal ileum.

Table I

| Author | Roentgenographic Appearance of the Stomach | Other Areas of Involvement |
|---|---|---|
| Comfort *et al.*[2] Case II 57 years, male | Hypotonicity, ineffective peristalsis, retained secretions | Duodenum and jejunum |
| Case IV 22 years, male | Atony, delayed emptying and ineffective peristalsis | Duodenum, jejunum, ileum and cecum |
| Case V 26 years, male | Negative | Duodenum |
| Goldgraber *et al.*[5] Case II 18 years, male | Gastric retention and obstruction at the pyloric canal without evidence of an ulcer crater | Ileocecal region |
| Heffernon and Kepkay[6] 48 years, male | Large, hypertrophic, and distorted gastric rugae with dilatation | Small intestine, esophagus |
| Johnson *et al.*[7] 35 years, male | Narrowing, lack of distention, and irregular margins about the gastric antrum | Jejunum and ileum |
| Martin and Carr[8] Case I 23 years, female | Absence of normal peristalsis in the pyloric antrum and cap; deficient mucosal pattern in the pyloric antrum; transition from normal to pathologic mucosa sharp and well defined; irregular margins in area sharp and well defined; no gastric stasis | Duodenum and ileum |
| Case II 24 years, female | Gastric dilatation, retained fluid, diminished gastric peristalsis, and normal gastric emptying | Duodenum and ileum |
| Miller *et al.*[9] 6 years, male (when first seen) | Over a 5 year period: Annular narrowing, distal portion of the gastric antrum; "thought to show gastric ulcer"; thickening of the stomach | Duodenum, small intestine and colon |
| Richman *et al.*[11] Case I 19 years, male | Distended stomach, delayed emptying and deformed bulb. (Antral narrowing noted on reproduction) | Duodenum and ileum |
| Case II 39 years, male | "Peptic ulcer," irregularity of prepyloric region | Duodenum and ileum |
| Case III 10 years, male | Narrow, rigid gastric antrum | Jejunum and ileum |
| Ross[12] 21 years, female | Initially, stomach interpreted as negative except for delayed gastric emptying, (cicatrizing disease and mucosal ulceration at surgery over one year later; no roentgenographic examination in the interval) | Ileum |
| Self[13] 35 years, female | Gastric capacity reduced, mucosal outline flattened and atrophic, constant incisura; consistent filling defect of the gastric wall of the pyloric antrum with poor peristalsis (diagnosed as linitis plastica) | Duodenum |

infiltration, benign lymphoid hyperplasia,[10] lymphoma, and other granulomatous diseases. The presence of a similar process in the duodenum and more distal intestine would exclude some of these possibilities. The observation of pulmonary disease and lymphadenopathy would be of further limiting value. Eosinophilic infiltration can involve both stomach and small bowel but is associated with peripheral eosinophilia and an allergic history.[1,4] Lymphoma and other granulomatous diseases confined to the gastrointestinal tract may be indistinguishable.

Terms such as nonspecific or focal granulomatous inflammation have been used in the pathologic description of this entity. There are several apparently unrelated conditions in addition to regional enteritis which may also have this appearance. Sarcoid, fungus infections, reactions to minerals and lipids, and Hodgkin's disease are among them.[5] When the lesions are confined to the stomach, they are considered by some to represent a distinct clinical-pathologic entity.[3]

The clinical diagnosis of regional enteritis is justified by a combination of clinical data with a characteristic roentgenographic appearance and, if feasible, pathologic support. The radiologist's role is to supply one of the diagnostic parameters and determine the longitudinal extent of the disease, its phase of involvement (ulcerative *vs.* stenotic), and evidence of complications (fistulae, obstruction). A thorough roentgenographic examination of the entire gastrointestinal tract is indicated when the disease is suspected by the clinician or compatible segmental lesions are detected at any level.

SUMMARY

Six cases of Crohn's disease with gastric involvement are presented. The roentgenographic findings are correlated with the previously reported cases. In all instances, additional segments of disease were demonstrated distally.

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# INTRAMURAL GASTRIC EMPHYSEMA*

*By* WILLIAM B. SEAMAN *and* RICHARD J. FLEMING†
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THE presence of gas within the wall of the stomach is not an entity *per se* but a sign of an underlying abnormality such as infection, mucosal ischemia, increased intraluminal pressure, or severe vomiting. Multiple terms have been applied to this phenomenon including intramural or interstitial gastric emphysema, emphysematous gastritis, gastric pneumatosis, nonbacterial gastric emphysema and pneumatosis cystoides. Its clinical importance depends upon the underlying pathology which ranges from phlegmonous gastritis and necrotizing enterocolitis, diseases of high mortality, to a sequela of gastroscopy, a condition of little importance.

Four cases are reported to illustrate its roentgenolgic aspects and clinical significance.

## REPORT OF CASES

CASE I. E.F., a 49 year old female with rheumatic heart disease and a history of multiple episodes of congestive heart failure had had several episodes of gastric dilatation and hypokalemic acidosis. The current hospital admission was preceded by 10 days of nausea and vomiting. She was again found to be hypokalemic.

Abdominal roentgenograms revealed a markedly distended stomach lying beneath an elevated left diaphragm. Within the gastric wall were two parallel lines of radiolucency which were interpreted as submucosal and subserosal gas (Fig. 1, 2 and 3). This intramural gas had completely disappeared 3 days later when a roentgenographic examination of the upper gastrointestinal tract was done. The latter was normal except for the presence of an organoaxial gastric volvulus and a diaphragmatic hernia. Partial gastrectomy and repair of the hernia were performed 1 month later. Microscopic examination of the resected antrum revealed a normal stomach.

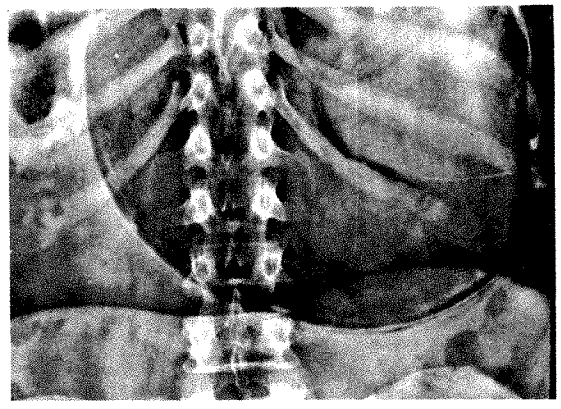


FIG. 1. Case I. Forty-nine year old female with gastric dilatation associated with a gastric volvulus. Note double radiolucent lines along greater curvature aspect due to submucosal and subserosal gas.

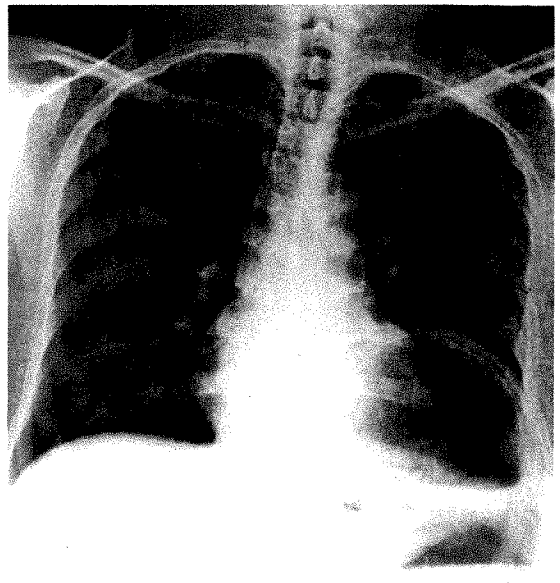


FIG. 2. Case I. Frontal roentgenogram of the chest illustrating elevated diaphragm on the left. Radiolucent intramural gastric gas is visible beneath the diaphragm.

CASE II. W.H., a 68 year old diabetic female, was admitted to the hospital because of a 3 day history of nausea and vomiting of coffee-ground

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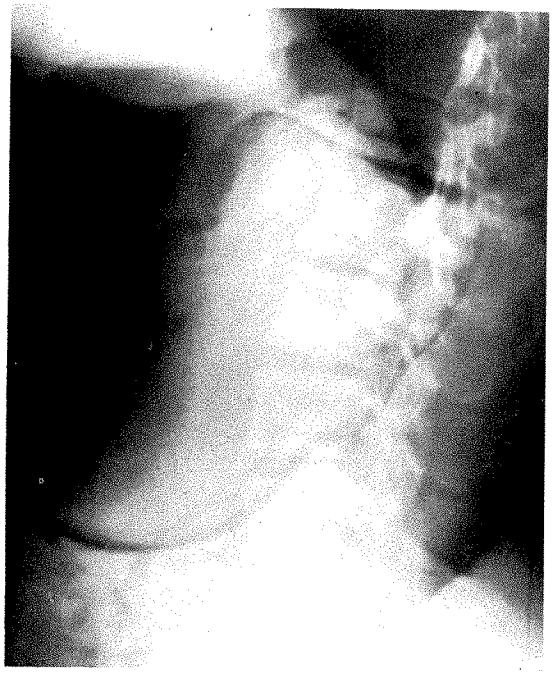


FIG. 3. Case 1. Lateral roentgenogram of the distended stomach.

material. Roentgenograms of the chest and abdomen on the day of admission revealed evidence of small intestinal obstruction. A positive guaiac test was obtained from the vomitus. Gastric intubation was carried out and a follow-up abdominal roentgenogram 24 hours after admission showed evidence of intramural gas in the fundus of the stomach and the proximal portion of the pars media (Fig. 4 and 5). Two days later the intramural gas had completely disappeared. Death resulting from a pelvic infection occurred 6 weeks later. At autopsy, the stomach was normal by gross and microscopic examination.

CASE III.* K.C., a premature male infant weighing 3 lb. 12 oz., was the result of a normal spontaneous vertex delivery. For the first 8 days all feedings and bowel movements were considered normal. Over a period of 2 days, the infant developed abdominal distention and some fecal and blood stained vomiting. Roentgenograms of the abdomen obtained at this time showed evidence of intramural air lining the entire contour of the stomach (Fig. 6). Abnormal contours of the small intestinal gas pattern were also noted. Death occurred 1 week later. At autopsy extensive necrotizing enteritis involving almost the entire gastrointestinal tract was noted.

CASE IV.† C.V. was a 9 day old premature infant with abdominal distention and jaundice since the third day of life. Abdominal paracentesis yielded 250 cc. of clear fluid from which no bacterial organisms could be cultured.

Six days later there was an episode of rectal bleeding, the liver was enlarging and the ascites continued to increase. *Aerobacter aerogenes* was cultured from the ascitic fluid. Abdominal roentgenograms taken at that time disclosed gas within the gastric wall and in the portal venous system. (Fig. 7, *A* and *B*). Death occurred on the seventh hospital day.

At autopsy the gastric wall was crepitant and gas-filled blebs were observed beneath areas of intact mucosa. The gastric mucosa in addition to being diffusely reddened and hemor-

* Case III supplied by Dr. Robert Hoffman, St. Petersburg, Florida.

† Case IV included in the series reported by Berdon *et al.*[2]

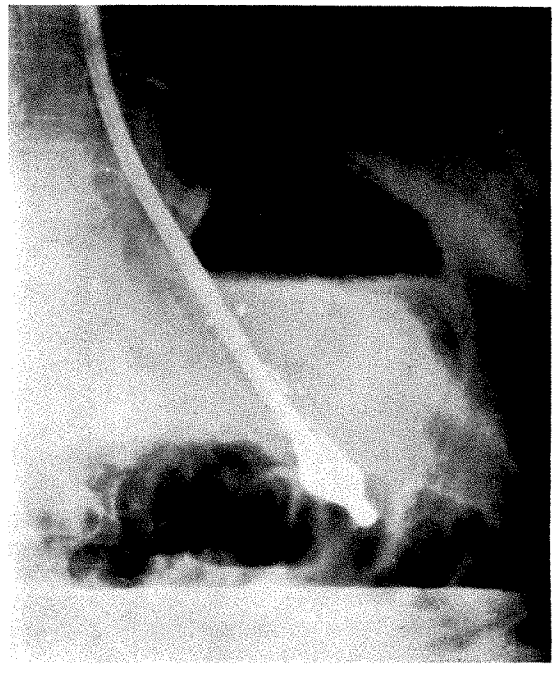


FIG. 4. Case II. Sixty-eight year old diabetic female with a 3 day history of nausea and vomiting of coffee-ground material. Linear streak of intramural gas is distributed over the fundus.

rhagic was ulcerated and necrotic in the fundal portion.

Microscopically, bacteria were noted to be growing on the surface of the stomach as well as in the superficial necrotic tissue and submucosa. Numerous large and medium sized cystic cavities were present in the submucosa.

Infiltration of viscera, lymph nodes and bone marrow by foamy macrophages was discovered compatible with a lipid storage disease. The gastric findings were thought to be compatible with a severe bacterial gastritis.

## DISCUSSION

A review of the medical literature has revealed only 15 examples of intramural gastric emphysema (Table 1). Analysis of these and the cases described above suggests that the pathogensis of the condition is probably related to the trauma of severe vomiting, increased intragastric pressure incident to obstruction, or gastroscopic inflation or impairment of mucosal integrity by ischemia or infection.

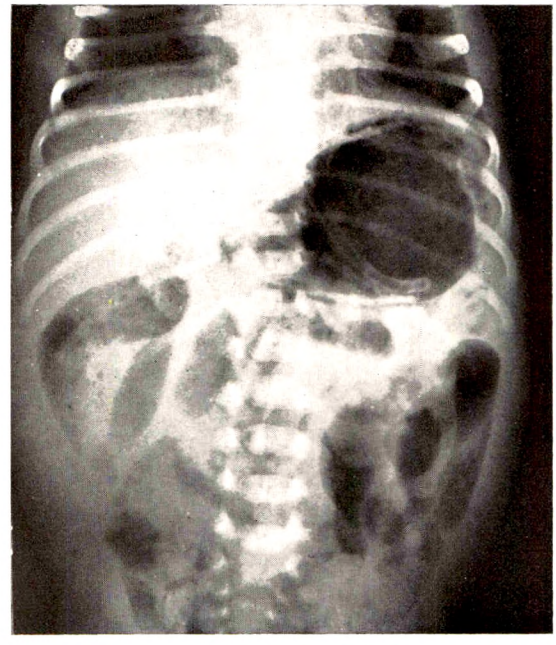


Fig. 6. Case III. Premature male infant with intramural gas outlining gastric contour. Necrotizing gastroenterocolitis found at autopsy.



Fig. 5. Case II. Lateral decubitus roentgenogram illustrating extension of intramural gas down the greater curvature and lack of mobility with change of position.

Myhre and Wilson[15] found 2 examples of gastric emphysema by taking erect roentgenograms of the abdomen of 119 patients 24 hours after gastroscopy. Similar observations have been made by Cancelmo,[3] Fierst *et al.*,[7] and Chamberlain.[4]

In the patients described by Schorr and Marcus,[19] Ward,[20] Druckmann *et al.*,[6] and Lumsden,[14] obstruction at the pylorus or distal antrum was the underlying abnormality.

In the 4 patients described by Plachta and Speer,[16,17] no other gastrointestinal abnormality was present. The condition was detected only at postmortem examination and the main pathologic finding was pulmonary emphysema. These authors postulated that the pathogenesis was the rupture of a pulmonary air bleb into the

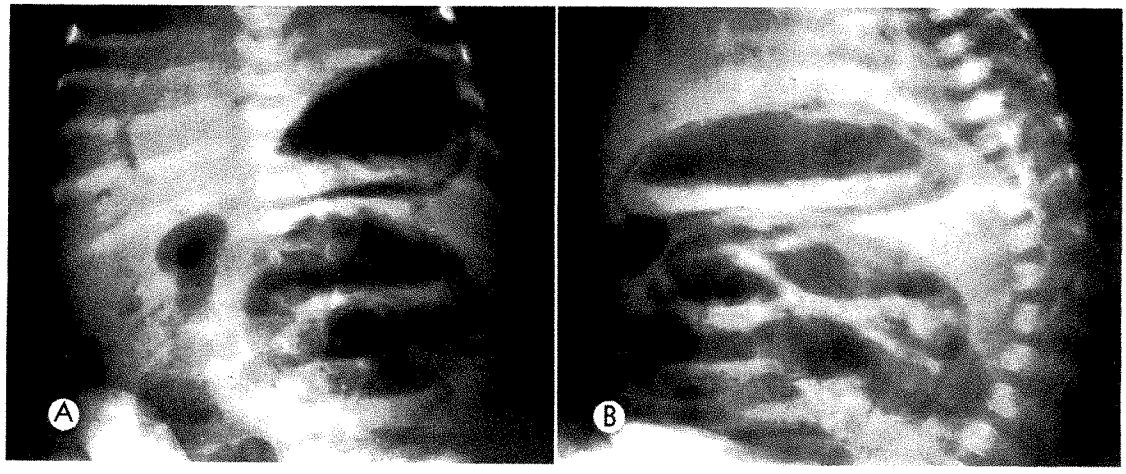



FIG. 7. Case IV. (*A*) Frontal and (*B*) lateral roentgenograms of a 2½ week old premature infant with intramural gastric emphysema and intrahepatic portal vein gas. Severe necrotizing gastroenterocolitis was found at autopsy. (Reproduced with permission of *Radiology*.[2])

para-esophageal areolar tissue with dissection into the gastric wall. A similar hypothesis for intestinal intramural emphysema has been proposed by Keyting *et al.*[12]

In half of the 21 cases of enterocolitis in newborn premature infants reported by Berdon *et al.*[2] there was gross and microscopic evidence of intramural gas in the small intestine. In two of the infants the stomach exhibited a similar phenomenon. Since both mucosal necrosis and gas forming bacteria were present in these infants, the intramural gas could be of either bacterial or intraluminal origin. A similar explanation is possible for the patient re-

TABLE I

INTRAMURAL GASTRIC EMPHYSEMA—REVIEW OF THE LITERATURE

| Author | Associated Disease | Comment |
|---|---|---|
| 1. Ward[20] | Annular cancer obstructing antrum | |
| 2. Lumsden[14] | Pyloric obstruction due to cancer | |
| 3. Kay-Butler[11] | Cancer of pancreas obstrucing duodenum. Stomach normal | Five days of vomiting |
| 4. Cancelmo[3] | Esophageal cancer | Severe vomiting |
| 5. Chamberlain[4] | | Gastroscopy |
| 6. Fierst *et al.*[7] | | Gastroscopy |
| 7. Dassel[5] | Infarction | |
| 8. Myhre and Wilson[15] | | Gastroscopy |
| 9. Myhre and Wilson[15] | | Gastroscopy |
| 10. Schorr and Marcus[19] | Obstructed duodenum | |
| 11. Druckmann *et al.*[6] | Pyloric obstruction | |
| 12. Plachta and Speer[16] | | Pulmonary emphysema |
| 13. Plachta and Speer[16] | | Pulmonary emphysema |
| 14. Plachta and Speer[16] | | Pulmonary emphysema |
| 15. Plachta and Speer[16] | | Pulmonary emphysema |
| 16. Berdon *et al.*[2] | Necrotizing enterocolitis | |
| 17. Seaman and Fleming* | Gastric obstruction due to gastric volvulus | |
| 18. Seaman and Fleming* | None | Persistent vomiting |
| 19. Seaman and Fleming* | Necrotizing enterocolitis | |

* This report.

ported by Dassel[5] where there was mucosal necrosis secondary to ischemia. Rigler and Pogue[18] described small intestinal intramural gas in 13 of 14 patients with intestinal necrosis secondary to infarction.

Emphysematous gastritis has been used to designate a special variety of severe phlegmonous gastritis, a term coined by Fraenkel in 1889 for a gastric infection due to gas forming organisms. This highly fatal disease is characterized by an explosive onset and a fulminating course, similar to the acute perforation of an abdominal viscus. It is fortunately rare, since according to Gonzalez *et al.*,[9] of 360 cases of phlegmonous gastritis reported in the medical literature only 5 were of the emphysematous variety. This clinical picture aids in its differentiation from other cases of intramural gastric emphysema. In 3 reported cases it followed ingestion of hydrochloric acid.[8,10,21]

Since the presence of gas within the wall of the gastrointestinal tract is merely a symptom of an underlying disease process and not a disease entity of itself, the term pneumatosis cystoides should be discarded. As described in older literature, the stomach was rarely involved. Only 4 examples of gastric involvement occurred in 213 cases of pneumatosis cystoides reviewed by Koss.[13] In one of these, reported by Baumann Schenker, the underlying disturbance was chronic small bowel obstruction in a patient with a gastrojejunostomy, thus suggesting increased intraluminal pressure as the etiologic factor.

## SUMMARY

Four examples of gas occurring within the walls of the stomach are presented. Analysis of these patients plus those reported in the medical literature suggests that the pathogenesis of this phenomenon is related to increased intragastric pressure incident to obstruction or gastroscopic inflation, the trauma of severe vomiting, or impairment of mucosal integrity by ischemia or infection. The gas is probably of intraluminal origin although in some instances it may also come from gas forming bacteria. The clinical significance of this finding depends on the nature of the underlying abnormality.

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VOL. 101, NO. 2

# ROENTGENOLOGIC AND PATHOLOGIC ASPECTS OF SUPERFICIAL SPREADING CARCINOMA OF THE STOMACH*

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ONE of the most intriguing tumors of the intestinal tract is the epithelial neoplasm which largely limits its growth to the gastric mucosa. Since 1908, when Verse[15] first discussed mucosal carcinoma of the stomach, many authors, including Ewing,[2] Mallory,[11] and Konjetzny,[10] have documented its existence and commented upon its many unique facets. Gutmann, Bertrand, and Péristiany[5] in their book titled "Le cancer de l'estomac au début," presented the earliest detailed analysis of mucosal carcinoma with its roentgenographic appearance.

The precise histologic definition of the lesion varies somewhat with different authors (Table 1). It is important to emphasize that the term superficial cancer does not refer to carcinoma *in situ* in which the malignant cells do not extend beneath the basement membrane. Mucosal carcinoma is not confined by the basement membrane; it extends into the tunica propria but does not extend beyond the muscularis mucosae, the anatomic boundary of the mucosa.

"Cancer au début," as described by Albot and Toulet,[1] is a small neoplasm which has not invaded the gastric wall but does exhibit limited invasion of the submucosa. This distinction may be somewhat artificial since limited invasion of the submucosa cannot be excluded without serial sections of the entire neoplasm as Mason[12] has demonstrated using the Swiss roll technique of microscopic examination.

The most liberal anatomic interpretation of this entity is found in the superficial spreading carcinoma of Stout.[14] In this entity, according to Golden and Stout,[4] the malignant disease usually penetrates through the muscularis mucosae and in some instances may penetrate between the muscle bundles of the tunica muscularis to reach the serosa, but does not destroy the muscle. The latter may be associated with lymph node metastases. Areas of superficial carcinoma are commonly seen in association with invasive cancer. This type is not included in this discussion.

We have analyzed a group of 27 patients, collected since the original report of Golden and Stout.[4] These tumors were limited to the mucosa except for occasional microscopic areas of penetration through the muscularis mucosae, thus conforming in general both to the mucosal cancer of Albot and Toulet[1] and the "cancer au début" of Gutmann, Bertrand and Péristiany.

## PROGNOSIS

The practical importance of distinguishing this entity is justified by its excellent prognosis, in sharp contrast to the dismal outlook for the other invasive gastric cancers. Friesen *et al.*[3] summarized the Mayo Clinic experience with superficial carcinoma of the stomach and reported a 5 year survival of 93 per cent, with 75 per cent at 10 years. Their group consisted of 65 cases accumulated between 1930 and 1954. Mason[12] studied 15 patients who were followed from 2½ to 10 years and discovered only one death due to disease during this period. Inokuchi *et al.*[7] reported 31 per cent 5 year survival of 81 patients with early infiltrative carcinoma of the stomach. Jutras[8] mentioned that of 26 cases of 5 year survival of gastric cancer studied at the Hôtel Dieu de Montréal, 14 had mucosal carcinomas.

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TABLE I

SUPERFICIAL CARCINOMA OF THE STOMACH

| | |
|---|---|
| Carcinoma *in situ* | Limited to epithelium and does not extend beyond basement membrane |
| Mucosal cancer (Albot and Toulet[1]) | Invasion of tunica propria but does extend beyond muscularis mucosae |
| Cancer au début (Gutmann, Bertrand, and Péristiany[6]) | Exhibits limited invasion of submucosa |
| Superficial spreading carcinoma (Golden and Stout[4]) | Invades submucosa and may penetrate into tunica muscularis but does not destroy muscle |

Of our 27 cases, 18 were available for analysis at 5 years. Twelve were alive and well, a 5 year survival of 66 per cent (Table II). Of the remaining 6, one was lost to follow-up, one committed suicide, one died of breast cancer, and another died 2 days following surgery. Only two deaths were directly attributable to gastric cancer. Of the remaining group of 9 patients, all are alive and well at varying periods of time less than 5 years.

Complete follow-up records were obtained on 17 patients from the original series of Golden and Stout.[4] The pathology of these was reviewed and in 13 it was found to satisfy the criteria mentioned above. Nine of this group survived 5 years (69 per cent) and 6 are at 10 years (46 per cent). Combining both groups the 5 year survival is 21 of 31 patients (69 per cent).

Lymph node metastases in 5 of our patients did not seem to adversely affect their survival as 3 of this group are 5 year survivors and 2 are alive and well for periods less than 5 years. One of the 5 year survivors died in his sixth year with generalized skeletal and pulmonary metastases.

TABLE II

CURRENT SERIES

| | | |
|---|---|---|
| Total No. of Cases | | 27 |
| No. Available for 5 Year Follow-up | | 18 |
| Five Year Survival | (66%) | 12 |
| *Cause of Death* | | |
| Postoperative —1 | | |
| Suicide —1 | | |
| Breast cancer —1 | | |
| Lost to follow-up—1 | | |
| Gastric cancer —2 | | |
| Alive and well less than 5 years (no deaths) | | 9 |

## CLINICAL FEATURES

The presenting symptoms and subsequent course of this group of patients generally was not characteristic. The age of presentation in both Friesen *et al.*[3] and Mason's[12] groups was 53 years. The age of our series ranged from 42 to 73 years with an average of 58 years, which is somewhat younger than the usual age of presentation of infiltrative cancer of the stomach. Symptoms were usually those of long-standing peptic ulcer disease, often associated with a recent change. Moderate gastric retention occurred in 2 patients and was severe in one other. Six patients presented with a hemoglobin of less than 11 gm. per cent. Eight patients of our current series were examined gastroscopically and in 3 a diagnosis of malignancy was entertained. Of the 8 gastroscoped patients in the Friesen *et al.*[3] series, the question of cancer was raised only once.

## ROENTGENOGRAPHIC FEATURES

Roentgenographic examinations were available for review in 23 instances including 6 cine-roentgenographic studies. In 1 patient, in whom a partial gastrectomy was performed for a chronic duodenal ulcer, the superficial gastric carcinoma was an incidental finding. His roentgenograms, made elsewhere, had been interpreted as normal and no abnormalities were detected on gross examination of the stomach.

The roentgenographic features are listed in Table III. An ulcer crater was detected in 17 patients, of which 4 were interpreted in retrospect as benign (Fig. 1), 9 malignant and 4 indeterminate. A Carman's sign was present in 5 of the malignant ulcers (Fig. 2). The occurrence of partial healing in 2 patients led to the initial interpretation of the ulcer as benign (Fig. 3, *A–D*). Not until the crater had returned to its original size was its malignant character recognized.

There was evidence of a mass lesion in 8 patients, including the 5 patients exhibiting a Carman's sign. Enlarged, tortuous rugae were a prominent feature on the roentgenograms in 4 patients (Fig. 4), and reverted to normal in 1 patient (Fig. 3, *A–D*), suggesting that the enlargement was inflammatory and not of neoplastic origin. Microscopic examination supported this concept since only submucosal edema and vascular congestion were present to explain the rugal thickening (Fig. 7, *A–C*).

A deformity of the gastric antrum occurred 6 times; in 2 patients it was associated with an ulcer, in another multiple polypoid nodules were seen, while in 3 it constituted the only gastric abnormality (Fig. 5; and 9).

Folds radiating towards an ulcer were observed in 2 patients. However, in both, the folds did not reach the margin of the crater but merged into the elevated area surrounding the niche producing the appearance of a mass (Fig. 5; and 12). A local segment of rigidity was observed only in 4 instances (Fig. 10, *A–C*). In most instances when the character of the peristaltic activity was commented upon, it was said to be normal.

Gutmann, Bertrand and Péristiany,[5] in their monograph, divide mucosal cancer into two main groups of infiltrating and ulcerating forms and describe several sub

Table III

SPECIFIC ROENTGENOGRAPHIC FEATURES

| | |
|---|---|
| 1. Visible ulcer crater | 17 |
| 2. Mass present | 8 |
| Carman's sign | 5 |
| 3. Antral deformity | 6 |
| associated with ulcer | 2 |
| without crater | 4 |
| 4. Grossly enlarged rugal folds | 4 |
| radiating folds | 2 |

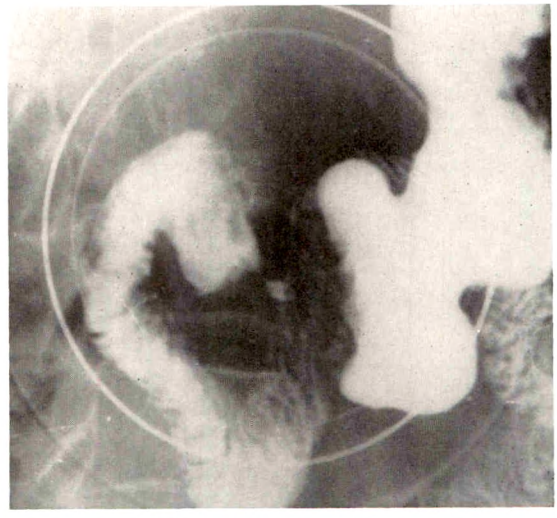


Fig. 1. Superficial gastric cancer having the appearance of a benign antral ulcer.

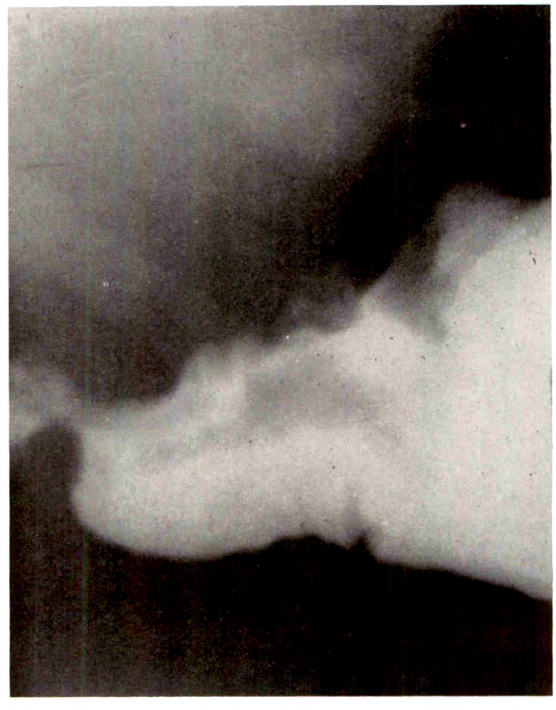


Fig. 2. Large ulcerated antral mass exhibiting typical Carman's sign.

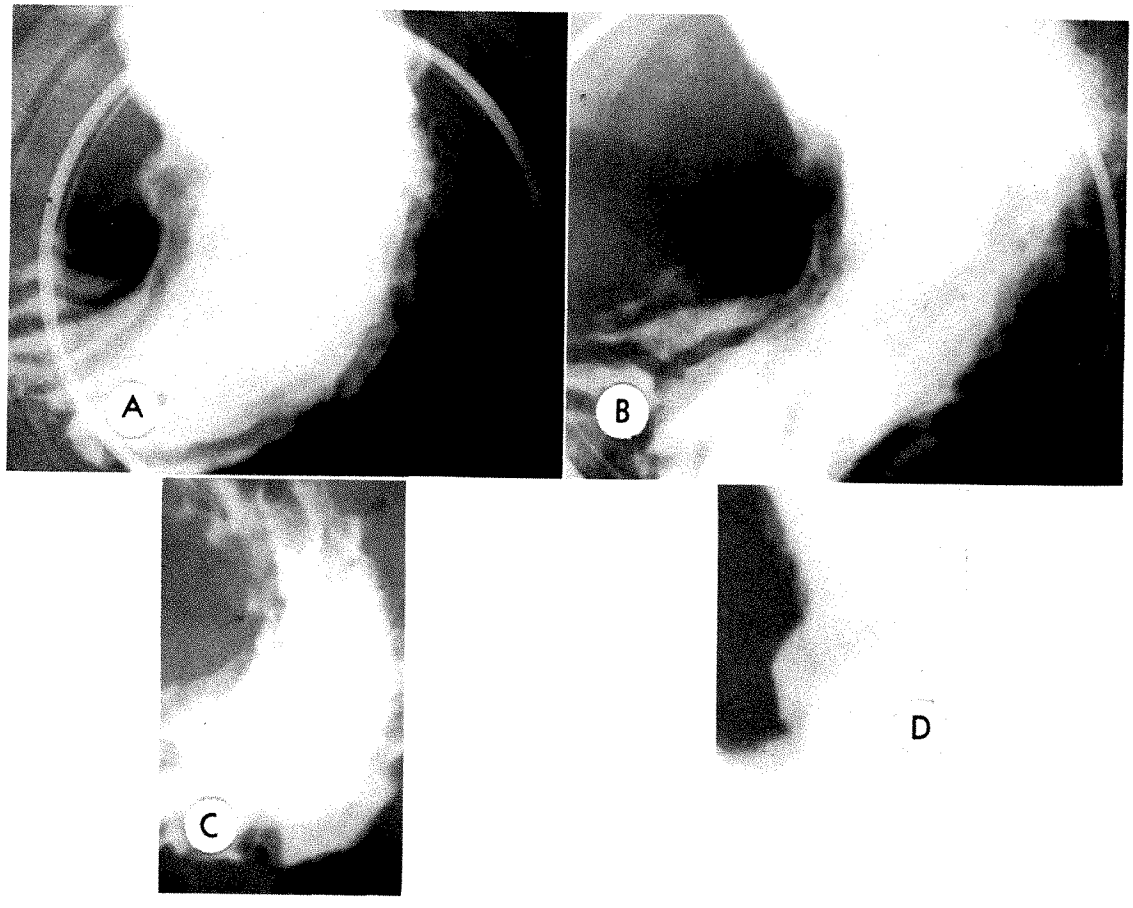



FIG. 3. (*A*) Pars media lesser curvature ulcer. (*B*) Marked regression of crater 6 weeks later. Rugae in the vicinity of the crater are still moderately thickened. (*C*) Five months after *A*. Rugal thickening has disappeared but a small crater persists. (*D*) Nine months after *A*. Crater has enlarged to its original size.

types in each group. Much of our material falls nicely into their groupings.

The earliest form (localized rigidity), produces relatively little change in contour and is best detected during active peristaltic activity when the stiffened segment responds to peristalsis like "a plank on the waves." Later the rigid segment may retract towards the gastric lumen so that it appears recessed or embedded ("aspect encastré"). Tiny erosions or ulcers occurring in the recessed area of rigidity occur in the muco-erosive form, which is felt to represent an intermediate phase between the infiltrating and ulcerating types. The patient illustrated in Figure 8, *A–D* exhibited all 3 of the above types, finally developing a small triangular niche with a retracted rigid segment corresponding to Gutmann's "niche encastré."

When located in the antrum the process tends to produce a narrowed antrum by circumferential growth. At times this may produce a scalloped appearance of the greater curvature of the distal antrum, distinguishable from contraction waves only by its constancy. This is the "forme nodulée" of Gutmann and is well illustrated in one of our cases where it was on every one of many roentgenograms of the antrum (Fig. 9).

The classical appearance of the ulcerating type of mucosal carcinoma is the "niche en plateau" of Gutmann. This ulcer usually straddles the lesser curvature of the antrum and appears as a segmental

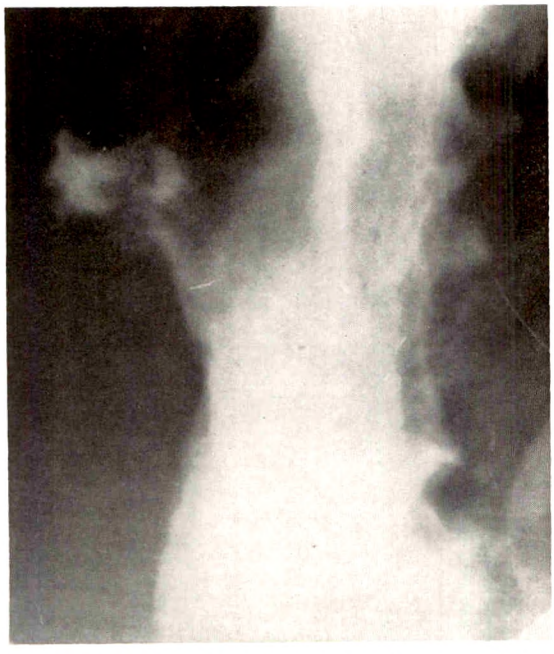


Fig. 4. Marked thickened rugae covering an extensive area around the ulcer crater.

area of stiffening, with a small indentation proximally and distally due to the elevated margins of the crater (Fig. 11).

Six of our patients had one or more cine-fluoroscopic examinations which on review did not help toward further identifying the lesion. In fact, in 1 patient the diagnosis was changed from a malignant to a benign process after observing normal peristalsis and antral flexibility on the cine examination. It is felt by Jutras[8] that cine techniques have increased the radiologist's ability to diagnose superficial spreading carcinoma.

PATHOLOGY

We have screened our current series to include only those cases of pure superficial

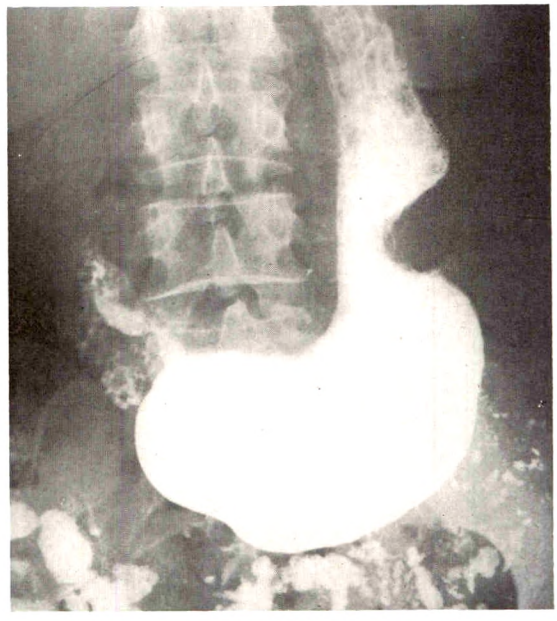


Fig. 5. Nonulcerating type of superficial cancer. Annular deformity of the antrum and mild gastric retention were the only roentgenographic abnormalities. A large area of superficial carcinoma measuring 6 cm. in diameter with a tiny (7 mm.) shallow central ulcer was found on the resected specimen.



Fig. 6. Small ulcer on lesser curvature surrounded by radiolucent rim. Note that radiating folds do not extend to the margin of the ulcer but blend into the elevated mucosa at the ulcer rim.

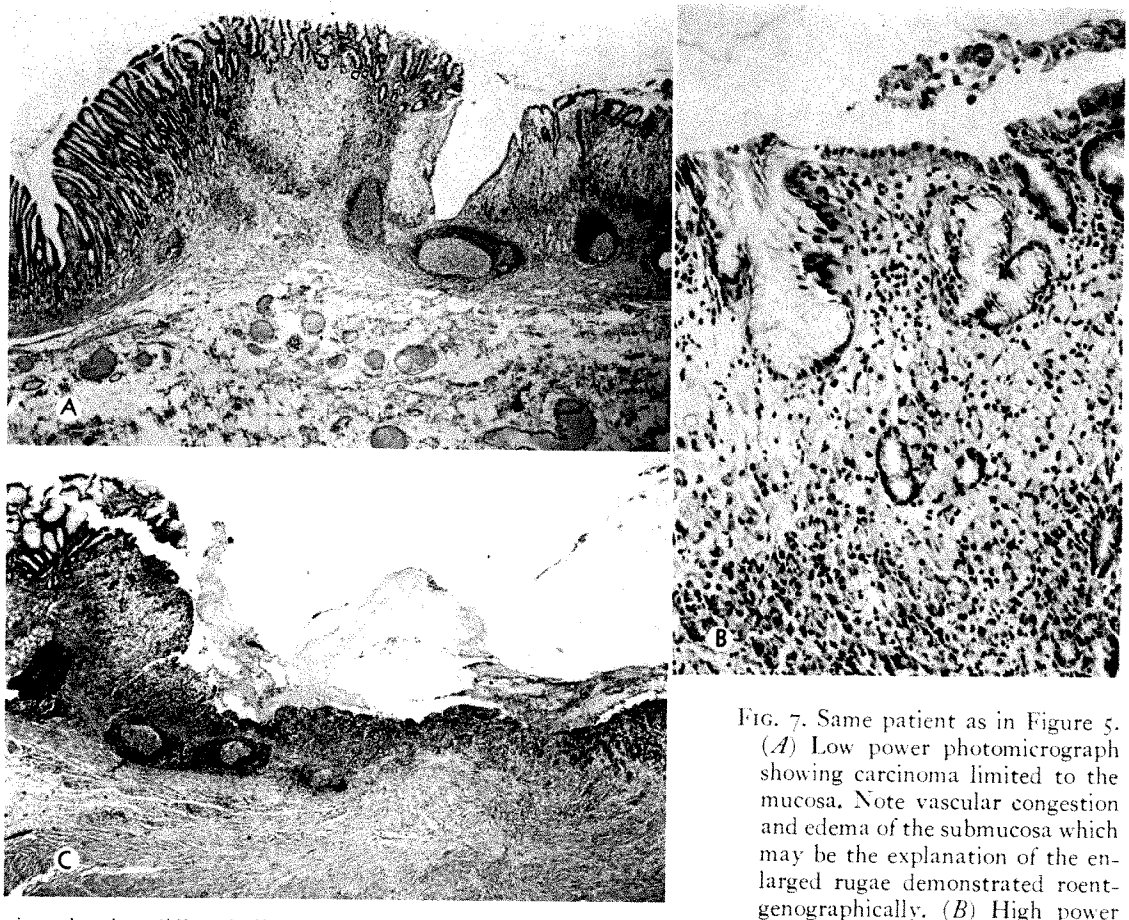



FIG. 7. Same patient as in Figure 5. (*A*) Low power photomicrograph showing carcinoma limited to the mucosa. Note vascular congestion and edema of the submucosa which may be the explanation of the enlarged rugae demonstrated roentgenographically. (*B*) High power view showing diffuse infiltration of the tunica propria with signet ring cells. (*C*) Section through the ulcer shows neoplastic cells in the ulcer base.

spreading carcinoma. It was not infrequent to discover foci of mucosal carcinoma adjacent to an obviously infiltrative lesion. For this reason we excluded 4 cases from the original Golden-Stout series[4] for inclusion in our current review.

The gross appearance of superficial spreading carcinoma assumes two basic patterns: (1) a nonulcerated type and (2) an ulcerated form. The nonulcerated lesion consists of an irregular zone of thinned mucous membrane in which the normal epithelium is largely replaced by neoplastic cells. There may be numerous, irregular pebbly elevations or superficial erosions. The involved mucosa, being atrophic and devoid of glandular elements, is thinner than the surrounding normal mucosa and may give the appearance of a depression roentgenologically when viewed in profile. Albot and Toulet[1] emphasized the neighboring nonmalignant histologic changes of submucosal sclerosis, edema, and vascular congestion which often make the lesion appear larger and more ominous to the radiologist.[4] The surrounding mucosal folds are often enlarged for the same reason.

The ulcerated form may grossly resemble a benign peptic ulcer. On histologic examination nests of malignant cells at the borders of the crater are found.

It is difficult to determine if the entire cancer arises from a single source or whether it is multifocal. The "islands" of tumor cells may actually be tongues ex-

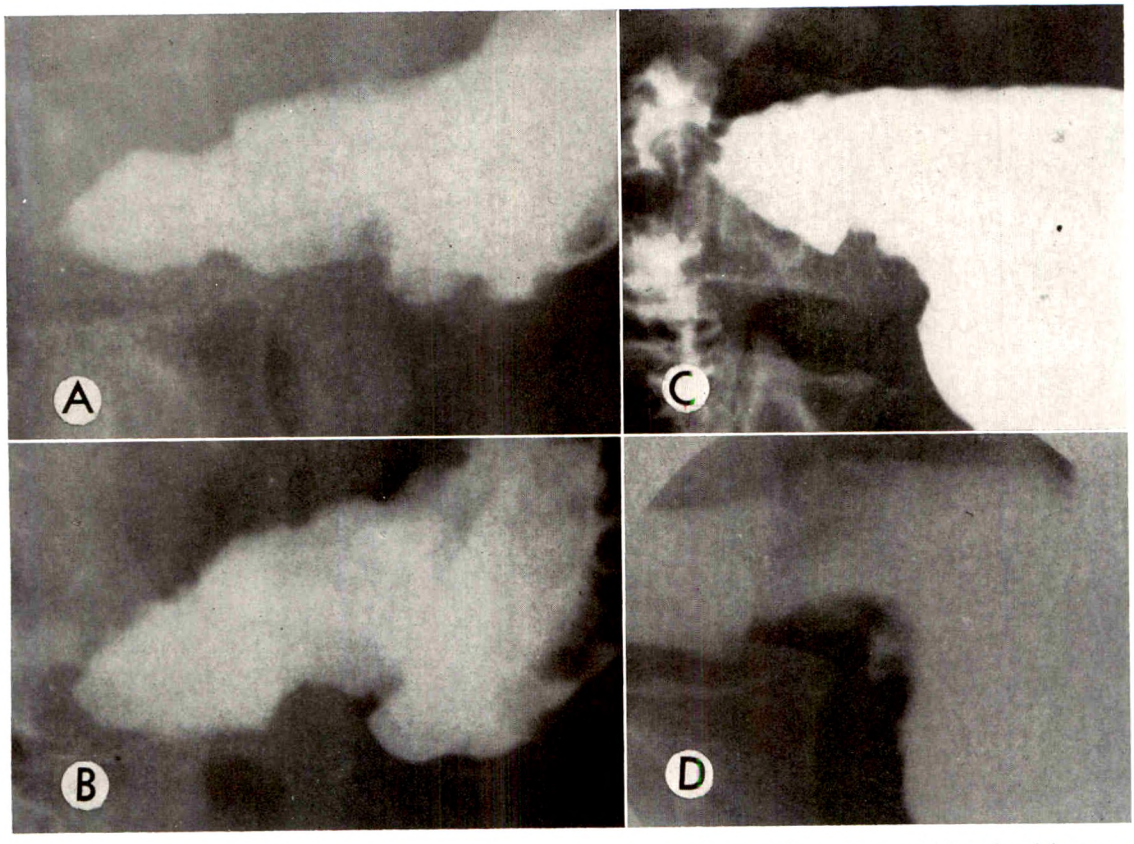



Fig. 8. Progressive stages in the development of mucosal cancer as observed in a 49 year old female with recent onset of a peptic ulcer history. (*A*) Small area of segmental stiffening on the greater curvature of the antrum. (*B*) One week later the stiffened area has retracted into the gastric lumen corresponding to the "aspect encastré" of Gutmann. (*C*) Ten weeks after *A*, the depressed area has an irregular contour suggesting the phase "muco-érosif." (*D*) Six months after *A*, a small triangular niche is visible within the retracted area—the typical "niche encastrée" of Gutmann.

tending out from the primary focus but appearing separate by conventional techniques of histologic sectioning. Minute foci of submucosal extension can be found and probably is related to the care by which the sections were obtained. In our current



Fig. 9. Although this antral deformity was persistent on multiple roentgenograms, a subsequent cine examination demonstrated striking antral flexibility and unimpaired peristalsis which led to the erroneous impression that this was a benign condition. Superficial cancer was found in the specimen.

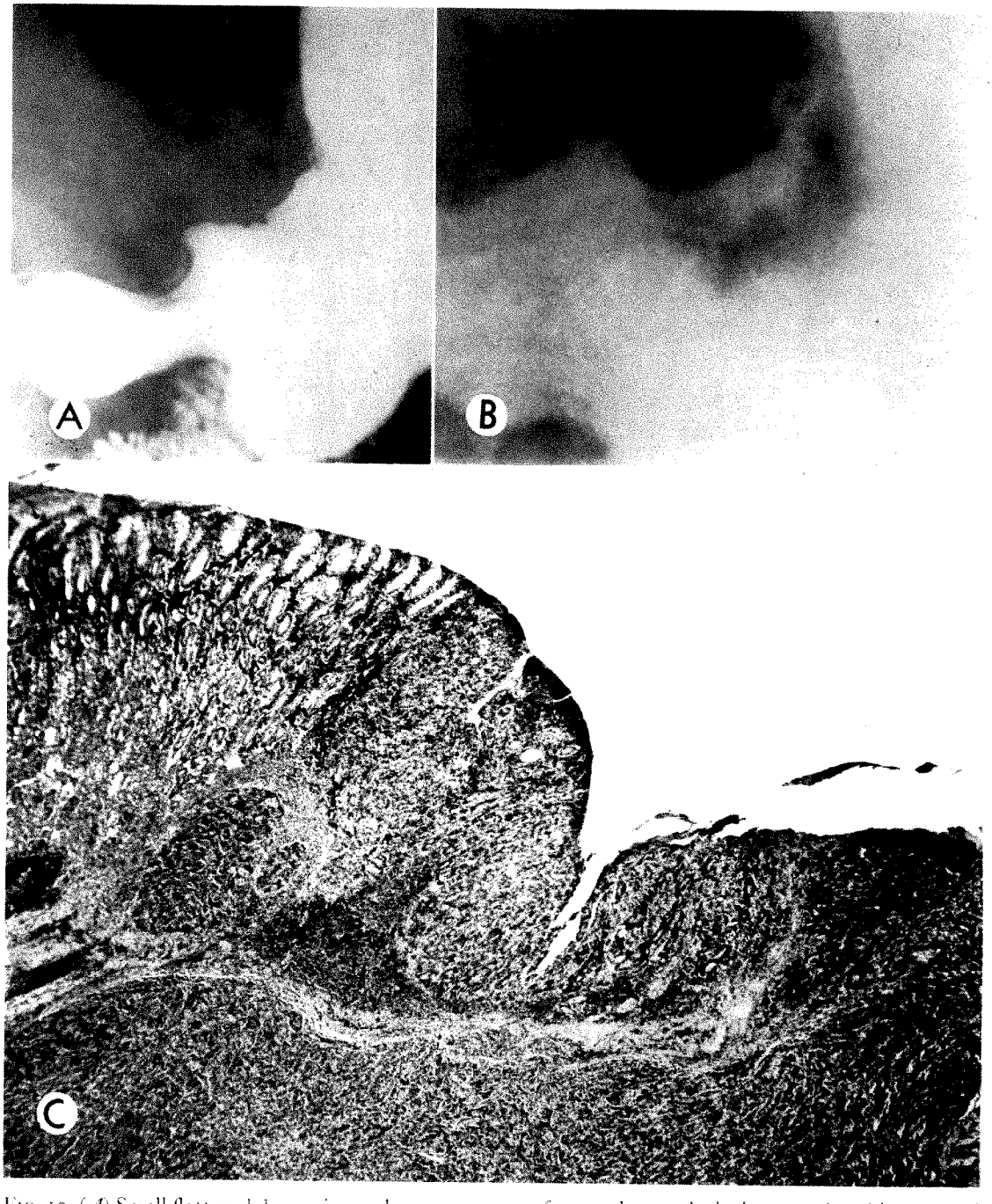



FIG. 10. (*A*) Small flattened depression on lesser curvature of stomach near the incisure produced by mucosal carcinoma. (*B*) Two months later it has progressed to produce the Carman sign. (*C*) Photomicrograph of a section taken through the margin of the crater demonstrating thickened margin due to neoplastic proliferation. Note minimal extension through the muscularis mucosae into the submucosa.

series, limited extension of this character did not appear to adversely affect the prognosis. Lymph node involvement also may occur. The incidence is variable and ranged from 4 in 65 of the Mayo series to 50 per cent of the original Golden-Stout series.[4] Five patients in our series showed positive lymph nodes.

The lesion frequently appears benign to both the surgeon and the pathologist on gross examination. In one case, no lesion was suspected either clinically or at surgery, as the gastrectomy was done for benign duodenal ulcer disease. On histologic sectioning of the stomach, a focus of superficial carcinoma was discovered to have been transected and a second exploration was required to extend the resection. In 11 instances the pathologist on gross examination commented that the lesion was benign and only on subsequent histologic study was the true nature of the malignant process established.

An unusual vegetative or exophytic lesion has been described by Mason.[12] Our series includes one example of an adenomatous polyp with foci of mucosal carcinoma confined to the polyp.

### DIFFERENTIAL DIAGNOSIS

On retrospective analysis of the roentgenograms of 23 patients, 12 appeared definitely malignant, 5 were considered benign and the remaining 6 could not be clearly placed in either category (Table IV). It is important to stress that the diagnostic accuracy of the roentgen study in predicting the malignant nature of the lesion exceeded that of all other diagnostic modalities, even that of the gross pathologic examination. This is due to the ability of this superficial neoplasm to evoke nonneoplastic changes in the surrounding mucosa and submucosa. Albot and Toulet[1] believe that these changes are partly the result of functional alterations in the muscularis mucosae, since they were able to enhance them pharmacologically with morphine and insulin.



FIG. 11. Classical "niche en plateau" of Gutmann in a 34 year old male.

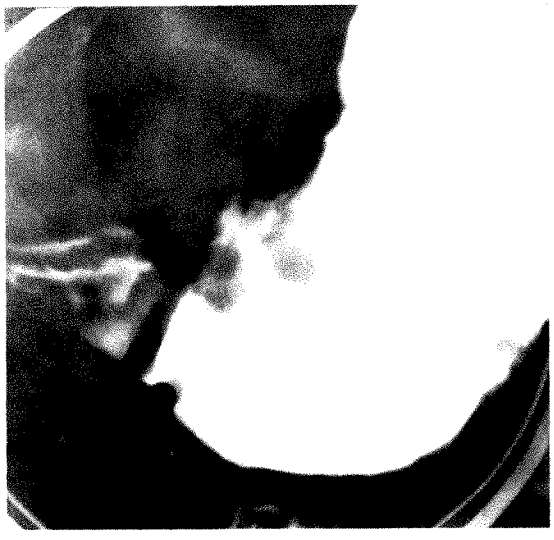


FIG. 12. Superficial gastric cancer with radiating folds.

Although cancer limited to the gastric mucosa can be recognized as a malignant process in the majority of cases, we do not think that it can usually be distinguished from invasive gastric cancer. Sherman and Wilner[13] reached similar conclusions. It must also be emphasized that many of the

TABLE IV

CURRENT ROENTGENOGRAPHIC SERIES

| | |
|---|---|
| Number reviewed | 23 |
| Prospective diagnosis—malignant | 14 |
| Prospective diagnosis—benign | 8 |
| Negative examination | 1 |
| Gastroscopy—number examined | 8 |
| malignant gastroscopic diagnosis | 3 |
| Gross pathology—benign | 11 |
| Retrospective diagnosis—malignant | 12 |
| Retrospective diagnosis—benign | 5 |
| Uncertain | 6 |

changes described above may be associated with benign lesions. However, when they are observed, the possibility of a superficial gastric cancer must be seriously entertained and the patient closely followed.

SUMMARY

Superficial spreading carcinoma of the stomach represents an unusual form of invasive gastric cancer with its predominant pattern of growth limited to the mucosa. Surrounding non-neoplastic changes within the submucosa and muscularis mucosae often make it appear more ominous and extensive roentgenographically. Our current series includes 27 cases with a 66 per cent 5 year survival. A reappraisal of 13 of the original Golden and Stout[4] cases showed a 5 year survival of 69 per cent and 46 per cent at 10 years. No characteristic roentgenologic pattern evolved in our group of cases; however, a correct diagnosis of malignancy was made prospectively in 14 of 23 cases. The roentgenologic appearance varied from normal in one instance, to benign gastric ulcer, to antral deformity alone, and to that of an obvious invasive carcinoma.

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# SQUAMOUS NEOPLASMS OF THE STOMACH*

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SQUAMOUS cell neoplasms of the stomach are very rare, especially *benign* squamous cell neoplasms. Boswell and Helwig[1] in their recent review of the pathologic appearance of such tumors, stated that a squamous cell carcinoma of the stomach was first reported in 1905, and that since then only about 66 additional cases have been related in the literature. Only 2 cases of benign squamous metaplasia of the stomach have been found.[2,6]

The author presents a brief description of the roentgenographic findings of one benign and one malignant primary squamous cell tumor of the stomach.

Malignant squamous cell tumors of the stomach exist as a pure squamous variety or as a combined glandular and squamous cell type (adenoacanthoma). These cancers have been described under various names: adenocanceroid polymorphous epithelioma, malpighian epithelioma, and mucoepidermoid carcinoma. Squamous cell carcinoma occuring in the cardia of the stomach or in conjunction with extension into the esophagus is not considered to be a primary gastric squamous cell carcinoma, but rather a primary esophageal tumor which began either from misplaced islands of squamous cells in the region of the cardia, or from the distal esophagus. Tumors must begin elsewhere in the stomach to be classified as primary gastric squamous cell carcinoma. There should be no evidence of squamous cell carcinoma in any other organ, such as lung, cervix, or pancreas. Pancreatic tumors are rarely squamous cell in type.

The origin of the gastric squamous cells is believed to be metaplasia of the normal glandular epithelium of the stomach. Such metaplasia has been described in the margins of peptic ulcers of humans and squamous metaplasia has been induced in the stomach of rats and rabbits.[3,4] Islands of squamous epithelium have been described along the lesser curvature of a newborn infant who died with acute gastric ulcers.[5]

Because poorly differentiated tumors of the stomach may resemble squamous cell tumors, Boswell and Helwig[1] established 4 criteria for primary squamous cell carcinoma of the stomach:

(a) Keratinizing cell masses with pearl formation
(b) Mosaic pattern of cell arrangement in which cell borders are sharp, some showing individual cell keratinization, with flattened, elongated nuclei, and intense cytoplasmic eosinophilia with whorl formation
(c) Presence of intracellular bridges
(d) Demonstration of high concentration of sulfhydryl or disulfide groups indicating the presence of keratin.

From the small number of cases of malignant squamous cell neoplasms of the stomach reported, the prognosis, age of patients, sex incidence and symptoms do not appear to be different from the usual type of gastric cancer.

## REPORT OF CASES

CASE 1. This 79 year old male with abdominal distress of 3 months' duration was referred for roentgenographic study. The physical examination and laboratory studies were within normal limits. The roentgenographic studies of the chest, skeleton, and gastrointestinal tract showed no abnormality; however, a huge filling defect in the body of the stomach was noted (Fig. 1). This defect produced a ragged contour deformity along the lesser curvature. The fundus and antrum showed no roentgenographic evidence of tumor. At surgery, the tumor was found to involve the anterior wall of the sto-

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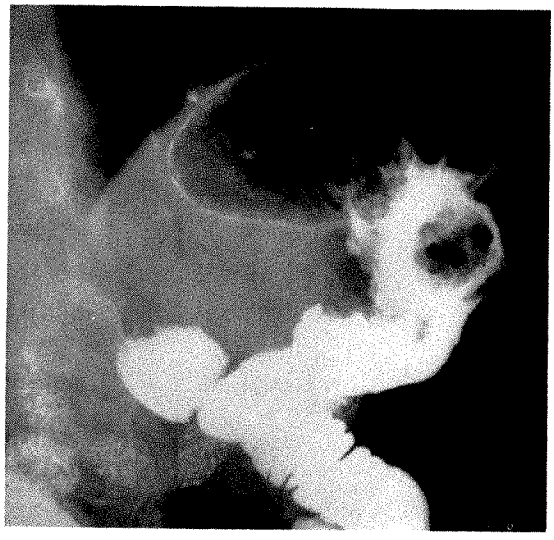


Fig. 1. Squamous cell carcinoma involving the body of the stomach.

mach extending from the lesser to greater curvature of the body of the stomach. The tumor did not involve the posterior wall, the fundus, or the antrum of the stomach. There was one enlarged lymph node in the gastrohepatic area which showed squamous cell carcinoma on microscopic examination. A subtotal gastric resection was performed and the tumor removed. A microscopic study showed pure squamous cell carcinoma. The patient died 3 months later at home and no autopsy was obtained.

Case II. This 47 year old Negro male was referred for roentgenographic study because of epigastric discomfort of 2 weeks' duration. All laboratory studies and the physical examination were negative except for a strongly positive serologic test for syphilis and a very extensive filling defect involving the body and antrum of the stomach (Fig. 2). The patient was treated for 10 days with large antiluetic doses of penicillin and the roentgenographic studies of the stomach were repeated following this therapy. Because it was concluded that there was no improvement in the appearance of the stomach as a result of the therapy, a total gastrectomy was performed for an extensive "cancer" that did not involve either the serosal surface of the stomach or the adjacent organ or lymph nodes. The microscopic studies of the lymph nodes showed no evidence of any tumor. Microscopic studies of the removed stomach showed mature squamous cells without any involvement of the muscular or serosal layers of the stomach. Following the surgery the patient was frequently seen in the out-patient clinics for the next 7 years for treatment of a megaloblastic anemia and chronic urinary tract infections. He never showed any evidence of recurrent tumor and after 7 years was lost to follow-up.

## DISCUSSION

A review of the previously reported cases of squamous cell carcinoma shows that reference to the roentgenographic findings was made in only a few cases. Most of the tumors were large and bulky, and found in either the body or the antrum of the stomach. The roentgenographic findings demonstrated no single feature which would distinguish them from the usual adenocarcinoma of the stomach.

Only 2 reports of benign squamous cell metaplasia have been found in the literature. Rezek[6] reported the histologic findings of our case in 1953 in the Austrian literature, and Carr and Squires[2] reported a similar case in 1962 in the American literature. Both cases were large bulky tumors that filled most of the stomach. The clinical, roentgenographic and surgical find-

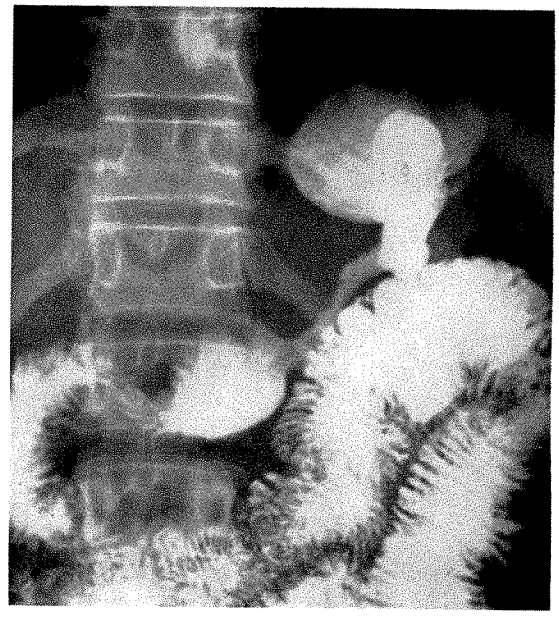


Fig. 2. Benign squamous cell papillomatosis of the stomach.

ings appeared to be typical of gastric malignancy, but on microscopic study showed no evidence of malignancy. The tumor consisted of mature squamous cells with no invasion of the stomach wall. Neither case showed any extension beyond the stomach submucosa, or involvement of any of the regional lymph nodes. The case of Carr and Squires died immediately following the postoperative period and autopsy showed no tumor elsewhere in the body. Our case lived for 7 years after gastrectomy and then was lost to follow-up. He showed no recurrent tumor at physical or roentgenographic examination when last seen. Squamous cell papillomatosis of the stomach is evidently a very rare condition and from the 2 cases reported, would appear to be indistinguishable roentgenographically from an extensive malignant tumor of the stomach.

## CONCLUSION

A case of uncommon primary squamous cell carcinoma of the stomach and a case of a very rare benign squamous papillomatosis of the stomach are reported with illustrations of the respective roentgenographic appearances.

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# THE GASTRIC INLET JET*

*By* HAROLD I. AMORY, M.D.†
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JET phenomena occurring in the cardiovascular system have been useful aids in roentgenologic diagnosis since the advent of angiocardiography; their observation is diagnostic of stenosed and incompetent valves and constricted vessels. Post stenotic dilatation of vessels resulting from the jet effect was recognized at an early date.

In excretory urography jets of opacified urine from a ureteral orifice coursing caudad and medially through nonopacified urine are a fairly common occurrence.[2]

Jets in other systems are not well known and descriptions of them in medical literature are nonexistant. They do occur, however, in the gastrointestinal tract and the purpose of this report is to describe and illustrate the jet phenomenon observed at the esophagogastric junction in patients having primary carcinoma involving this region.

The only reference to a related effect was found in the description by Pollard and Nebesar.[1] A little squirt of barium going through the gastric inlet in a case of Menetrier's disease was mentioned by these authors and the designation "squirt sign" was used. Personal communication with numerous colleagues has failed to reveal knowledge of gastrointestinal jets.

## REPORT OF CASES

Case I. P.D., a male 67 years of age, began experiencing regurgitant episodes and he sought aid from his local physician. Barium swallow study, upper gastrointestinal series, and cholangiography revealed cholelithiasis; so cholecystectomy was performed. Postoperatively he continued to vomit and began to lose weight (26 pounds in 3 months). Two weeks prior to admission, he began having sharp knife-like pains substernally radiating to his back and becoming rather continuous during the past 2 days. He again consulted his local physician who obtained a second barium swallow examination which revealed a mass in the distal esophagus. He had not experienced hemoptysis, dysphagia, cough, hoarseness or melena; he had become quite weak and pale recently. He stated that he is always hungry but cannot hold his food down.

Upper gastrointestinal series disclosed mild dilatation of the esophagus. While observing the air filled cardiac portion of the stomach in the upright position, a solid .6 cm. diameter stream of barium suspension was seen projecting from the gastric inlet in an arc through the Magenblase toward the greater curvature of the stomach. The stream of barium joined the fluid already in the stomach without surface disturbance. The stream was at a maximum trajectory when the esophagus was full. As the column of fluid in the esophagus diminished reducing the hydrostatic pressure at the esophagogastric junction, the stream diminished and decreased in trajectory until it merely trickled down the Magenstrasse. The jet stream could be repeated at will and was identical in size and arc at each occurrence. Spot roentgenograms were made of the stream, showing its variation in trajectory relative to the esophageal hydrostatic pressure. The sign could not be recorded cinefluorographically as the equipment available was not designed for upright studies (Fig. 1–4).

Observation of the cardia revealed thickened gastric wall and constriction of the gastric inlet. A tentative diagnosis of carcinoma was made and adenocarcinoma was confirmed pathologically following an esophagogastrectomy. The specimen consisted of four-fifths of the stomach and the distal end of the esophagus. The gastric inlet was funnel shaped and the orifice was constricted to a diameter of several millimeters.

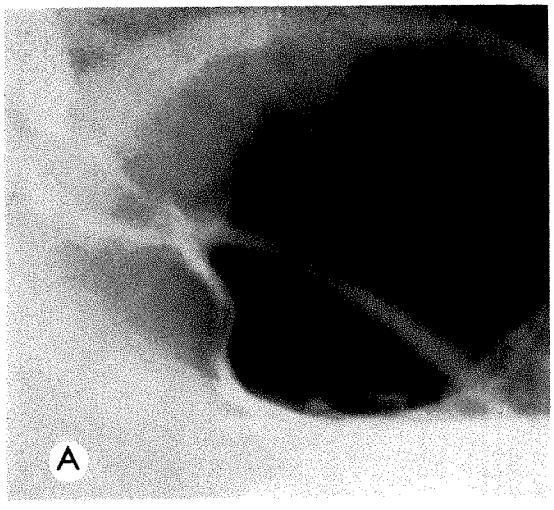
Case II. C.T., a male 50 years of age, upon admission stated that roentgenographic examination for ulcer had indicated that tumor may be present in the top of his stomach. Four weeks previously he began having difficulty swallowing solid food which seemed to lodge in

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the lower esophagus. Fluids could be swallowed without difficulty and there was no nausea or vomiting.

Upper gastrointestinal series revealed constriction and nodular irregularity of the distal 10 cm. of the esophagus and a small zone of irregularity and thickening of the adjacent gastric wall was demonstrated. A roentgen diagnosis of esophagogastric carcinoma was made. Esophagoscopy and biopsy revealed adenocarcinoma of the esophagus.

During the fluoroscopic examination in the upright position, 2 jets of barium were seen projecting from the gastric inlet. The streams in Case 1 passed through the gas bubble arcing downward toward the greater curvature of the stomach. The stomach contained a smaller quantity of gas than in Case 1 and the jets were



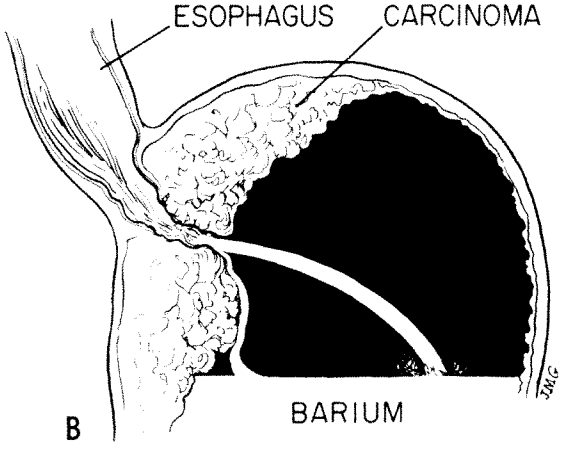




Fig. 1. Case 1. (*A*) High pressure jet, long flat trajectory. (*B*) Constriction of esophagogastric junction by carcinoma producing jet.

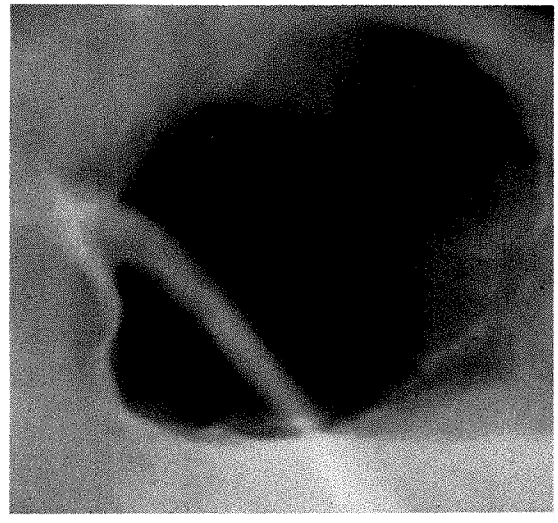


Fig. 2. Case 1. Moderate pressure jet, decreasing trajectory. Stream penetrates into less opaque barium pool.

of shorter duration and had a lower trajectory. They could, however, be repeated at will and at each occurrence the size of the streams and their location were identical (Fig. 5).

## DISCUSSION

In the cases presented the jets were the result of constriction and fixation of the gastric inlet by primary adenocarcinoma, while the spray mentioned by Pollard and Nebesar[1] resulted from hypertrophic gastric mucosal folds. It is conceivable that jets



Fig. 3. Case 1. Low pressure jet, short trajectory.

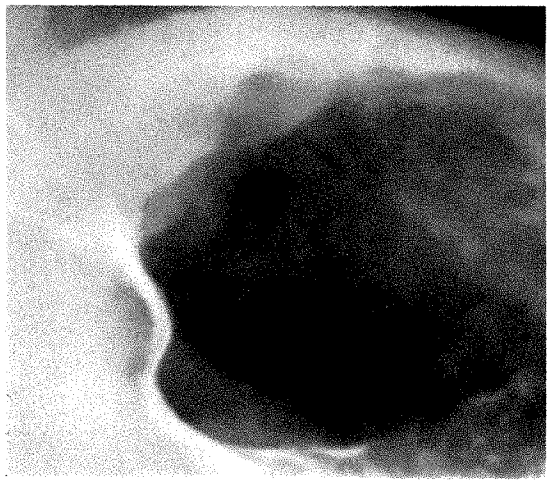


FIG. 4. Case I. Slight pressure, negative jet. Flow of barium down deformed Magenstrasse.

and sprays could result from stricture, cardiospasm, or anatomic variants but reports of such occurrences could not be found. Jets have not been observed by the author in the normal gastrointestinal tract, although special efforts have been made to produce them. Barium coursing down the Magenstrasse may stimulate the jet but the true situation becomes evident when the patient is rotated to place the Magenstrasse in a position tangential to the roentgen-ray beam.

In view of the sparsity of reports, the esophagogastric junction jet would appear uncommon. It may occur frequently but is not observed as efforts to elicit it have not been made. A review of all pertinent cases of carcinoma on record in the author's institution is being made in an attempt to discover jets that may have been overlooked, and future gastrointestinal series will include maneuvers designed to disclose their presence. Although its incidence is not now known, the sign is of such specific value that attention during fluoroscopy should be devoted to determine whether or not it is present. The upright position is, of course, mandatory and a substantial quantity of gas in the cardia is helpful. The patient should be placed in the right oblique position and the initial bolus of barium carefully observed as it enters the stomach. A jet stream or spray of barium which can be identically repeated at will should be interpreted as a positive jet sign indicating organic fixation of the gastric inlet. When the sign is present, carcinoma should be given first consideration and a careful search is warranted.

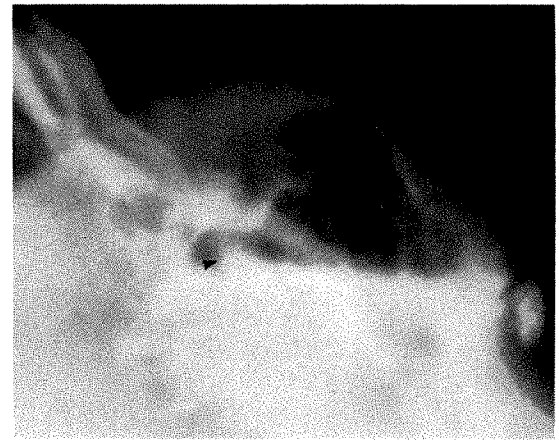


FIG. 5. Case II. Forked jet. Two streams repeatedly observed fluoroscopically.

SUMMARY

Two cases of carcinoma of the esophagogastric junction showing the jet sign are reported.

The gastric inlet jet sign is described and illustrated. Its observation is an aid in the detection of carcinoma in a region where diagnostic failures are common—the esophagogastric junction.

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# ROENTGENOLOGIC DIAGNOSIS OF PYLORIC HYPERTROPHY IN ADULTS*

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DESCRIPTIONS of the roentgenologic signs usually associated with hypertrophy of the pyloric muscle in adults[2,6] would lead one to believe that the diagnosis, made in this way, is relatively accurate and that the remarkable lengthening of the pyloric canal described is due to thickening of the overlying muscle. More recent study,[8] in addition to follow-up of a number of cases showing these characteristic roentgenologic signs, has suggested, however, that even though muscular thickening of the most distal pyloric muscle may cause some elongation of the canal, muscular thickening in the precanal region has little to do with the production of these signs. This implication stimulated us to carry out (1) a more methodical analysis of the anatomic findings at the pyloric canal in patients on whom such a roentgenologic diagnosis was made and (2) an evaluation of roentgenograms of patients whose diagnosis of pyloric muscle hypertrophy rested on more direct assessment at laparotomy.

Roentgenologically, the pyloric canal normally is 0.8 to 1 cm. long.[3,4,6] It has been reported to be 2 to 4 cm. long with hypertrophy of the pyloric muscle.[4,6] Muscle thickness at the pyloric canal of normal adults has been reported to vary from 3.8 to 8.5 mm., with a mean of about 5 mm.[5,8] Thickening has been found microscopically to be due chiefly to hypertrophy and hyperplasia of the circular muscle fibers, with a smaller contribution made by the longitudinal fibers and by edema of the mucosa and submucosa.[7] Reduction in the number of myenteric ganglion cells and nerve fiber tracts has also been reported in this condition.[1]

The purpose of this study is to evaluate the generally accepted roentgenologic signs of pyloric muscle hypertrophy by attempting to correlate the pathologic anatomy with the corresponding roentgenologic anatomy.

## MATERIALS AND METHODS

Two methods were used to evaluate the roentgenologic signs.

In the first approach (roentgenologic group), 100 consecutive cases diagnosed roentgenologically as pyloric muscle hypertrophy in an adult and including satisfactory roentgenograms were studied. These cases represented those seen in most of the year 1959 and all of 1960 and 1961. The patients ranged in age from 20 to 79 years; 75 were in the sixth or seventh decade (the one 20 year old patient had congenital hypertrophic pyloric stenosis). There were 85 men and 15 women. In 81 patients the primary symptom was epigastric pain or fullness.

In the second approach (surgical group), 50 consecutive cases in which there were satisfactory preoperative stomach roentgenograms and in which the diagnosis was made either by the surgeon at laparotomy or by the pathologist on examination of the resected specimen were evaluated. These cases covered the 7 year period, 1956 through 1962. The patients ranged from 27 to 80 years of age, with 30 in the sixth or seventh decade; 39 were men and 11 were women. Forty patients had epigastric pain or fullness.

The 4 roentgenologic signs evaluated were: (1) lengthening of the pyloric canal; (2) degree of narrowing of the pyloric canal; (3) crescentic indentation at the base of the duodenal bulb; and (4) intact mucosal relief in the pyloric region—criteria used by Kirklin and Harris and several other au-

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thors. The contrast-filled cleft in the pyloric canal was also noted if seen.

In both groups all available pathologic specimens were studied. The mean of the maximal thickness of the pyloric ring muscle on the lesser and greater curvatures of the stomach was determined. The thickness also was measured at 1 cm. intervals for a total of 4 cm. proximal to the most distal portion of the pyloric ring muscle. Specimens of the muscle were taken at right angles to the pyloric ring muscle and reviewed microscopically for ganglion cells and nerve fibers. Among the 100 consecutive cases diagnosed roentgenologically, 28 cases had been explored surgically and tissue was available for review from 11 of these cases. From the 50 consecutive cases confirmed surgically, 37 specimens were available for study (the remaining patients in this group had had pyloroplasty).

In addition, 10 stomach specimens obtained at autopsy in cases without significant gastrointestinal symptoms or apparent disease were similarly studied to obtain normal values.

## RESULTS

### NORMAL

The pyloric muscle thickness in the 10 normal stomachs ranged from 0.4 to 0.7 cm. The measurements at 1 cm. intervals proximal to the gastroduodenal junction were: at 1 cm., 0.2 to 0.5 cm.; at 2 cm., 0.2 to 0.4 cm.; at 3 cm., 0.2 to 0.3 cm.; and at 4 cm., 0.2 to 0.3 cm.

### ROENTGENOLOGIC GROUP

*Original Diagnosis.* Among the 100 cases diagnosed roentgenologically, 38 were originally diagnosed as hypertrophic pyloric muscle without any additional abnormality, 46 as hypertrophic pyloric muscle plus peptic ulcer (duodenal in 29, canal in 12, and gastric in 5), and 4 as hypertrophic pyloric muscle with the possibility of neoplasm in the pyloric region; in addition to hypertrophic pyloric muscle, there was coexisting esophageal hiatal hernia in 7 cases and various other miscellaneous conditions in 5.

The upper abdominal region had been explored surgically in 28 of these 100 cases. In 10 the stomach was considered to be completely normal by the surgeon. In 8 the pyloric muscle was thickened, either as judged by the surgeon or as determined by measurement of the fixed specimen. Pyloric carcinoma was present in 5 cases; the question of carcinoma had been raised on the preoperative roentgenologic examination in 4 of these. The hypertrophic pyloric muscle was the sole abnormality in 2 of the 8 cases and was associated with duodenal or gastric ulcer in 6.

*Retrospective Review.* The roentgenograms in these 28 surgically explored cases were restudied with special note taken of abnormalities in the pyloric region, which might be suggestive of hypertrophy (Fig. 1*A*). In 5 cases they were thought to show no evidence of pyloric hypertrophy; none of these 5 had pyloric abnormality on surgical exploration. In 10 cases the roentgenograms were thought to show good evidence for hypertrophic pyloric muscle with no other abnormalities; at surgical exploration, the stomach was normal in 5 cases, had pyloric carcinoma in 2, had hypertrophic pyloric muscle (surgeon's opinion) in 1, and had gastric ulcer plus hypertrophic pyloric muscle (1.3 cm. thick in resected specimen). The tenth case was congenital hypertrophic pyloric stenosis, treated surgically, in a 2 month old infant. In 9 cases the roentgenograms were thought to show good evidence of hypertrophic pyloric muscle plus peptic ulcer; surgical exploration showed a normal stomach in 1 case, hypertrophic pyloric muscle (surgeon's opinion) in 1, duodenal ulcer plus hypertrophic pyloric muscle (by measurement of resected specimens) in 4, and gastric ulcer, 1 with hypertrophic pyloric muscle (by measurement of resected specimens), in 3. In 4 cases, the roentgenograms suggested the possibility of carcinoma of the pyloric region; at surgical exploration, carcinoma was found in 3 of these cases (Fig. 2) and an obstructing duodenal ulcer in 1.

*Pathology.* Specimens were available for review in 11 of the 28 surgically explored

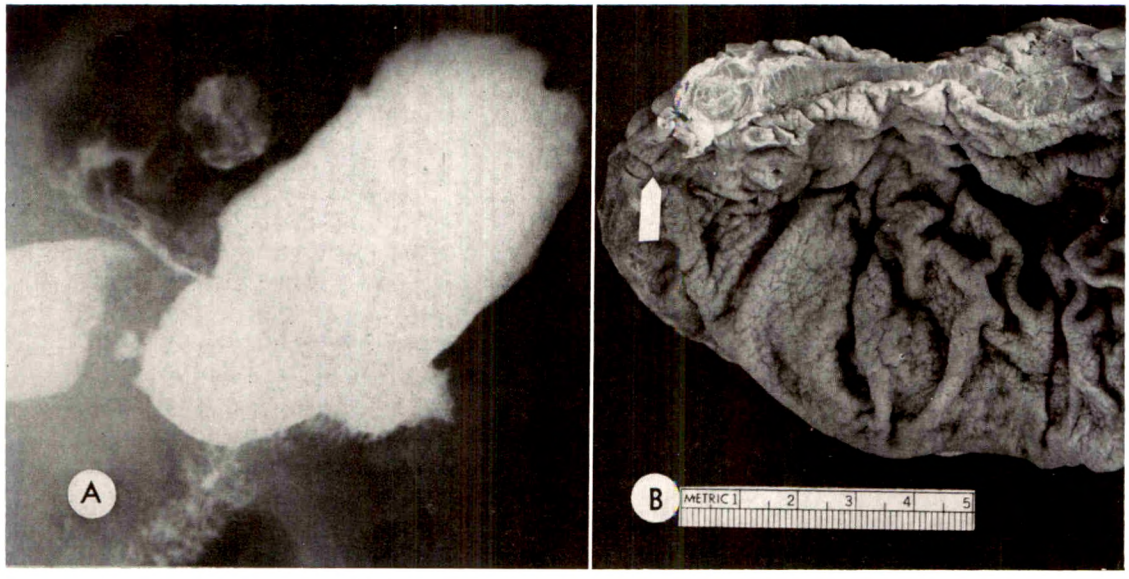



Fig. 1. Case diagnosed preoperatively as pyloric hypertrophy in 47 year old man. At operation, a gastric ulcer was found 9.0 cm. from the pyloric ring. (*A*) Roentgenogram shows all the conventional signs of pyloric hypertrophy. Pyloric canal roentgenographically was 2.0 cm. long. (*B*) Surgical specimen removed 7 years later. Pointer is at the gastroduodenal junction. Normal number of ganglion cells and neural fibers were found microscopically. Pyloric ring was 1.3 cm. thick; prepyloric muscle was not thick.

cases. Six of the specimens could be considered to have hypertrophic pyloric muscle alone and 3 others had pyloric carcinoma with associated muscular hypertrophy. Among these 9 specimens, the mean pyloric muscle thickness was 1.0 cm. (range, 0.9 to 1.3 cm.; median, 0.9 cm.). One of the pyloric carcinoma specimens had carcinoma cells infiltrating the pyloric ring muscle, which contributed to the thickness of the pyloric muscle. On microscopic review, 8 cases were found to have a normal number of ganglion cells and neural fibers and one had a reduced number of nerve elements.

An attempt was made to evaluate the roentgenologic narrowing and lengthening in the pyloric region relative to the thickness of the pyloric muscle. Of the 9 specimens that were 0.9 cm. or more thick at the pyloric ring, only 2 had increased thickness of the prepyloric muscle beginning at the 2.0 cm. interval when compared with the above measurements. Both of these patients had pyloric carcinoma. All of the 9 patients showed elongation and narrowing of the pyloric canal on the roentgenograms: the length of the pyloric canal on the roentgenograms varied from 1.5 to 3.0 cm. and the narrowing was almost equally divided among slight, moderate, and marked. Thus, the rather striking canal elongation seen on the roentgenogram in this group of patients is not due to overlying prepyloric muscle hypertrophy.

*Other Roentgenologic Signs.* In 6 of the 9

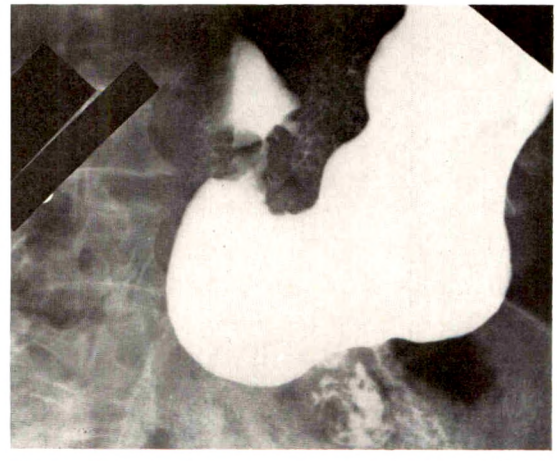


Fig. 2. Preoperative roentgenographic diagnosis was duodenal ulcer and hypertrophic pyloric muscle in a 60 year old woman. Questionably intact mucosa was in the pyloric region when the roentgenogram was reviewed. At operation, Grade II prepyloric adenocarcinoma was found. The pyloric ring in the specimen measured 0.8 cm.

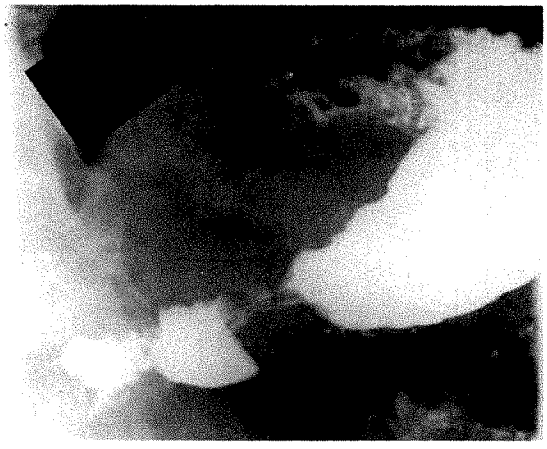


FIG. 3. Roentgenogram of 62 year old man shows signs of hypertrophic pyloric muscle plus pyloric canal ulcer. The pyloric ring measured 1.0 cm. in the specimen and was associated with increased thickness of the prepyloric muscle for 3.0 cm.; however, on the roentgenogram the pyloric canal measured only 2.0 cm. A 0.5 cm. ulcer near the pyloric ring was also present in the specimen.

cases with a pyloric muscle 0.9 cm. or more thick, there was a prominent indentation at the base of the duodenal cap. However, 3 of these 6 specimens had pyloric carcinoma.

Of the 4 cases in which there was a question of intact mucosa, 3 proved to be pyloric carcinoma.

A contrast-filled cleft in the pyloric canal was observed on the roentgenogram in only 1 of the 11 cases in which specimens were available for review (Fig. 1*B*). Among the 72 cases not explored surgically, a contrast-filled cleft in the pyloric canal was present on the roentgenogram in 9.

SURGICAL GROUP

Among these 50 cases, the pyloric region was not resected in 13 and the diagnosis of hypertrophic pyloric muscle was made on the basis of surgical inspection. In 37 cases the pyloric region was resected and the diagnosis was made by the surgeon, the pathologist, or both.

*Original Diagnosis.* Preoperatively a pyloric canal abnormality was diagnosed roentgenologically in 20 cases. These diagnoses included possible canal ulcer in 6 cases, possible pyloric carcinoma in 5, pyloric hypertrophy in 4, hypertrophy with duodenal ulcer in 1, duodenal ulcer in 3, and antral spasm in 1.

The 30 cases in which no pyloric abnormality was noted roentgenologically included 19 diagnosed as peptic ulcer, 6 as normal, and 5 as a variety of abnormalities.

*Retrospective Review.* Only 19 cases fulfilled the conventional roentgenologic criteria for pyloric hypertrophy.

*Pathology.* A pathologic-anatomic diagnosis of hypertrophy without other associated abnormalities was made in 12 cases. Other associated pathologic findings were noted in the remaining 38 cases, with 23 having either a duodenal or gastric ulcer.

In the 37 resected specimens examined, the pyloric muscle thickness varied from 0.7 to 1.5 cm. Only 19 of the 37 specimens qualified pathologically as representing hypertrophic pyloric muscle; the mean thickness in these was 1.1 cm. (range, 0.9 to 1.5 cm.; median, 1.0 cm.). Microscopic examination of the 19 specimens with thickened muscle revealed 13 with a normal number of ganglion cells and nerve fiber tracts, 4 with a reduced number of nerve elements, and 2 with inflammatory foci and some reduction in neural elements.

An attempt was also made in this group to correlate the roentgenologic pyloric lengthening with the prepyloric muscle thickness. Among the 19 cases with pyloric muscle measuring 0.9 cm. or more, only 7 manifested roentgenologic evidence of pyloric hypertrophy on the basis of conventional criteria (Fig. 1*A*; and 3). Roentgenograms showed slight narrowing in 5 of these 7 cases and moderate narrowing in 2. Two of the specimens had pyloric ring hypertrophy with no increased thickness of the prepyloric muscle but, in contrast to the anatomic findings, the roentgenograms showed pyloric elongation of 2.0 and 2.5 cm. In 4 of the other 5 specimens, the prepyloric muscle was thicker than normal up to 3.0 cm. proximal to the pyloric ring; in 3 of these 4 the roentgenograms showed pyloric elongation of 2.0 cm. and in the other, of only 1.0 cm. (Fig. 4); the remaining specimen was abnormally thick for 4.0 cm. but

had a canal elongation of only 1.5 cm. on the roentgenogram.

The thickness of the prepyloric muscle was also measured in 7 cases in which the pyloric muscle was 0.9 cm. or more thick but in which none of the conventional roentgenologic criteria were present. In 4 of these the prepyloric muscle was thicker than normal (for 2.0 cm. in 1, for 3.0 cm. in 2, and for 4.0 cm. in 1).

Thus, in these 19 specimens and roentgenograms, no correlation between the roentgenologic pyloric elongation and the prepyloric muscle thickness could be found. None of the 19 roentgenograms had the contrast-filled cleft in the pyloric canal.

In 18 specimens the pyloric muscle was less than 0.9 cm. thick. Thirteen of the 18 cases had none of the roentgenologic signs of hypertrophy. In the 5 cases that fulfilled conventional roentgenographic criteria for this entity (Fig. 5), none of the specimens had thick prepyloric muscle.

Among the 13 cases in which no tissue was available for review, 3 had characteristic roentgenologic signs of hypertrophy. Surgical exploration in these 3 cases revealed, in addition to pyloric hypertrophy, duodenal ulcer in 1, cholelithiasis in 1, and heterotopic pancreas in 1. The remainder included 5 cases with no associated abnormality and 5 with cholelithiasis plus pyloric hypertrophy.



Fig. 4. Absence of classic roentgenologic signs of hypertrophic pyloric muscle in 33 year old man. Preoperative diagnosis was an obstructing postbulbar duodenal ulcer, which was found at operation. Hypertrophic pyloric muscle (1.1 cm.) was found in the specimen.

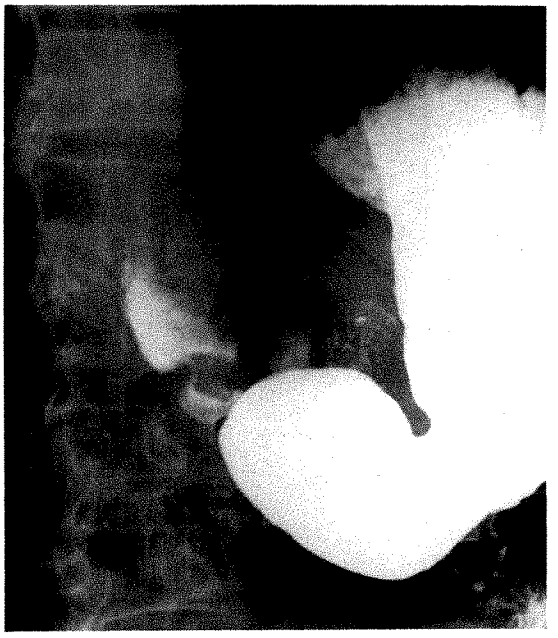


Fig. 5. Roentgenologic signs of hypertrophic pyloric muscle in a 66 year old man; note particularly the pyloric canal elongation. Preoperative roentgenologic diagnosis was antral spasm. The pyloric ring measured only 0.7 cm., which is within the limits of normal.

## DISCUSSION

Elongation and narrowing of the pyloric canal have been reported to be the main roentgenologic signs of hypertrophic pyloric muscle in an adult.[4,6] No direct correlation between prepyloric muscle thickness and roentgenologic pyloric elongation and narrowing was found in this study. Pyloric canal length of 1.0 to 2.0 cm. on the roentgenogram may conceivably be due to pyloric ring hypertrophy but a persistent length of more than 2.0 cm. probably is due to muscular contraction or infiltration rather than thickening of the prepyloric muscle. In this review, some specimens had thick prepyloric muscle but no pyloric elongation on the roentgenogram and in others elongation was present on the roentgenogram but was not comparable to the length of prepyloric thickening.

A crescentic indentation at the base of the duodenal cap has also been claimed to be an important sign of hypertrophic pyloric muscle.[6] This review of roentgenograms and specimens showed that the indentation at the base of the duodenal cap was not specific for pyloric hypertrophy in adults. In many of the cases of surgically confirmed hypertrophy there was a prominent indentation but there was also an almost equal number of confirmed cases of hypertrophy in which there was no duodenal cap indentation on the roentgenogram.

Although the contrast-filled cleft in the pyloric canal has been said to be pathognomonic of pyloric hypertrophy,[9] it proved to be the least valuable roentgenologic sign in this review. Only one specimen with verified hypertrophic pyloric muscle had the contrast-filled cleft on the roentgenogram.

If the mucosal pattern in the pyloric region is questionable, pyloric carcinoma must be excluded by surgical exploration. Three cases in this series had such findings when the roentgenograms were reviewed; there was pyloric carcinoma in 2 and marked scarring secondary to a duodenal ulcer in the other.

An associated gastric or duodenal abnormality is present with pyloric hypertrophy in an adult in the majority of cases.[6,7] Among 30 surgically and pathologically confirmed cases of hypertrophy (11 from the roentgenologic group and 19 from the surgical group) a gastric or duodenal ulcer was present in 19.

## SUMMARY

One hundred consecutive roentgenologically diagnosed and 50 consecutive surgically diagnosed cases of hypertrophic pyloric muscle in adults were studied. The roentgenograms, available specimens, and clinical histories were reviewed. An evaluation of the commonly accepted roentgenologic signs of this condition was attempted. It is likely that the roentgenographic appearance of pyloric elongation and narrowing is due to a combination of pyloric muscle hypertrophy and prepyloric muscular contraction rather than to actual thickening of the prepyloric muscle. A crescentic indentation at the base of the duodenal cap may or may not be present in hypertrophic pyloric muscle and by itself means very little. The mucosal pattern in the pyloric region is very important in the evaluation of any abnormality; if there is any question of whether the mucosa is intact when the pyloric region is narrowed and elongated, carcinoma probably is present. The contrast-filled cleft in the pyloric region was only infrequently present. A gastric or duodenal ulcer was associated with confirmed pyloric hypertrophy in the majority of patients.

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VOL. 101, NO. 2

# THE RETAINED GASTRIC ANTRUM*

## PREOPERATIVE ROENTGENOLOGIC DIAGNOSIS OF AN IATROGENIC SYNDROME

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THE recognition of the antral mucosa of the stomach as an endocrine organ[32] was a significant step forward in our understanding of peptic ulcer disease and its surgical therapy. Edkins[9] in 1906 had already concluded that gastric acid secretion from parietal cells occurs in response to a hormone released into the circulation from the gastric antrum. It was, however, not until 1951 that the importance of his work was confirmed. Studies at that time by Dragstedt and his co-workers[6] on the function of the antrum in the gastric phase of ulcerogenesis resulted in a reappraisal of the rationale for operative procedures.[21,22,23]

The so-called "antral-exclusion operation" represents a surgical procedure for duodenal ulcer disease with transsection of the stomach, end-to-side gastrojejunostomy and retention of the gastric antrum in continuity with the pylorus and duodenum (Fig. 1). This procedure was described by von Eiselsberg[30] in 1910, Finsterer[10] in 1918, and Devine[5] in 1925.

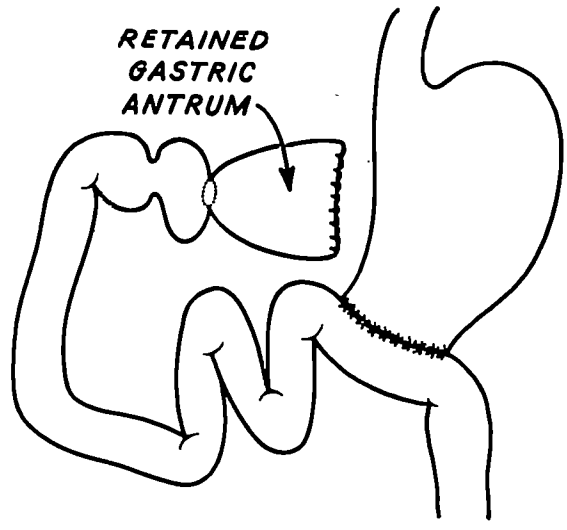



FIG. 1. Diagrammatic drawing of antral-exclusion operation. The stomach is transsected distally and a portion of the body of the stomach may be resected. The proximal gastric cut end is anastomosed in an end-to-side fashion to the jejunum. The distal cut end of the stomach transsection is closed and the antrum is left in place in continuity with the pylorus and duodenum. Acid secretions from the fundus and body of the stomach no longer reach the antrum, which is now bathed in alkaline reflux.

It soon became apparent that the antral-exclusion procedure was followed by a very high rate of ulcer recurrence. McKittrick *et al.*[20] reported ulcer recurrence 3 to 36 months after an antral-exclusion operation in 9 patients. Allen and Welch[1] found ulcer recurrence in 5 out of 9 patients undergoing this procedure. Ogilvie[24] performed antral exclusion in 22 patients with ulcer recurrence in 9 instances. Kay[17] reported 8 patients with ulcer recurrence out of 20 antral-exclusion operations. At a second operation, the antrum was removed with no further recurrence in all 8 cases. Even after vagotomy was added to antral exclusion, failure was reported by Waddell and Bartlett,[31] although only 9 jejunal ulcers developed in a series of 153 patients.

The high incidence of ulcer recurrence after antral-exclusion operations compares to a marginal ulcer rate of between 3 and 6 per cent in cases of conventional partial gastrectomy with resection of the antrum. The high rate of marginal ulcers after antral exclusion has been well described in

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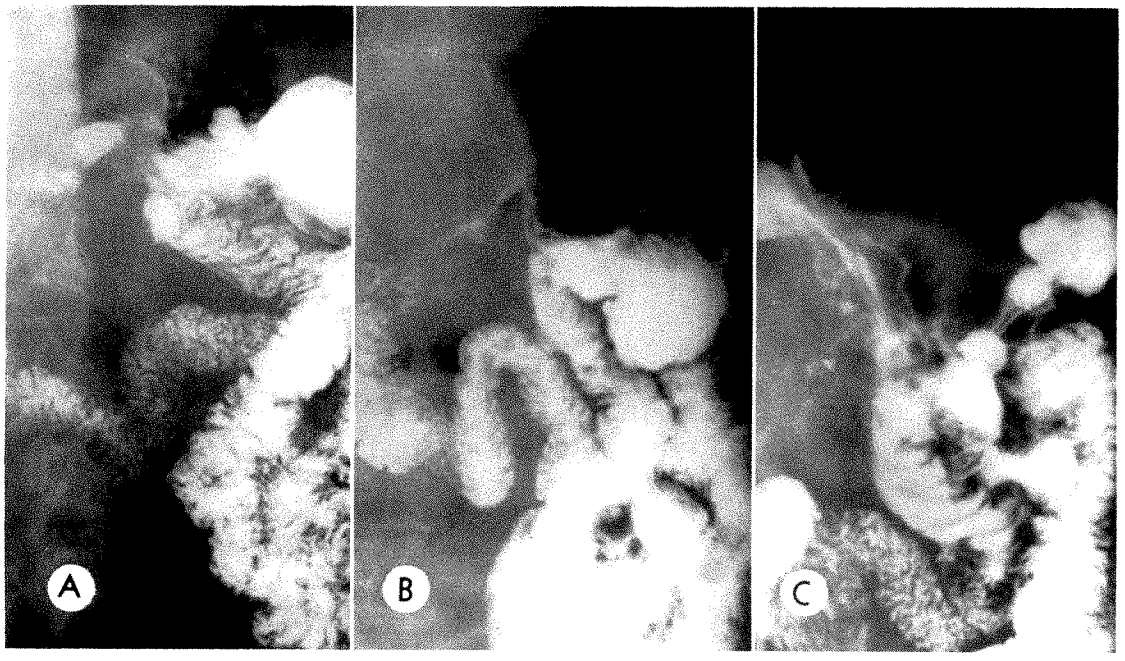



FIG. 2. Case 1. (*A*). Partial distal gastrectomy and gastrojejunostomy with marginal jejunal ulcer. (*B*) Filling of afferent proximal jejunum. (*C*) Filling of colon from anastomotic ulcer. Marginal ulcer and a gastrojejunocolic fistula were confirmed at surgery. A portion of the antrum had been left in place. It is not clear from these roentgenograms whether reflux occurred from a deformed duodenum into the retained antrum.

the surgical literature during the past decade. Although Rauch[26] stated in 1952 that the antral-exclusion operation was abandoned due to a high ulcer recurrence rate of 16.5 per cent, the procedure is apparently still practiced. This report deals with 4 cases of retained gastric antrum with ulcer recurrence referred to the University of California San Francisco Medical Center during the past 6 years. In none of the 4 cases was it clear from the history whether the antrum was intentionally left *in situ*. In no instance was the antrum diseased.

## REPORT OF CASES

CASE 1. S.H. This 38 year old male had a 2 year history of duodenal ulcer preceding an episode of obstruction in 1960. A 70 per cent distal gastrectomy was performed at that time with an end-to-side gastrojejunostomy. The anastomosis was antecolic. The patient was symptom free for 5 months, when midline epigastric pain recurred. Medical therapy was instituted with temporary relief. The pain became more persistent and vomiting occurred occasionally. Stool examinations were positive for occult blood. Eight months postoperatively, the patient noticed undigested food in his bowel movements. He lost 20 pounds of weight. The patient was referred with a clinical diagnosis of gastrojejunocolic fistula due to marginal ulcer. This was confirmed by roentgen study (Fig. 2, *A*, *B* and *C*). Filling of the proximal jejunum and duodenum occurred, outlining a deformed duodenum, and possibly some reflux into a retained antrum. This was not recognized roentgenographically. At exploration, a 1.5 cm. marginal ulcer was found with perforation into the colon and presence of a jejunocolic fistula. During the revision of the anastomosis, it was noted that a 5 cm. segment of gastric antrum had been retained. The antrum was resected and the duodenal stump was closed.

The patient regained 10 pounds of weight following the revision of the gastrojejunostomy. A 2 year follow-up revealed no recurrence of ulcer symptoms. Some diarrhea was present and was attributed to the vagotomy which had been added at re-exploration.

CASE II. G.A. This 48 year old male was referred in 1960 with a history of persistent postoperative marginal ulcer demonstrated on

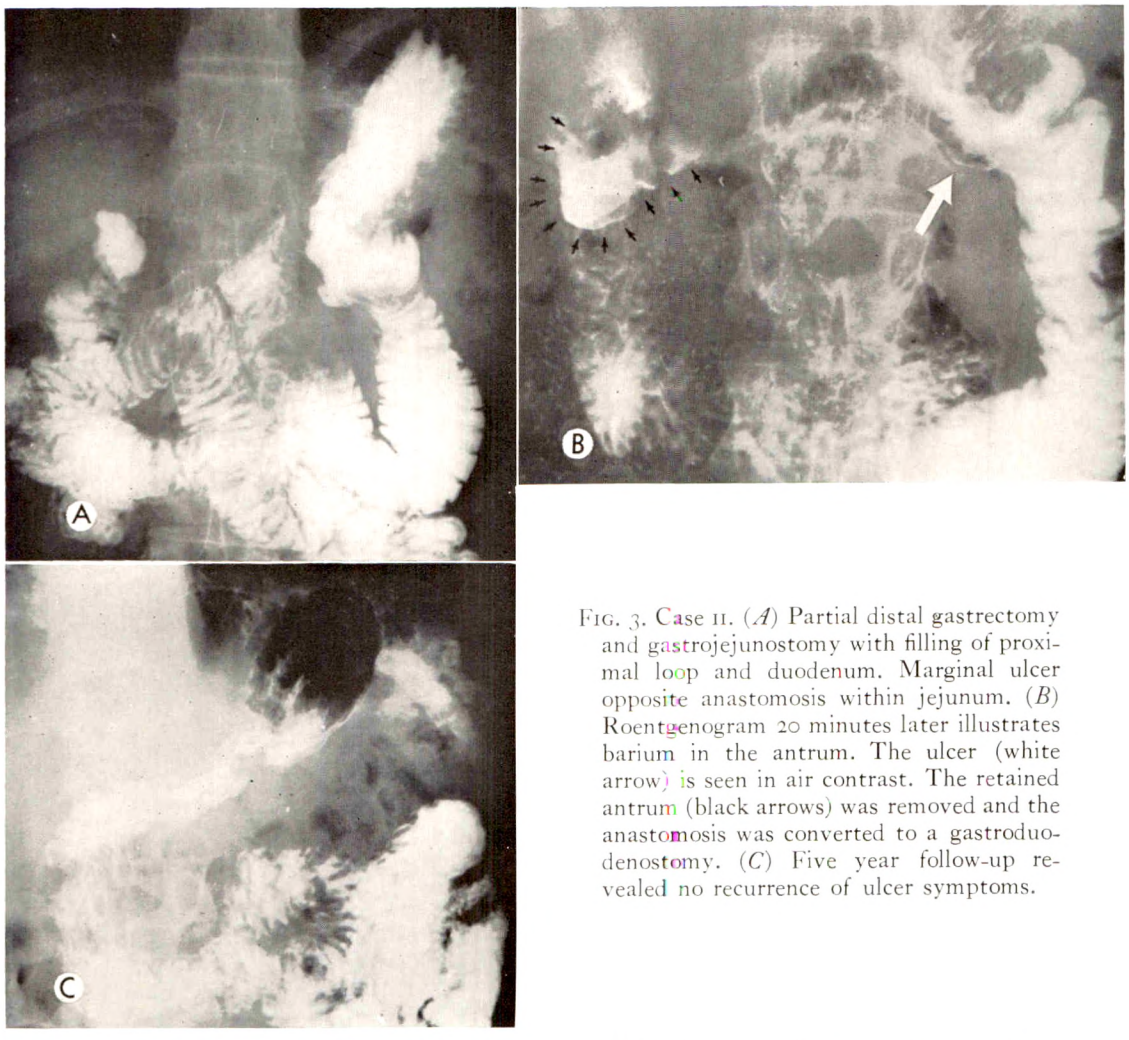



Fig. 3. Case II. (*A*) Partial distal gastrectomy and gastrojejunostomy with filling of proximal loop and duodenum. Marginal ulcer opposite anastomosis within jejunum. (*B*) Roentgenogram 20 minutes later illustrates barium in the antrum. The ulcer (white arrow) is seen in air contrast. The retained antrum (black arrows) was removed and the anastomosis was converted to a gastroduodenostomy. (*C*) Five year follow-up revealed no recurrence of ulcer symptoms.

several roentgen examinations. In 1957, a two-third gastrectomy had been performed for duodenal ulcer. The symptoms recurred 6 months following surgery. Medical therapy resulted in some relief of epigastric pain. Radiation therapy to the area of the gastric pouch was given in 1959 with delivery of 1,200 r to the skin. The symptoms, however, persisted and the patient was referred for surgical therapy.

Preoperative roentgen examination revealed a 2 cm. marginal ulcer in the jejunum adjacent to the gastrojejunostomy. Filling of the proximal jejunum and duodenum occurred with outlining of a deformed duodenal bulb (Fig. 3*A*). Reflux into a retained antrum was noted after the patient was 20 minutes in the right lateral decubitus position (Fig. 3*B*). A preoperative roentgenologic diagnosis of marginal ulcer and retained gastric antrum was made.

A 2 cm. penetrating marginal ulcer was found at exploration and 7 cm. of the retained gastric antrum was resected. The anastomosis was converted into a gastroduodenostomy (Fig. 3*C*).

The patient was reported well in 1965, except for occasional nausea.

Case III. P.B. A 58 year old female was referred in 1963 with sharp epigastric pain of 7 months' duration. Nine months previously, a Billroth II gastrojejunostomy had been performed for a duodenal ulcer history of 4 years. The patient had been admitted to four other hospitals since operation with 48 pounds weight loss, laboratory evidence of occult intestinal bleeding, and poor response to medical therapy. Twelve hour overnight secretions were measured and reported as 67 degrees.

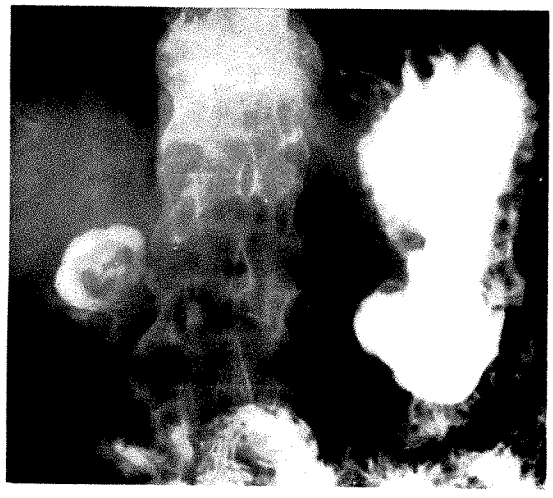


FIG. 4. Case III. Partial distal gastrectomy and gastrojejunostomy with giant marginal ulcer distal to the anastomosis. Barium reflux into duodenum shows prolapse of gastric mucosa into the duodenal bulb.

The preoperative roentgen examination showed a giant marginal ulcer. The clinical diagnosis of either a Zollinger-Ellison syndrome or a retained gastric antrum was entertained (Fig. 4).

An 8 cm. jejunal ulcer was found at operation extending to the anastomosis and a 9 cm. segment of retained gastric antrum was resected. A bilateral vagotomy was performed with revision of the gastrojejunostomy.

A 3 year follow-up examination revealed the patient asymptomatic since removal of the gastric antrum.

CASE IV. E.P. A 51 year old female was referred in 1965 with intractable epigastric pain and melena occurring 4 months after a gastrojejunostomy for duodenal ulcer.

Roentgenographic examination with filling of the proximal jejunum and deformed antrum demonstrated reflux into a retained antrum (Fig. 5*A*). A preoperative roentgen diagnosis of marginal ulceration and retained gastric antrum was made.

At exploration, 2 jejunal marginal ulcers were found measuring 1 cm. each in diameter and located in the jejunum opposite the stoma. A retained antrum measuring 10 cm. in length and 6 cm. in width was resected (Fig. 5*B*). Vagotomy and revision of the gastrojejunostomy were performed. Follow-up visits 11 months after surgery revealed no evidence of recurrence.

## DISCUSSION

It is not known which cells in the antrum produce the hormone gastrin;[14] however, its effect on the parietal cells in the body and fundus of the stomach is well understood and supported by experimental work.[23] Ulcerogenesis due to gastrin production

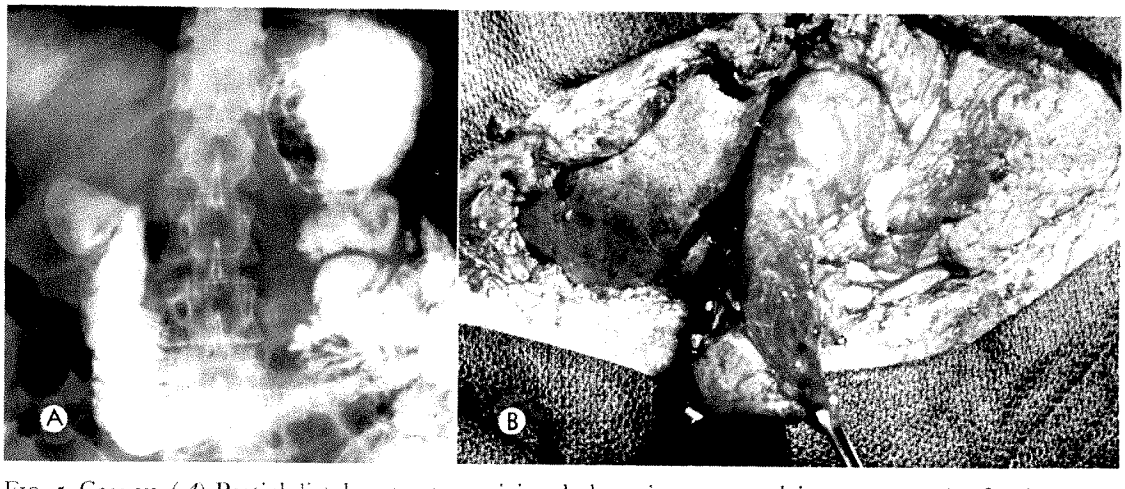



FIG. 5. Case IV. (*A*) Partial distal gastrectomy, jejunal ulceration at gastrojejunostomy and reflux into duodenum and antrum. Note the position of the antrum to the right of the scarred duodenal bulb. (*B*) Findings at operation. The descending duodenum adjacent to the liver edge is inverted. The pouch demonstrated at the end of the mobilized duodenum and to the right was found to represent retained gastric antrum when examined histologically.

from a retained antrum occurs particularly if the continuity of fundus and antrum has been surgically disrupted. The retained antrum in these cases is in an alkaline environment, is uninhibited and continues to release excessive amounts of gastrin with the result of a high incidence of ulcer recurrence. The mechanism, therefore, is similar to the hormonal effect produced by ulcerogenic tumors of the pancreas in the Zollinger-Ellison syndrome[8] or in cases of polyglandular adenomas. In 1960, Gregory and his co-workers[12] extracted a gastrin-like substance from the pancreatic tumor in a case of Zollinger-Ellison syndrome. The same authors prepared pure gastrin from the antral mucosa in 1964.[13] Both the syndromes of ulcerogenic tumor and of retained gastric antrum, therefore, have to be included in the differential diagnosis of marginal ulcers.

In the intact stomach, gastric acid in the antrum appears to have an absolute inhibitory effect on gastrin production if the pH value reaches between 1.5 to 2.0.[4,29] After gastric resection, only alkaline solutions bathe the retained antrum via the pylorus. The reflux phenomenon is roentgenographically illustrated with barium. Only 2 such examples were found in the literature.[2,7] However, there is no illustration and no discussion of the syndrome of the retained gastric antrum available in the radiologic literature.

The importance of *the preoperative diagnosis* of a retained gastric antrum becomes apparent when we realize that surgical therapy for marginal ulcer consists of vagotomy and/or revision of the gastrojejunostomy. Since the region of the duodenal bulb, pylorus, and possible retained antrum in these instances is not routinely explored, and if the diagnosis of a retained gastric antrum is not entertained before operation, this entity may be overlooked at re-exploration. The antrum may again be left in place with further ulcer recurrence. The exception would be the case of a retained gastric antrum after total gastrectomy (Fig. 6). Similar to the Zollinger-Ellison syndrome, no ulcer recurrence is expected if all parietal cells have been removed.[34]

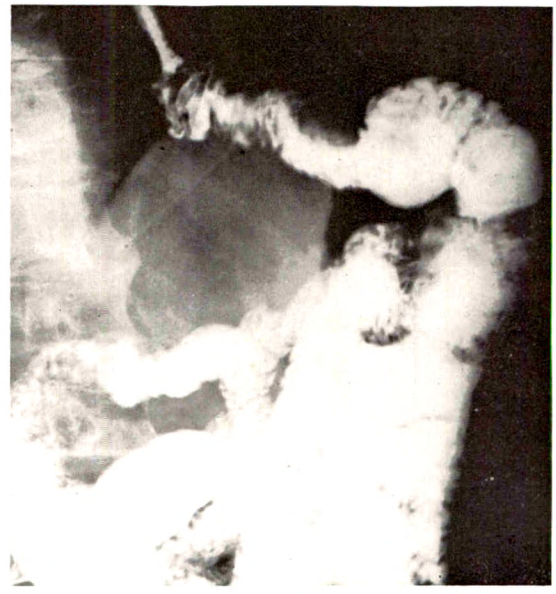


Fig. 6. The roentgenographic demonstration of a retained gastric antrum after total gastrectomy is of no significance since the acid-bearing portion of the stomach has been removed.

*The extent of the gastric antrum* and gastrin-producing cells is not clearly defined in the literature. Jenkinson[16] stated that the antrum extends from the incisura angularis to the pylorus. Bergström and Broomé[3] used litmus powder to determine the boundary between the gastric antrum and fundus in 28 patients. They found a slight individual variation in the position of the boundary and felt that resection in radical antrectomy is frequently more extensive than necessary. On the other hand, Ruding and Hirdes[27] examined the extent of the antrum histologically in 13 stomachs and found that at least 80 per cent of the lesser curvature must be resected in order for the surgeon to be reasonably certain that no antral mucosa is left *in situ*. Furthermore, the antral mucosa may extend beyond the pylorus muscle by 0.5 cm. distally into the base of the duodenal bulb. Grossman,[14] therefore, prefers the term "pyloric

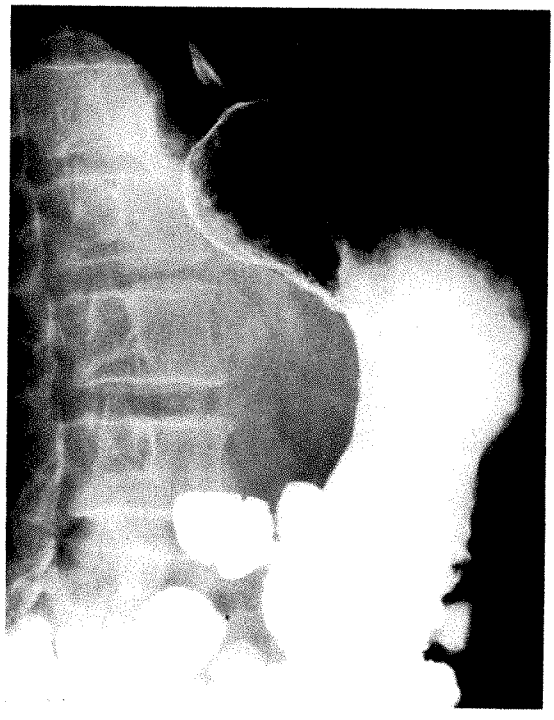


FIG. 7. Transsection of the stomach at the pylorus and gastroenterostomy. A circular fold is often present and helps to identify the antrum.

gland area" to the term gastric antrum. The average area occupied by pyloric glands represents 14 per cent of the total mucosal area of the stomach according to Landboe-Christensen.[19] The individual variation in the boundary between fundic and pyloric glands is described by Oi and Sakurai.[25] It was found from 2 to 14 cm. from the pylorus.

*The roentgenologic technique* of filling of the proximal jejunum and duodenum becomes important if a retained gastric antrum is suspected from the history or from the roentgen findings. The radiologist should be suspicious if a giant marginal ulcer is present, if several marginal ulcers are seen, if there is a large amount of retained secretion in the stomach, if the mucosa of the jejunum in the anastomotic loop and proximal portion of the small bowel is edematous, or if a gastrojejunocolic fistula has developed. Cases of retained gastric antrum usually give a history of marginal ulceration that is quite stubborn to medical therapy. This is particularly true in cases where irradiation for reduction of gastric acidity showed poor results (Case II). Marginal ulcers due to ulcerogenic tumors similarly show poor response to radiation therapy, whereas Kiefer and Smedal[18] reported good results for stomal ulcers after conventional gastrectomy.

Filling of the proximal jejunum and duodenum is best accomplished by manual obstruction of the distal or efferent loop. This may not be successful in all cases. We have found flooding of the gastric pouch with a large amount of barium helpful during this maneuver. Even with filling of the jejunum and duodenum, the barium may not reflux into the gastric antrum. In one of our cases, positioning in the right decubitus after proximal loop filling was successful in demonstrating the antrum with barium (Fig. 3*B*).

The *direct roentgenologic sign* for a retained antrum is reflux of barium from the duodenum via the pylorus into the antrum. The radiologist must realize that the retained antrum may not occupy its original location to the left of the pylorus. As is demonstrated in Cases II and IV (Fig. 3*B*; and 5, *A* and *B*), the antral pouch may be superimposed on the duodenum or to the right of it. The antrum probably falls into this position after it is transsected from the remainder of the stomach. Roentgenographic demonstration of gastric mucosa is helpful in identifying the antrum, particularly if a circular antral fold is visualized (Fig. 7).

An *indirect roentgenologic sign* of a retained gastric antrum was present in Case III (Fig. 4). The characteristic roentgenographic appearance for prolapse of gastric mucosa into the base of the duodenal bulb may be seen both in the lateral and axial projections. It is suspicious of a retained antrum. The appearance of a surgically inverted duodenal stump usually gives a different roentgenographic appearance, with asymmetry and shortening of the deformed duodenal bulb.

In the *differential diagnosis* of the retained gastric antrum (Table 1), the radiologist may be mislead by a barium-filled duodenal diverticulum in the area of the duodenal bulb. The presence of additional diverticula in the duodenum or jejunum is helpful in differentiating these 2 conditions (Fig. 8). Furthermore, the retained antrum may exhibit circular mucosal folds (Fig. 3*B*).

The similarity of the mechanism of ulcerogenesis involved in the retained gastric antrum and in ulcerogenic tumors has been discussed. Scobie *et al.*[28] reported a case of excluded gastric antrum simulating the Zollinger-Ellison syndrome. Both conditions may present to the radiologist with marginal ulcers. His index of suspicion should be high if the recurrent ulcers are large, multiple (Fig. 9), or unusual in location.

If the gastric antrum is diseased and if surgical removal of the antrum is difficult, the surgeon may elect to leave the wall of the antrum in place and shell out the gastric mucosa. This procedure was described by Bancroft[15] and represents in effect the surgical removal of the endocrine gland of the antrum. The radiologist may be misled after reflux into a retained antrum in these cases.

The surgeon may also decide to remove the gastric antrum at a second operation according to McKittrick *et al.*,[20] if removal of a diseased antrum would be difficult at the time of initial surgery.

Garnjobst[11] reviewed 24 cases of prepyloric duplications of the antrum. He found intractable duodenal ulcer disease associated in two instances with cystic duplication lined with well developed antral mucosa. A causal relationship was suggested on the basis of antral hyperfunction.

Bioassay for gastrin is a promising development in the management of ulcerogenic tumors. The same technique may be of future use in the diagnosis of the retained antrum.

TABLE I

ROENTGENOLOGIC DIFFERENTIAL DIAGNOSIS OF THE RETAINED GASTRIC ANTRUM

1. Filling of duodenal diverticulum
2. The Zollinger-Ellison syndrome
3. Polyglandular adenomas
4. Retained gastric antrum in Bancroft procedure
5. McKissock two-stage antral removal
6. Duplication of the antrum

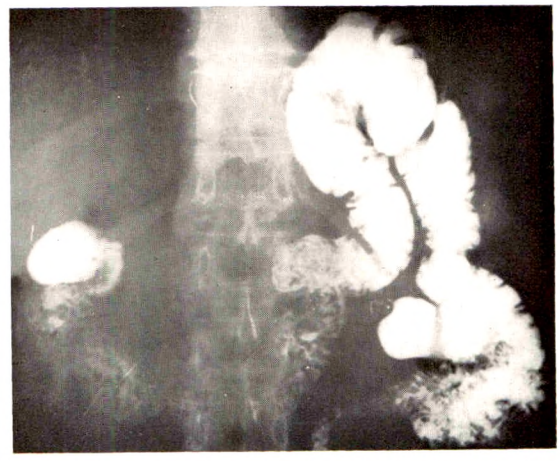


FIG. 8. A duodenal diverticulum in the first portion of the duodenum may be misinterpreted as a retained antrum. The presence of an additional diverticulum in the jejunum is of diagnostic help in the case illustrated.

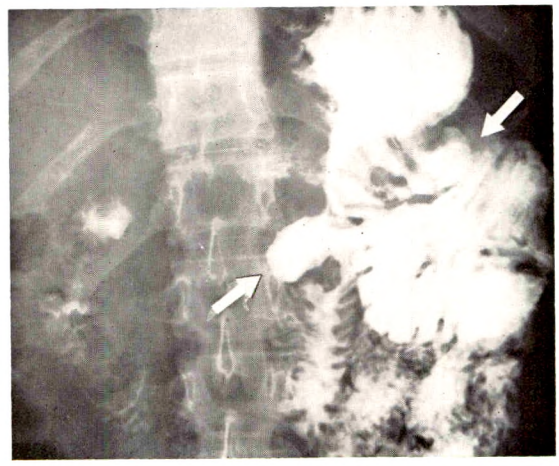


FIG. 9. The mechanism of recurrent ulcerogenesis is similar for both the syndrome of the retained gastric antrum and ulcerogenic tumors. The radiologist's index of suspicion should be high if the marginal ulcers (arrows) are very large, unusual in location, or multiple as in this case of Zollinger-Ellison syndrome.

SUMMARY

Surgical retention of the endocrinologically active gastric antrum results in a high incidence of recurrent ulcer disease after partial gastrectomy.

This introduction of the iatrogenic syndrome of the retained gastric antrum to the radiologic literature includes the report of 4 cases. Symptoms of ulcer recurrence had occurred within 2 to 5 months following surgery. All 4 patients are symptom free after resection of a retained antrum with follow-up ranging from 11 months to 5 years.

The roentgenologic technique and diagnosis of the retained gastric antrum are discussed with differential considerations. The importance of the roentgenologic diagnosis before re-exploration is stressed.

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# ENHANCED VISUALIZATION OF ESOPHAGEAL VARICES BY DEXTRAN INFUSION*

## A PRELIMINARY REPORT

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VISUALIZATION of small esophageal varices on roentgenographic examination with barium may be difficult.[8] Successful visualization varies from 20 per cent[2] to 70 per cent.[7] The true rate of failure is not known because of difficulties also inherent in esophagoscopy.

The difficulties in the roentgenologic diagnosis are mainly related to the small caliber of the submucosally situated varices, their easy compressibility, and their differentiation from mucosal folds. These difficulties may be compounded if the patient is hypovolemic from hemorrhage, or unable to cooperate fully during the roentgenographic procedure.

Large esophageal varices are easy to detect. Towards this end, the Valsalva maneuver has been used to achieve maximum varix distention. Often, coordination of this maneuver with the other esophageal events is not satisfactory.

A more accurate roentgenologic diagnosis might be anticipated if the varices could be temporarily distended without hazard. A possible method of accomplishing such a distention, based on a rapid increase in blood volume, seemed worthy of investigation.

### HEMODYNAMIC PRINCIPLES

A rapid infusion of dextran in cirrhotics was reported to have elevated portal venous pressures by 120 mm. of water, while elevation of central venous pressure was only about half of this amount.[1] Elevation will persist, somewhat, for about 1 hour after the infusion of 1,000 cc. of 6 per cent dextran.

It is hypothesized that the increased systemic portal venous gradient will increase flow through already functioning collateral channels, such as the esophageal veins. If resultant flow increases the size of these distensible veins, it should aid their roentgenologic detection. Distention of the esophageal veins would seem to entail the risk of rupture. Except in patients with ascites, however, blood volume expansion has not initiated hemorrhage from varices.[3,4,6,9]

### MATERIAL AND METHODS

Fourteen men with the clinical diagnosis of cirrhosis of the liver were selected. Patients with a history of ascites were not included. Seven of the 14 patients had had gastrointestinal bleeding, the source of which had not been determined. The esophagus of all 14 had been examined roentgenologically without varices having been detected.

An infusion of 1,000 cc. of 6 per cent dextran in physiologic saline was introduced into the antecubital vein with an 18 gauge needle in 30 to 40 minutes. Careful observation was maintained during this time for signs of adverse cardiorespiratory changes. As a precaution, 2 units of blood were available for each patient.

Spot roentgenograms of the esophagus after ingestion of thick and thin barium suspension were obtained before and immediately after the dextran infusion.

### RESULTS

No ill effects were apparent from the rapid infusion of dextran; in particular,

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no varices ruptured nor were there signs of circulatory overload.

The roentgenograms of all 14 patients gave no evidence of varices on the initial ingestion of barium. After dextran infusion, the barium examination revealed varices in 7 of the patients. Figures 1 and 2 show the appearance of the esophagus before and after dextran infusion in 1 of the patients.

The roentgenograms of these 7 patients were reviewed by 8 radiologists who were not informed of their sequence. In general, their opinion confirmed that the yield of varices was greater in those roentgenograms obtained after dextran infusion (Table 1). Gastric varices were not sought. In particular, no radiologist stated that varices were present in the roentgenograms before dextran but absent on those after dextran infusion. Of these 112 assessments (2 examinations in each of 7 patients by 8 radiologists), 7 were "indeterminate for presence of varices." These all related to the pre-dextran roentgenograms and are classified as negative in Table 1. In each



Fig. 1. Esophagram. Equivocal evidence of varices.

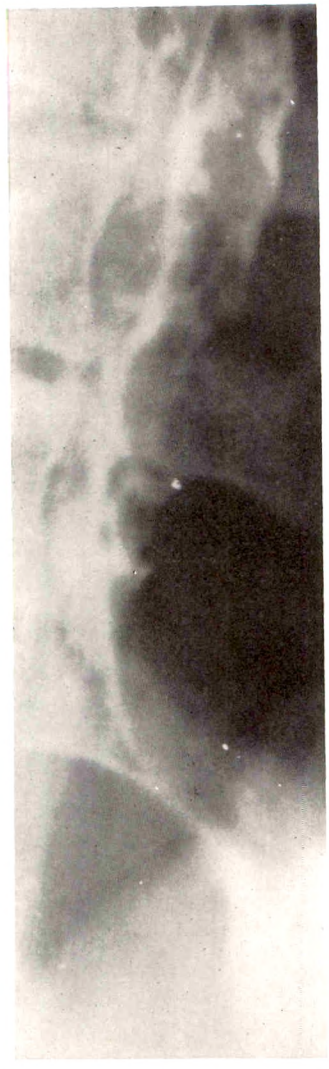


Fig. 2. Esophagram. Enhanced visualization of varices after infusion of 1,000 cc. of 6 per cent dextran.

TABLE I

RESULTS OF ROENTGENOGRAPHIC REVIEW BY 8 RADIOLOGISTS

| Patient No. | History of Gastrointestinal Bleeding | Liver Biopsy | Varices on Endoscopy | Pre-Dextran | | Post-Dextran | |
|---|---|---|---|---|---|---|---|
| | | | | No Varices | Varices | No Varices | Varices |
| 1 | + | N.P. | N.P. | 8 | 0 | 0 | 8 |
| 2 | + | N.P. | + | 8 | 0 | 0 | 8 |
| 3 | + | N.P. | + | 6 | 2 | 1 | 7 |
| 4 | + | N.P. | + | 8 | 0 | 1 | 7 |
| 5 | + | Cirrhosis | + | 6 | 2 | 1 | 7 |
| 6 | — | N.P. | + | 8 | 0 | 6 | 2 |
| 7 | — | Cirrhosis | N.P. | 8 | 0 | 4 | 4 |

N.P. = Not performed.

indeterminate case the assessor considered varices to be present after the dextran infusion.

## DISCUSSION

That an increased splanchnic blood volume will lead to distention of esophageal varices that may be present in patients with cirrhosis, appears logical. The application of this postulate on a clinical level has given encouraging preliminary results. A by-product of this work has been the realization that roentgenologic examination of the hypovolemic patient for esophageal varices may be expected to be highly inaccurate.

Previous animal experiments[5] suggest that a similar gradient can be induced if transfusions in hypovolemic dogs are given rapidly. None of our patients was hypovolemic or in shock. After reconstitution of blood volume in patients with bleeding varices, more varices should be detected roentgenographically.

Many examinations will need to be conducted to confirm the value of induced portal venous hypertension in the diagnosis of esophageal varices. At present, ascites, rare blood groups, severe cross-matching problems, congestive heart failure, and significant secondary aldosteronism constitute precautionary exclusions from this procedure.

A patient whose esophageal varices are easily identifiable need not be subjected to this procedure. Its eventual usefulness in patients with cirrhosis and unexplained gastrointestinal bleeding, however, may lead to greater diagnostic accuracy.

This preliminary report may encourage others to independently assess this method.

## SUMMARY

Preliminary observations suggest that esophageal varices become more prominent after blood volume expansion with 1,000 cc. of 6 per cent dextran.

The technique is not suitable for patients with ascites.

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# LEIOMYOMA OF THE ESOPHAGUS PRESENTING AS A MEDIASTINAL MASS*

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LEIOMYOMA of the esophagus is a rare lesion. The first recorded case was described by Sussius in 1559.[5] The tumor in this patient filled the lower third of the esophagus and caused death by suffocation. The pathologic features of leiomyomata were described by Virchow in 1867.[20]

Harrington and Moersch[10] reviewed 7,450 postmortem examinations, dating from 1933 to 1944; only 32 leiomyomata were found. Daniel and Williams[4] in 1950 did not find one example in 4,000 autopsies at the Vanderbilt Hospital. In 1954 Sweet and his associates[19] observed 4 cases from 13,000 necropsies. Johnston and Clagett[11] reported a total of 18 leiomyomata at the Mayo Clinic from 1920–1949. Schafer and Kittle[15] in 1947 found 11 benign esophageal tumors in 6,001 autopsies at the University of Chicago.

The first surgical removal of a leiomyoma was reported by Sauerbruch in 1932 employing the transpleural route for a lesion of the lower third. Oksawa repeated this feat in 1933. Both results were reported as satisfactory.[6]

## CLINICAL FEATURES

### AGE AND SEX DISTRIBUTION

There have been no reported cases below the age of 10 years and the lesion is considered extremely rare below the age of 20 years.[2] The oldest patient was 70 years of age.[6] Ninety per cent of all reported leiomyomata of the esophagus occurred between the ages of 20 and 50 years.[7] The operative peak incidence is the 4th decade.[2] The lesion is found more frequently in males, 1.8:1,[6] and is rare in Negroes.[8]

### LOCATION

Leiomyomata are least common in the upper third of the esophagus and in that location are almost entirely confined to males. Seven per cent of leiomyomata of the esophagus are found in the upper third; 34.8 per cent in the middle third; and 55.2 per cent in the lower third. Nearly 10 per cent of the latter involved the stomach.[7] Other investigators have confirmed this distribution.[14,18]

### SIGNS AND SYMPTOMS

Leiomyomata run the gamut from asymptomatic to highly symptomatic lesions. The most frequent combination of symp-

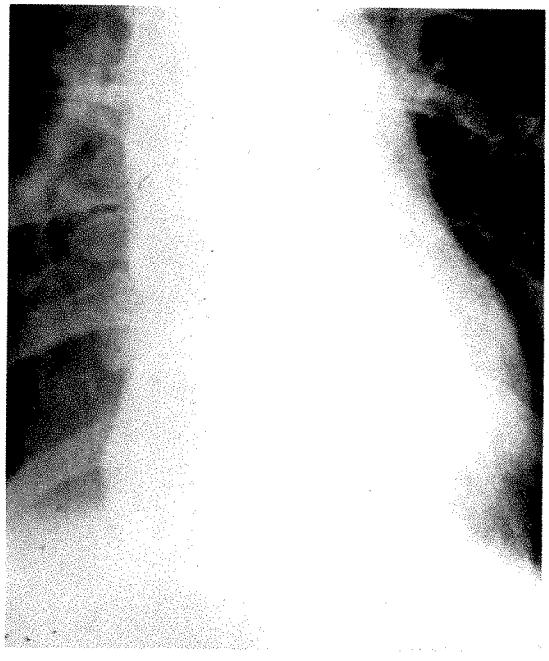


FIG. 1. Case 1. Posteroanterior chest roentgenogram. Note the soft tissue mass in the right cardiophrenic angle.

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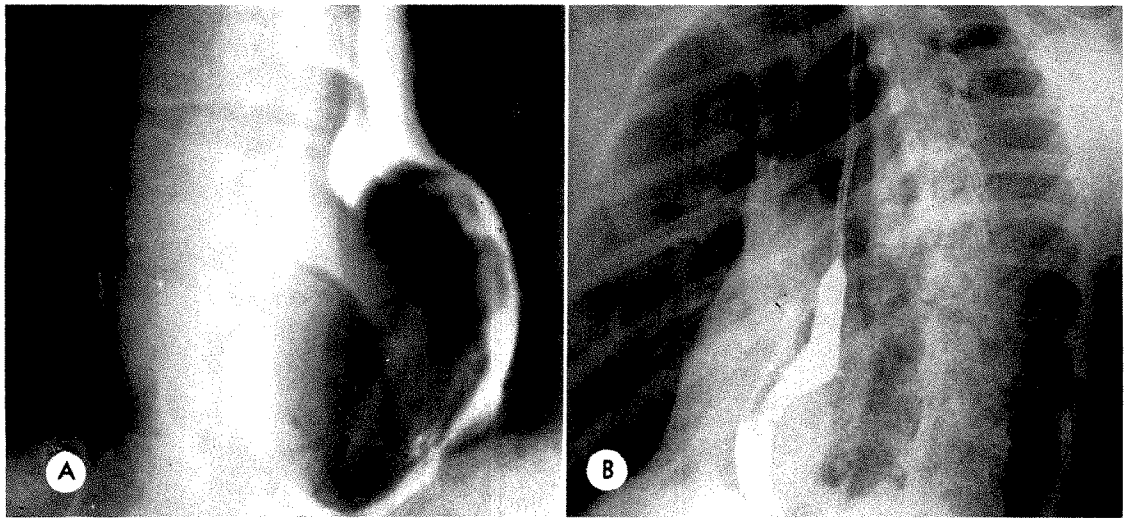



FIG. 2. (*A* and *B*) Barium swallow study. Note the sharply defined filling defect in the lower third of the esophagus. The upper and lower margins have acute angles demonstrating the typical sulci.

toms is dysphagia and pain. The only other symptoms often occurring together are weight loss with pain in 11.9 per cent of cases and weight loss with dysphagia in 11.3 per cent.[6] Bleeding resulting from a smooth muscle tumor in the esophagus is rare; there are only 5 reported cases in the literature.[1] Other symptoms worthy of note are a feeling of fullness, passive regurgitation and dyspnea. The clinical symptoms as listed by Harper and Tiscenco[9] are: (a) intermittent dull retrosternal pain or pressure sensation; (b) epigastric pain with no constant relation to eating; (c) anorexia; (d) dysphagia—often intermittent; (e) occasional, and/or copious vomiting due to esophageal dilatation; and (f) weight loss.

## PATHOLOGY

The tumor may arise from the circular and longitudinal muscle coats, muscularis

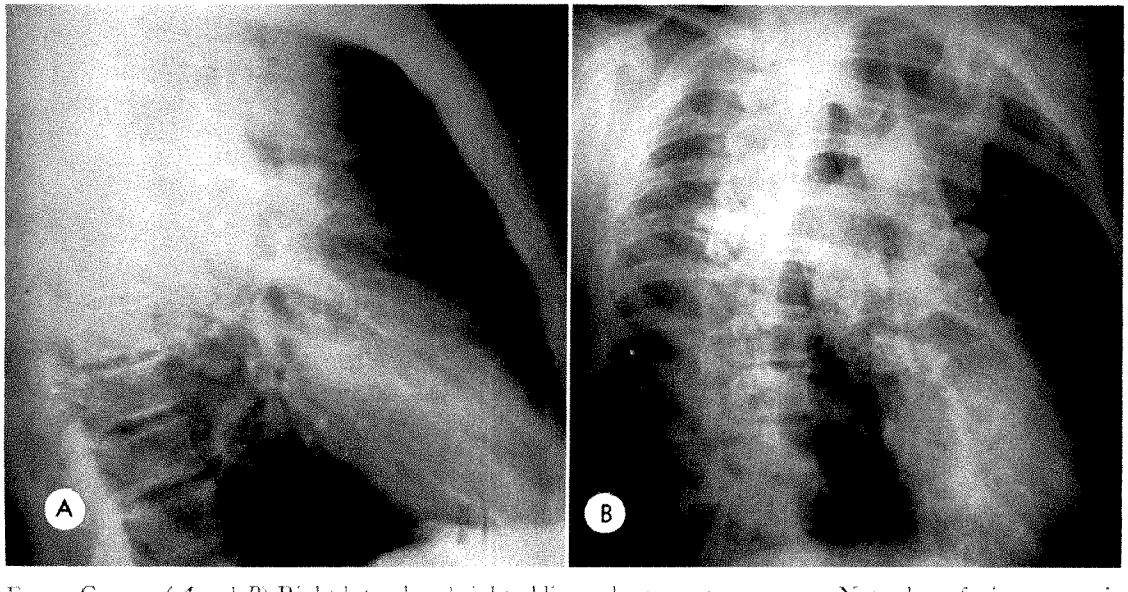



FIG. 3. Case II. (*A* and *B*) Right lateral and right oblique chest roentgenograms. Note the soft tissue mass in the mediastinum displacing the trachea anteriorly.

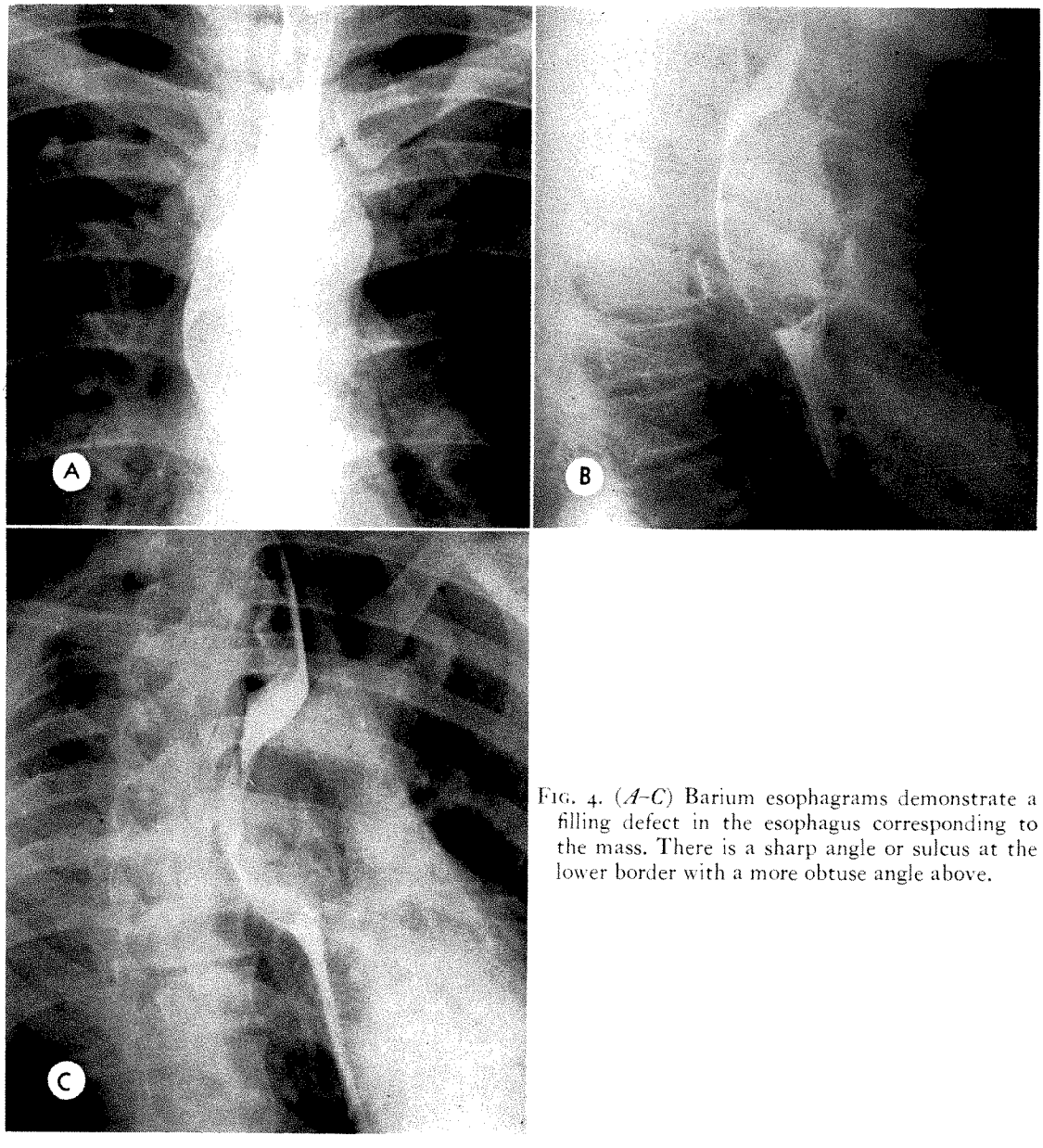



FIG. 4. (*A–C*) Barium esophagrams demonstrate a filling defect in the esophagus corresponding to the mass. There is a sharp angle or sulcus at the lower border with a more obtuse angle above.

mucosae or from smooth muscle in the walls of the esophageal arteries. Calcification may occur in leiomyomata;[17] ulceration is rare.[18]

The tumors are sharply demarcated from the surrounding tissue. The fibers are arranged in whorls separated by connective tissue septa. There are variable amounts of smooth muscle and connective tissue elements. Hyaline and mucoid degeneration is common.[3]

The growth characteristics[1,12,13] are as follows:

(1) Leiomyomata may grow into the lumen and present with signs and symptoms indistinguishable from a mucosal lesion.

(2) The expanding intramural tumor may become horseshoe or annular in shape. This type can easily be mistaken for a napkin ring deformity seen in adenocarcinoma of the gastrointestinal tract.

(3) An elongated pedunculated tumor has been described and is polypoid in nature.

(4) The solitary and eccentrically growing leiomyoma is the variety to be discussed in this presentation. It is usually symptomless and thus grows to large proportions. It can be seen on routine chest roentgenograms. Nine of 315 leiomyomata from 1559 to 1959 were noted as accidental discoveries on routine chest roentgenograms.[6]

## REPORT OF CASES

Case I. E.L.B., a 22 year old white male was admitted to Fitzsimons General Hospital June 18, 1952 with a one year history of burning epigastric and retrosternal pain, during and shortly after meals, and occasional eructation of food. He had been treated with antacids and antispasmodics with no results, and expressed an intolerance for acid foods.

The posteroanterior chest roentgenogram (Fig. 1) revealed a soft tissue density mass behind the heart at the right cardiophrenic angle. Barium swallow study (Fig. 2, *A* and *B*) showed a filling defect in the distal third of the esophagus which was sharply defined, oval, non-obstructing, and had acute angles at its upper and lower margins. The roentgenologic diagnosis was benign intramural tumor of the esophagus.

At operation there was a large irregular mass in the esophageal wall, extending from the esophageal hiatus upward a distance of 8 cm. The overlying mucosa and musculature were thinned out over the tumor and the mass had many friable projections. Due to the tumor size, resection of the distal esophagus was necessary.

The pathologic diagnosis was intramural leiomyoma of the esophagus.

Case II. L.R., a 31 year old white male was admitted to Tokyo Army Hospital subsequent to an allergic drug reaction, and was found to have clubbing of the fingers. During a pulmonary work-up, he was found to have a mediastinal mass displacing the trachea. He then gave a history of dysphagia, necessitating swallowing two or three times in rapid succession, on occasion, to force food into his stomach. There was no pain, but he had a mild productive cough. Clinical impression at that time was lymphoma.

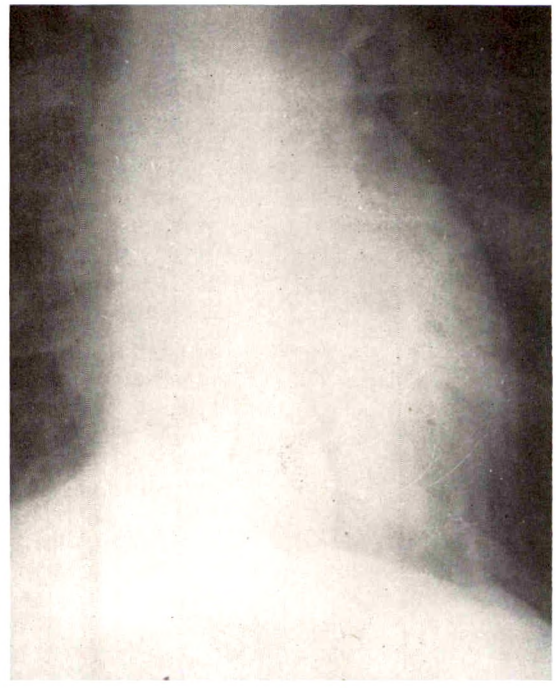


Fig. 5. Case III. Chest roentgenogram. There is a soft tissue density in the retrocardiac area projected lateral to the left spinal border.

At Fitzsimons General Hospital in May 1953, plain chest roentgenograms (Fig. 3, *A* and *B*) showed a soft tissue mass in the mediastinum, pushing the trachea anteriorly. Esophagrams (Fig. 4, *A–C*) demonstrated a filling defect in the esophagus corresponding to the mass. This middle third mass was sharply outlined, oval, and had a sharp angle at the lower border, with a more obtuse angle above. It did not obstruct the flow of barium. The diagnosis was intramural, submucosal tumor of the esophagus.

At operation there was a spindle-shaped intramural tumor at the junction of the upper and middle thirds of the esophagus. The tumor was shelled out, measuring 7.2×4.3×3.4 cm. and weighing 62 gm. Microscopic study revealed a leiomyoma.

Case III. D.S., a 22 year old white male was sent from Viet Nam because of neuropsychiatric complaints and for evaluation of a mass detected on a survey chest roentgenogram. He had no dysphagia, chest pain, or other symp-

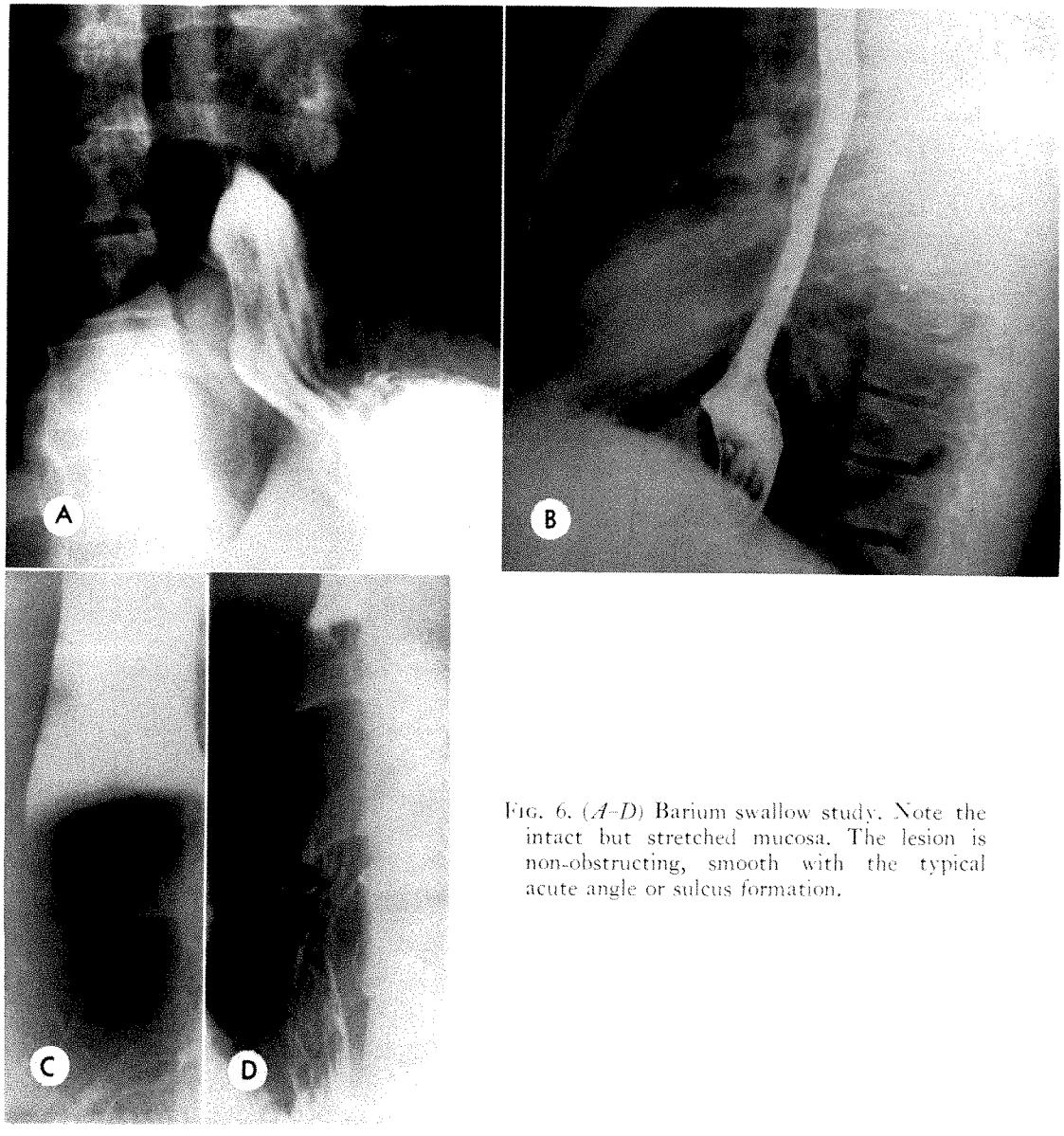



FIG. 6. (*A–D*) Barium swallow study. Note the intact but stretched mucosa. The lesion is non-obstructing, smooth with the typical acute angle or sulcus formation.

toms related to the chest. Gastroesophagoscopy was normal.

The plain chest roentgenogram (Fig. 5) revealed a soft tissue density mass behind the heart projected just lateral to the left spinal border. Barium swallow study (Fig. 6, *A–D*) showed the mass to involve the esophagus, producing a smooth, non-obstructing, convex filling defect. The upper border was outlined by a relatively acute angle, and intact, but stretched, mucosa was present over the mass. The roentgenologic diagnosis was intramural esophageal tumor, probably a leiomyoma.

Surgery was not recommended for this patient, and no microscopic study was obtained.

CASE IV. C.R., a 42 year old white male was admitted to Fitzsimons General Hospital January 29, 1966 for repair of bilateral inguinal hernias. He gave a history of a prior chest roentgenographic examination which demonstrated a right hilar mass. He had no dysphagia, chest pain, or other chest symptoms. Laboratory studies revealed an erythrocytosis.

Chest roentgenograms (Fig. 7, *A–C*; and 8) demonstrated a mediastinal mass displacing

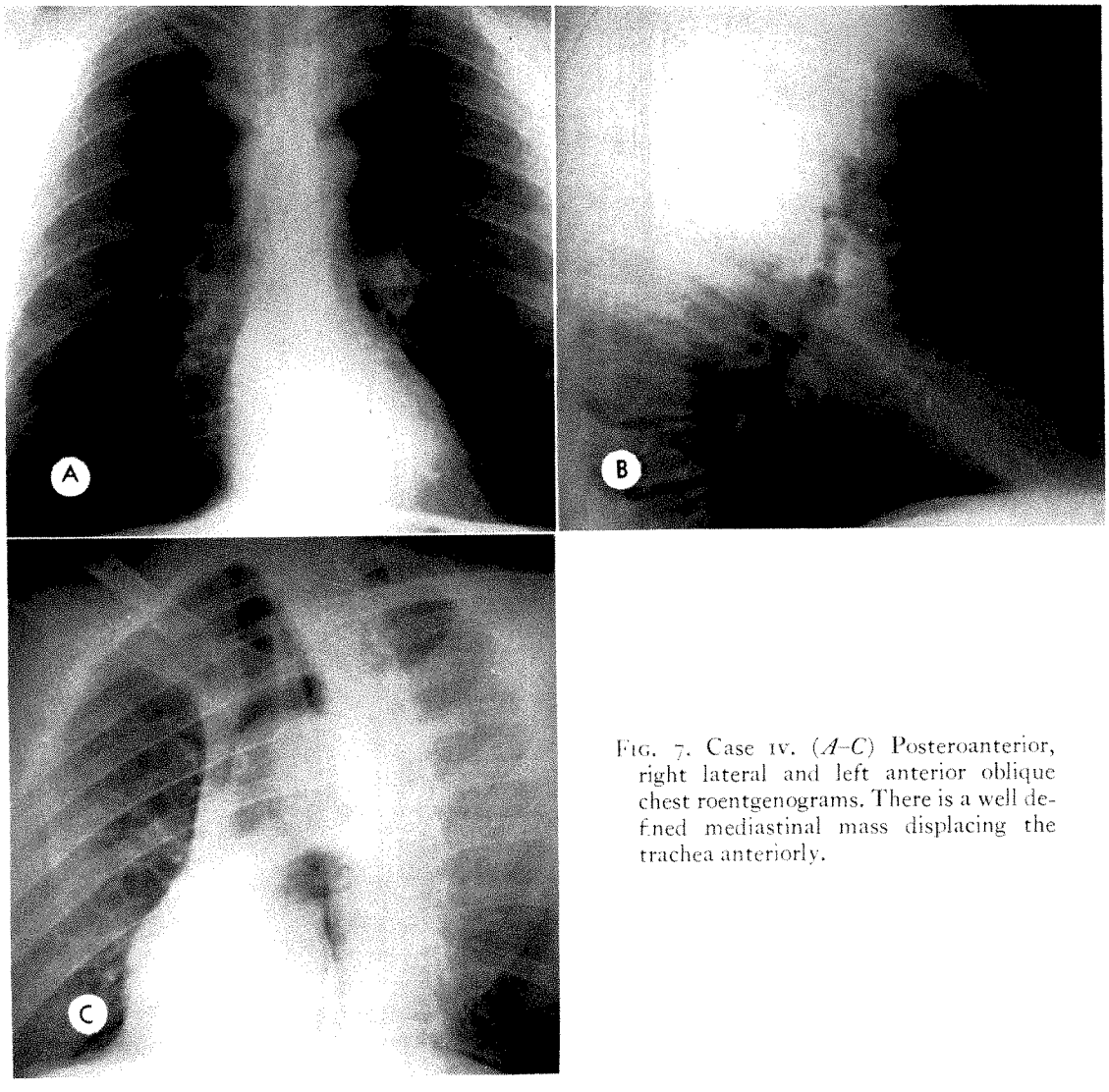



Fig. 7. Case IV. (*A–C*) Posteroanterior, right lateral and left anterior oblique chest roentgenograms. There is a well defined mediastinal mass displacing the trachea anteriorly.

the aortic knob to the left, and the trachea anteriorly. The mass was not calcified, and had smooth contours. On barium swallow study (Fig. 9, *A* and *B*) there was a tapered, smooth, eccentric narrowing of the esophagus, with a sharp upper angle shown in one view. The esophagus was distensible, with no significant obstruction. The impression was a benign esophageal intramural tumor.

Surgery revealed a firm middle third mass overlaid by muscle layers. The mass encircled the esophagus, but was entirely intramural; it was removed without entering the mucosa. The pathologic diagnosis was benign leiomyoma.

Case V. C., a 33 year old male had a 2 year history of dysphagia, described as a feeling that food was sticking in the esophagus. There was also a sensation of postprandial pressure in the mid-chest. These difficulties were worse with solid foods.

Plain chest roentgenograms (Fig. 10, *A* and *B*) showed a mass posterior to the heart to the left of the spine. It was smooth and well demarcated. The barium swallow study (Fig. 11, *A–D*) proved the mass to be an esophageal tumor; oval, smooth, with sharp borders. The tumor involved one side of the esophagus and produced no mucosal distortion. The diagnosis was leiomyoma of the esophagus.

At operation an intramural tumor was removed which proved to be a leiomyoma.

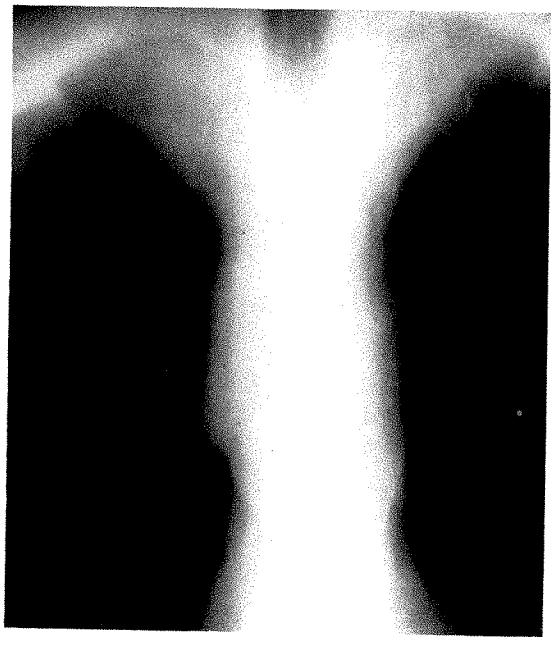


FIG. 8. Tomographic study of upper mediastinum. The mass is well seen with a smooth contour and no calcification.

## DISCUSSION

Although Schatzki and Howes[16] found only 4 reported cases of intramural tumors of the esophagus examined roentgenologically prior to 1942, many have been reported since that time. The majority of these intramural tumors were leiomyomata, and the roentgenographic characteristics have been well described. The presentation of these tumors as masses on plain chest roentgenograms, however, is unusual.[8] It is this feature which makes these cases of particular interest.

The roentgenographic features of intramural tumor, as described by Schatzki and Howes,[16] are:

(a) Mucosal folds visible and normal

(b) Upper and/or lower margin of filling defect forms a sharp angle with the uninvolved esophagus

(c) Mass moves with the esophagus on breathing or swallowing

(d) Esophagus not rigid

(e) Mass sharply outlined

(f) Esophagus may be narrowed in one plane, but stretched and widened in the opposite diameter.

The importance of correlating the soft tissue mass with the location of the esophageal filling defect should be stressed.[9] The column of barium may assume a "forked" appearance as it encounters the mass protruding into the lumen.[8] Distensibility of

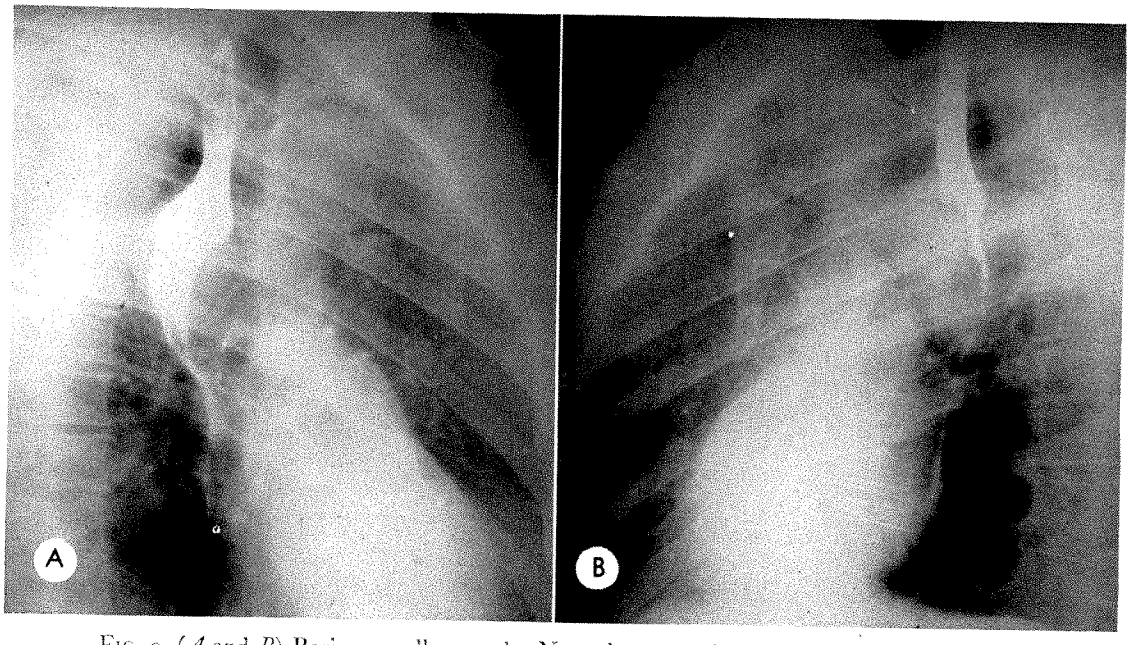



FIG. 9. (*A* and *B*) Barium swallow study. Note the eccentric narrowing of the esophagus with a sharp upper angle.

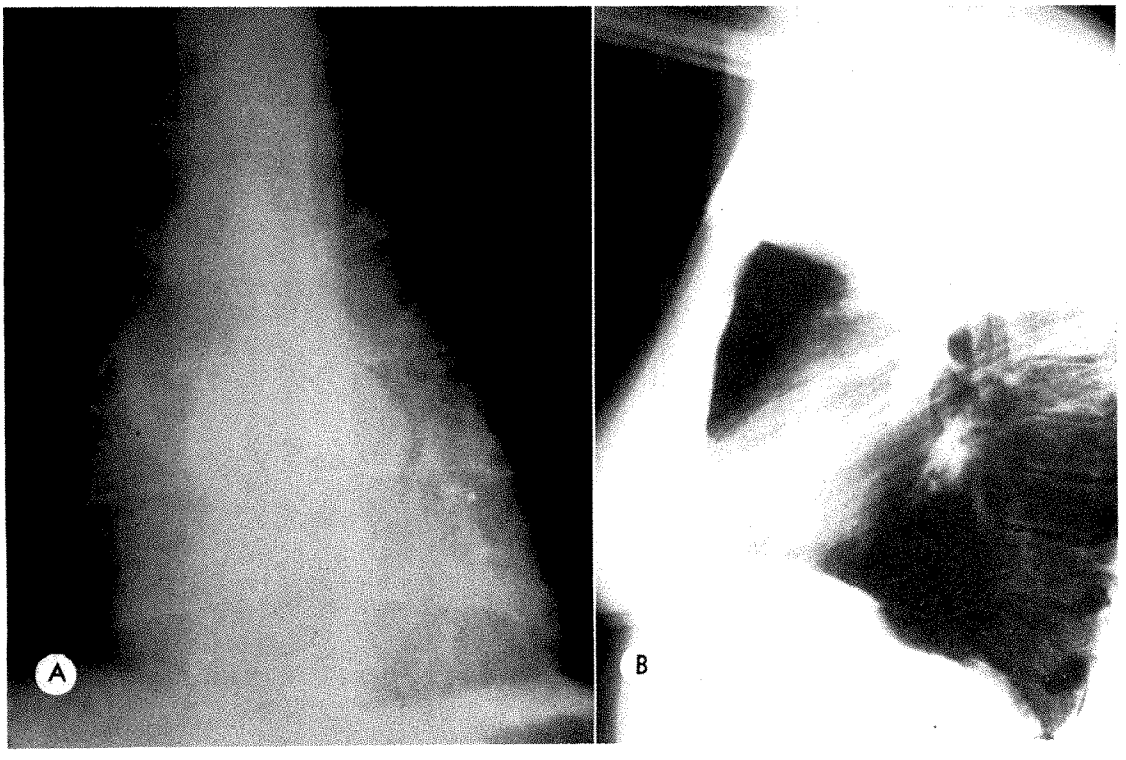



FIG. 10. Case v. (*A* and *B*) Posteroanterior and left lateral chest roentgenograms. Note the well defined left retrocardiac density.

the esophagus is another important feature to be observed at fluoroscopy.

The greater mass of the tumor may protrude toward the outer wall of the esophagus, producing an "iceberg" phenomenon. The tumors are also occasionally multiple. The highest incidence is in the lower third with the middle third a near second. Only a small per cent occur in the cervical esophagus, presumably due to its smaller amount of smooth muscle.[2]

All of the cases presented demonstrate many of the typical roentgenographic findings, in addition to the rather unusual feature of presenting as a mass on chest roentgenograms made without barium in the esophagus.

SUMMARY

Five cases of leiomyomata of the esophagus presenting as mediastinal masses are described. The barium swallow study is critical in making the diagnosis; the roentgenographic features are typical.

The presentation of leiomyomata of the esophagus as mediastinal masses is unusual, but should be considered in any differential diagnosis of mediastinal lesions.

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We would like to thank Dr. Marvin Daves for the use of Case v.

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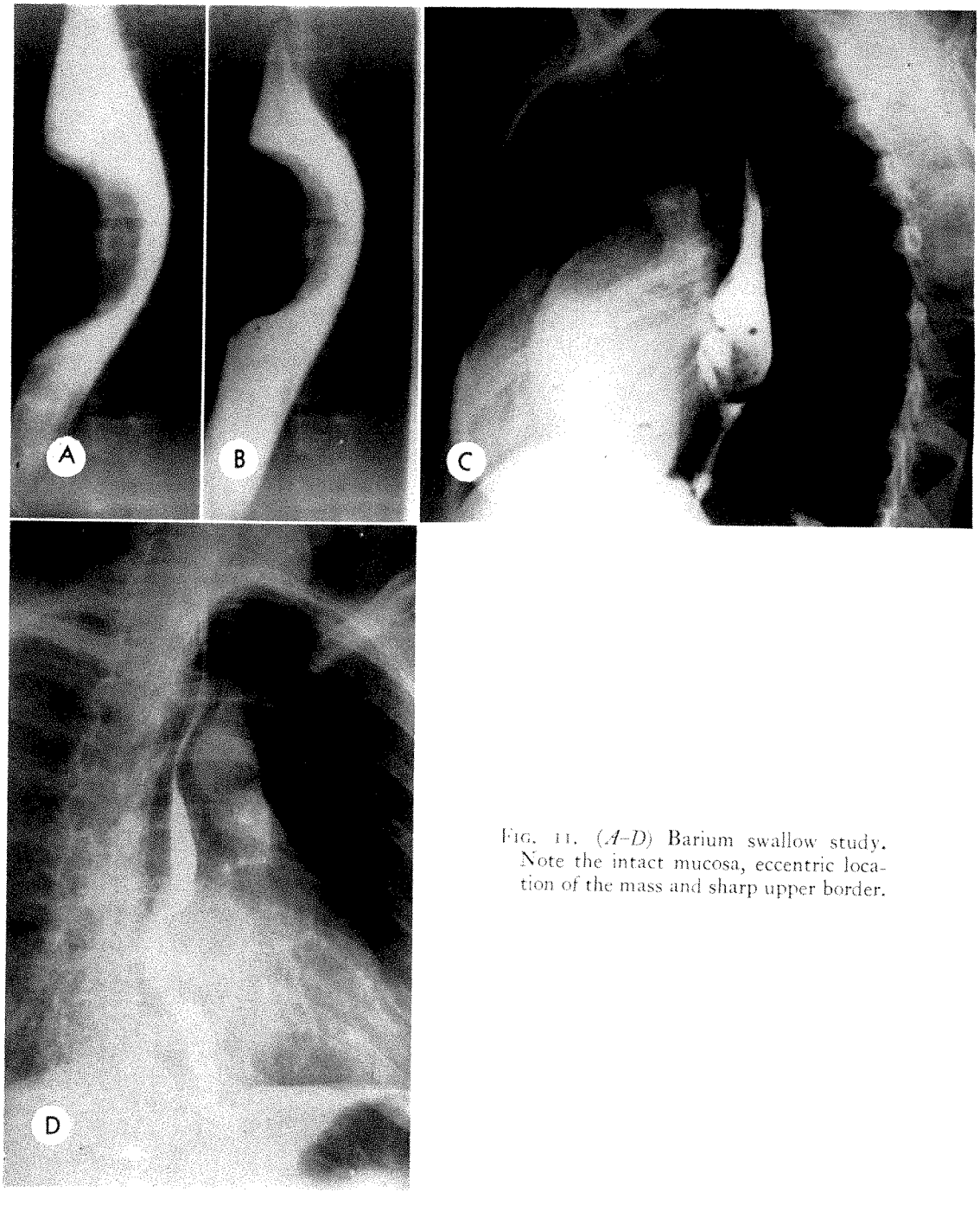



FIG. 11. (*A–D*) Barium swallow study. Note the intact mucosa, eccentric location of the mass and sharp upper border.

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# CARCINOSARCOMA OF THE ESOPHAGUS*

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CARCINOSARCOMA, a rare cancer composed of both mesodermal and epithelial elements, has been reported in the uterus, pharynx, lung and esophagus. Talbert *et al.*[7] found only 23 acceptable documented cases in the esophagus and added 4 of their own. Moore *et al.*[3] have since added 1 case. The last mention of esophageal carcinosarcoma in the radiologic literature available to us was that of Ende *et al.*[2] in 1951. Because of its rather typical roentgenographic appearance and some clinically important aspects of behavior, the radiologist should be familiar with it.

## REPORT OF A CASE

This 84 year old white man presented with a 6 week history of progressive dysphagia, especially with solid foods, associated with some subxyphoid discomfort. He had lost an undetermined amount of weight. The physical examination disclosed a somewhat weak elderly man but was otherwise unremarkable.

Barium swallow study showed a 3×5 cm. lobulated, polypoid mass in the distal third of the esophagus, with a rather narrow attachment to the postero-medial wall. The esophagus was neither fixed, rigid, nor thickened. No ulcerations were seen (Fig. 1 and 2). These findings suggested the diagnosis of carcinosarcoma.

At esophagoscopy the polypoid mass had a smooth surface, a pedicle about 1 cm. in width, and could be moved up and down the lumen.

Because of the patient's age and clinical condition, it was elected to remove the mass endoscopically. Symptomatic relief was complete. Because of the limited resection, postoperative radiation therapy was given.

Microscopic examination disclosed several areas with a fibrocollagenous stroma, which in one place was accompanied by bone formation. Most of the stroma, however, was composed of elongated and intertwining sarcomatous cells harboring nests of anaplastic squamous cell carcinoma. In the more differentiated areas, squamous cell pearls were present (Fig. 3).

## DISCUSSION

This patient illustrates many of the important features of carcinosarcoma. Although its rarity precludes valid statistical analysis, it seems to have a predilection for the lower half of the esophagus, especially in elderly males over 50 years of age. Of the previous 28 reported cases, 22 have been in males over 50 years of age. The symptoms are those of any obstructing esophageal lesion and include epigastric distress, retrosternal pain, dysphagia, and weight loss. Melena has been reported.[3]

As opposed to the usual esophageal carcinoma, carcinosarcoma characteristically presents on barium swallow study as a

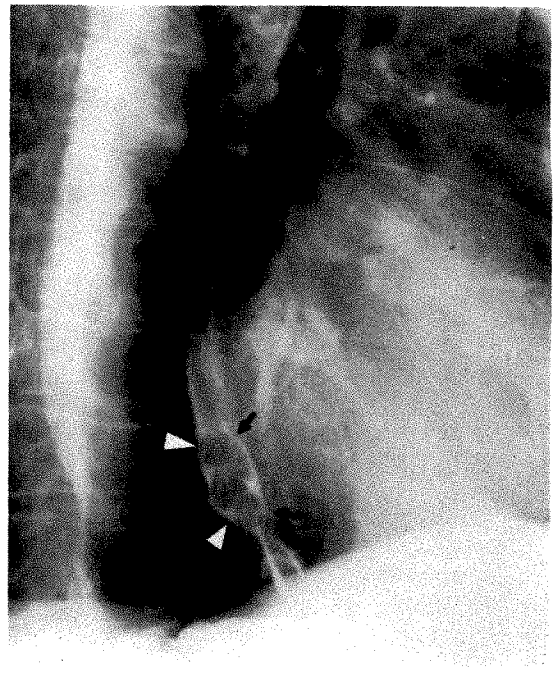


FIG. 1. Barium swallow study shows the lobulated mass in the distal third of the esophagus (arrows).

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polypoid, frequently lobulated filling defect in the lumen, of varying size, occasionally ulcerating with or without thickening and rigidity of the esophageal wall. Occasionally the narrow stalk may be identified. Other polypoid lesions of the esophagus are fibroma, lipoma, myoma, papilloma, leiomyoma, leiomyosarcoma and even melanosarcoma.[1]

Although the polypoid nature of carcinosarcoma is not diagnostic, its characteristic combination of roentgenographic findings and clinical picture permitted us

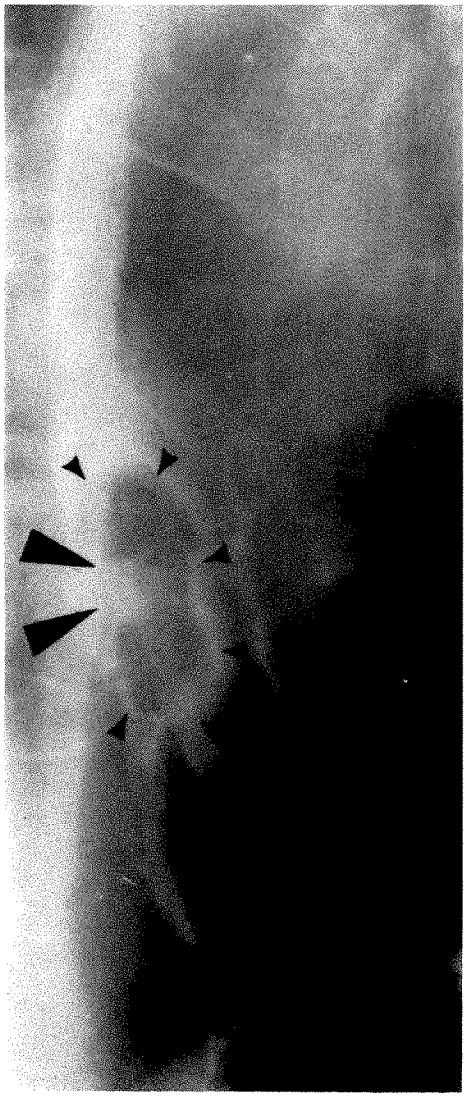


Fig. 2. Note the polypoid tumor (small arrows) with a narrow pedicle (large arrows) attached to the postero-medial wall.

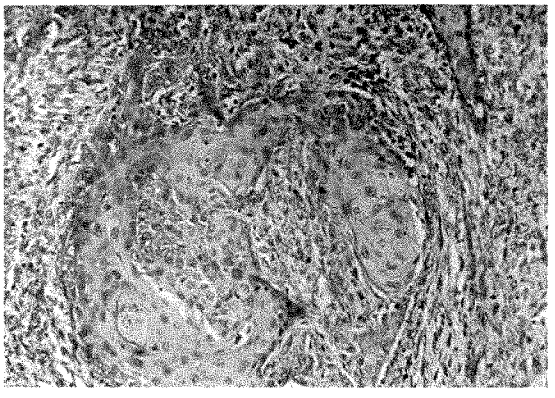


Fig. 3. Much of the stroma is composed of intertwining sarcomatous cells. There are nests of anaplastic squamous cell carcinoma and in the more differentiated areas, squamous cell pearls.

to suggest the diagnosis in our case. Carcinosarcoma is especially suggested when the biopsy is reported as carcinoma since pure polypoid squamous cell carcinoma apparently has not been described.[6] In previously reported cases, endoscopic biopsy never gave a diagnosis of carcinosarcoma but rather of carcinoma, sarcoma or undetermined lesion,[3] the diagnosis of carcinosarcoma being made only after examination of the surgical specimen.

It is clinically important to differentiate carcinosarcoma from the usual esophageal carcinoma. Several authors[3,5,7] have noted the relatively benign biologic behavior of these lesions. Although lack of a significant series prevents valid conclusions regarding the results of treatment, carcinosarcoma tends to remain superficial, to invade the muscularis propria slowly, and to metastasize late. The patient of Moore *et al.*[3] was well $5\frac{1}{2}$ years after only local resection of the tumor with a wide mucosal cuff at the base of the pedicle.

The exact histogenesis of carcinosarcoma is controversial. Indeed, Pearlman[4] concluded that its existence was questionable. A complete discussion of the pathologic features has been presented by Talbert *et al.*[7] and need not be repeated here. The polypoid nature, the slowly invasive character, and the microscopic pathology are quite different from the usual esophageal cancer. The sarcomatous element fre-

quently is an undifferentiated "spindle cell" sarcoma, but rhabdomyosarcoma,[2] leiomyosarcoma, and fibrosarcoma have all been reported. Although the epithelial component is most frequently epidermoid carcinoma in varying degrees of differentiation, adenocarcinoma is also seen.

## SUMMARY AND CONCLUSIONS

1. The radiologist should consider carcinosarcoma in the differential diagnosis of lower esophageal polypoid tumors, especially in elderly males and when the biopsy is reported as carcinoma.

2. Carcinosarcoma tends to remain superficial, to invade the esophageal wall slowly, and to metastasize late; thus, its biologic behavior is quite different from the usual esophageal carcinoma.

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# THE RADIOPAQUE ESOPHAGEAL MARSHMALLOW BOLUS*

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BARIUM sulfate suspended in water has been used for many years as the standard contrast medium for the roentgenographic study of esophageal function and disease. However, the fluid nature of this mixture limits its value in the assessment of the caliber of the esophageal lumen. As a result, attempts have been made over the years to make the vehicle in which the barium is suspended more viscous. Cylinders of gelatin[8] impregnated with barium, cotton pledgets, and pieces of meat dipped in barium have been used. Tablets of barium sulfate[6] and gelatin capsules filled with barium sulfate[5] have also been suggested for evaluating the caliber of the lower esophageal ring. In this report a new bolus is described in which barium sulfate is suspended in a marshmallow matrix. This bolus will be shown to be a useful technique in the evaluation of dysphagia as well as in the study of esophageal neuromuscular disorders such as diffuse esophageal spasm.

## EVALUATION OF THE SYMPTOM OF DYSPHAGIA

Dysphagia signifies the failure of an orderly passage of a swallowed bolus through the esophagus and into the stomach. The cause of dysphagia will usually be revealed by fluoroscopic study of the esophagus during the swallowing of a barium suspension. However, because of the liquid nature of this material the fluoroscopist may fail to detect an abnormality despite the presence of symptoms. Thus, when dysphagia occurs, it is advisable that a bolus of sufficient size and consistency to elicit the symptoms of dysphagia be introduced into the esophagus, and that the bolus be given along with barium and its progression down the esophagus observed.

TABLE I

PREPARATION OF THE BARIUM MARSHMALLOW

*Ingredients*

1. Gelatin —14 grams (2 packages of Knox Gelatin)
2. Water —½ cup
3. Cornstarch—2 tablespoons
4. Corn syrup—¾ cup (use clear colored syrup)
5. $BaSO_4$ —1 cup
6. Sugar —½ cup

*Method*

Dissolve gelatin in water for 5 minutes. Place on double boiler and liquefy. Add sugar to double boiler and dissolve.

Add this mixture to rest of dry ingredients and mix for 10 to 15 minutes.

Place in greased pan or beaker and allow to gel at room temperature overnight. Cut with mold and roll in cornstarch or confectionary sugar. Store in plastic bag free of air.

This procedure will usually demonstrate that the dysphagia is caused either by a defect of esophageal peristaltic activity or by an organic constriction of the esophageal lumen.

Ideally, a bolus should possess the following characteristics: (1) it should be nonfluid, plastic, elastic and of such size that it may be easily swallowed whole by the patient; (2) it should distend the esophagus maximally; and (3) it should possess the radiopacity of barium sulfate. Elasticity is a very desirable feature, since it will permit greater ease of swallowing. Plasticity permits it to be molded by surface irregularities and constrictions of the esophagus. These will alter the contour of the bolus and, as in a metal casting, the bolus will complement the mold.

The composition of the bolus should be

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such that should it become lodged at a point of obstruction, it will dissolve spontaneously and not require surgical or endoscopic removal. Once beyond a narrowed segment, the elasticity allows the bolus to re-expand and fill the esophageal lumen.

When a thin barium sulfate and water suspension is swallowed, the head of the barium column reaches the distal esophagus in 1 to 2 seconds. In the distal esophagus there is a momentary holdup of the passage of barium into the stomach, resulting in variable degrees of distention of the upper esophagus. The retained barium is then swept out of the esophagus by a peristaltic wave which begins within less than 1 second of the initiation of the swallow. This wave sweeps down the esophagus at a rate of 2 to 4 cm. per second,[1] requiring 4 to 9 seconds to traverse the entire esophagus,[2] the speed of the peristaltic wave varying with the consistency of the barium mixture. Thus, the degree to which the esophagus is distended by a barium and water mixture depends upon 3 factors, namely: (1) the consistency of the barium mixture; (2) the speed of the peristaltic complex; and (3) the duration of the holdup of swallowed material in the distal esophagus.

To slow down esophageal transit time, Kelly[4] introduced the commercial marshmallow as an esophageal bolus. It is pliable, elastic and distends the esophageal lumen well but is not radiopaque. Barium is swallowed along with the marshmallow, the bolus being visible as a radiolucent filling defect in a long column of barium. If the

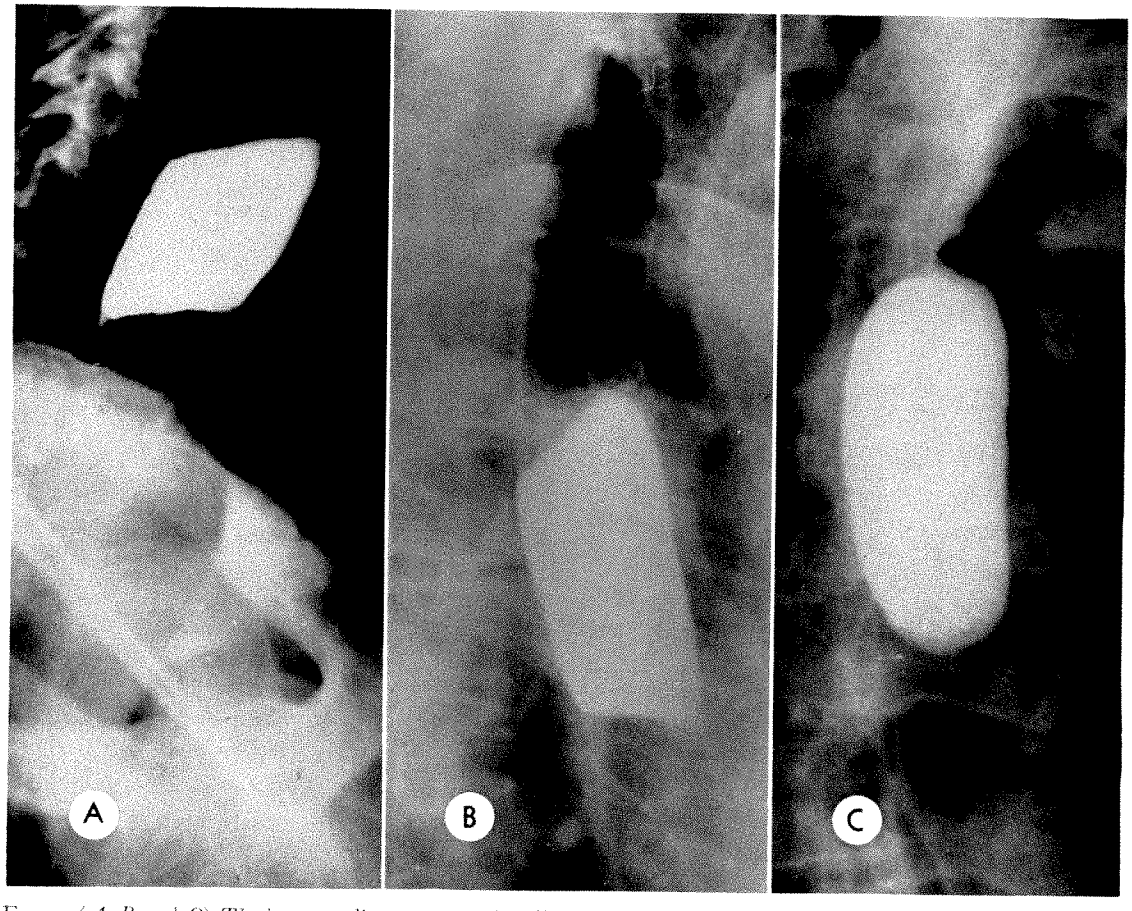



FIG. 1. (*A*, *B* and *C*) *The intact radiopaque marshmallow at various levels in the esophagus.* In the hypopharynx the radiopaque bolus is cuboidal in shape but as it progresses through the esophagus it is molded by the contours of the esophageal lumen.

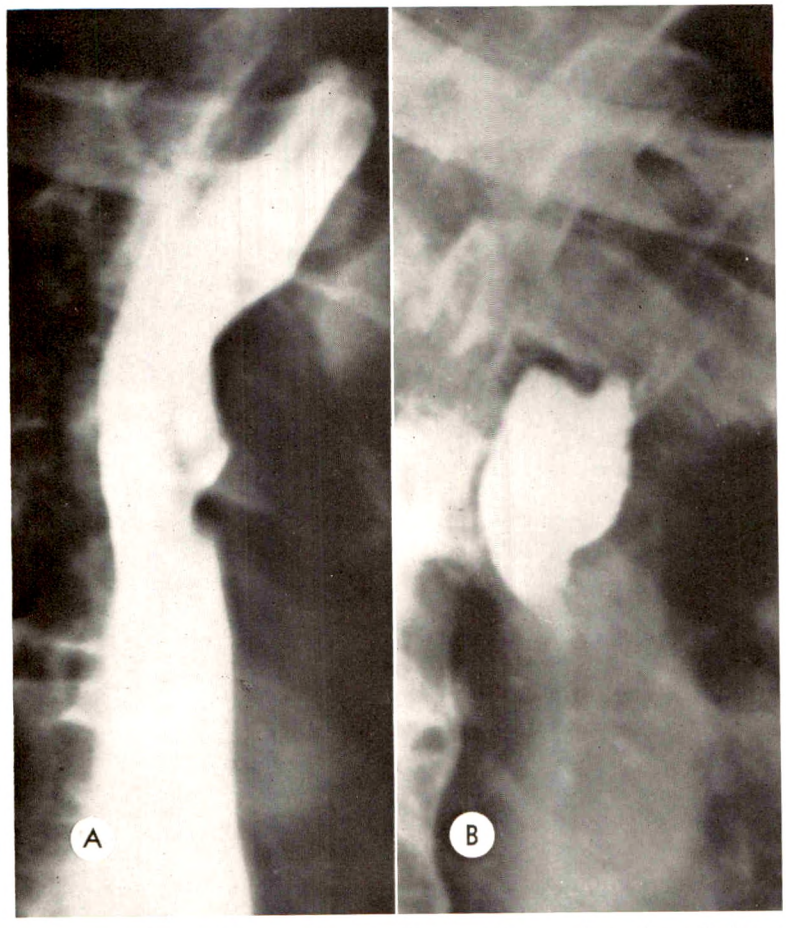



Fig. 2. *Carcinoma of the esophagus.* (*A*) A barium swallow study reveals an irregularity of the wall of the esophagus just below the clavicle. (*B*) The radiopaque marshmallow is held up and deformed by the tumor mass.

marshmallow should become arrested in an area of narrowing, it is dissolved in approximately 10 minutes by salivary ptyalin. By adding barium to the basic ingredients of the marshmallow, a radiopaque bolus has been produced which possesses all of the desirable features outlined above.

The radiopaque marshmallow, a mixture of water, gelatin, cornstarch, corn syrup, cane sugar and barium sulfate, can be prepared in a loaf of variable size (Table 1). Cubed segments of 8 to 10 cc. volume are cut as needed. The patient is instructed to place the bolus in his mouth and to swallow it whole without chewing. Once past the cricopharyngeus, the marshmallow distends the esophageal lumen and is propelled slowly down the esophagus (Fig. 1, *A–C*), reaching the esophagogastric junction in 10 to 20 seconds. If an obstruction is encountered (Fig. 2, *A* and *B;* and 3, *A* and *B*), symptoms of dysphagia occur and the marshmallow is deformed and forced through the area by the peristaltic wave. Once past the area of constriction, the bolus resumes its cuboidal shape with a prompt abatement of the dysphagia symptoms.

### Use in Motor Abnormalities of the Esophagus

The radiopaque marshmallow bolus can also be used for evaluation of peristaltic activity. In diffuse esophageal spasm, the bolus does not progress down the esophagus in an orderly fashion but instead is moved

up and down by the segmental, spontaneous contractions of the esophageal wall which replace primary peristalsis.

In acute esophagitis, the progression of the bolus may be halted and distorted by a spasmodic contraction of the lower esophagus. When this occurs the patient may experience severe substernal pain. However, the spasm is only transient and with relaxation of this area of the esophagus, the marshmallow will resume its normal shape and the patient will be relieved of his substernal discomfort.

The radiopaque marshmallow is also of value in the detection of sliding hiatal hernias. Figure 4, *A* and *B* shows the marshmallow lodged in the diaphragmatic hiatus. This failure of progression of the bolus is not associated with dysphagia and results from the absence of peristalsis in the herniated cardia of the stomach.

SUMMARY

A bolus made of marshmallow impregnated with barium sulfate is described and suggested as the "ideal" bolus for fluoroscopic evaluation of esophageal disease. Its elasticity, plasticity, shape, size, ease of swallowing and radiopacity contribute to the information which may be derived from fluoroscopic study of the esophagus.

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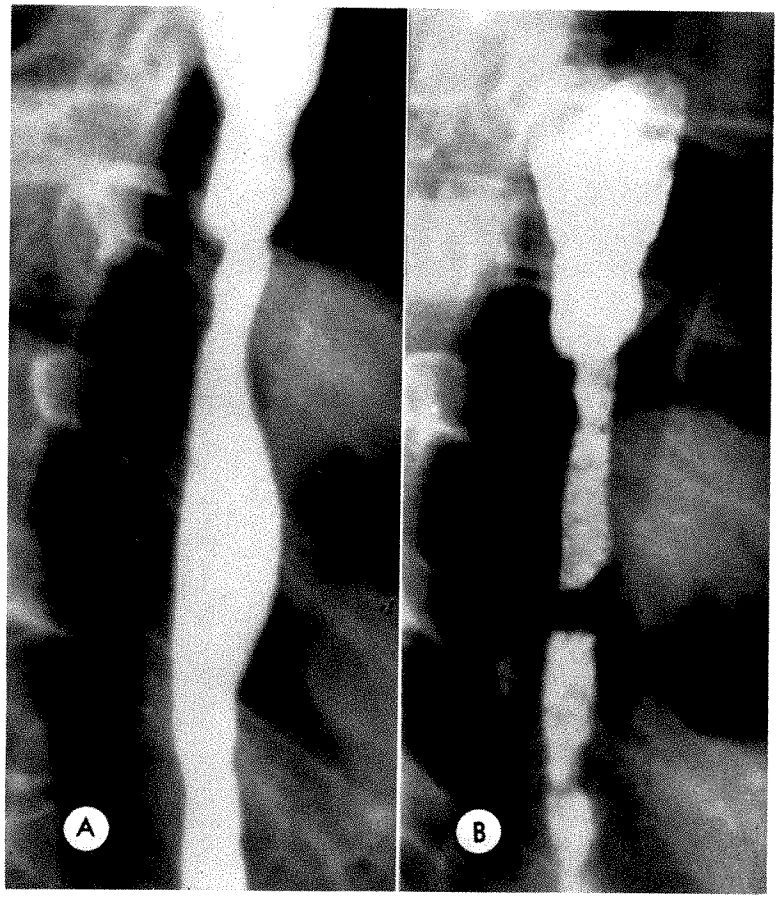



FIG. 3. *Lye stricture of the esophagus.* (*A*) A barium swallow study reveals a stricture of the midesophagus. (*B*) The radiopaque marshmallow is held up and deformed by the stricture. The radiopaque material distal to the stricture represents disintegrating marshmallow.

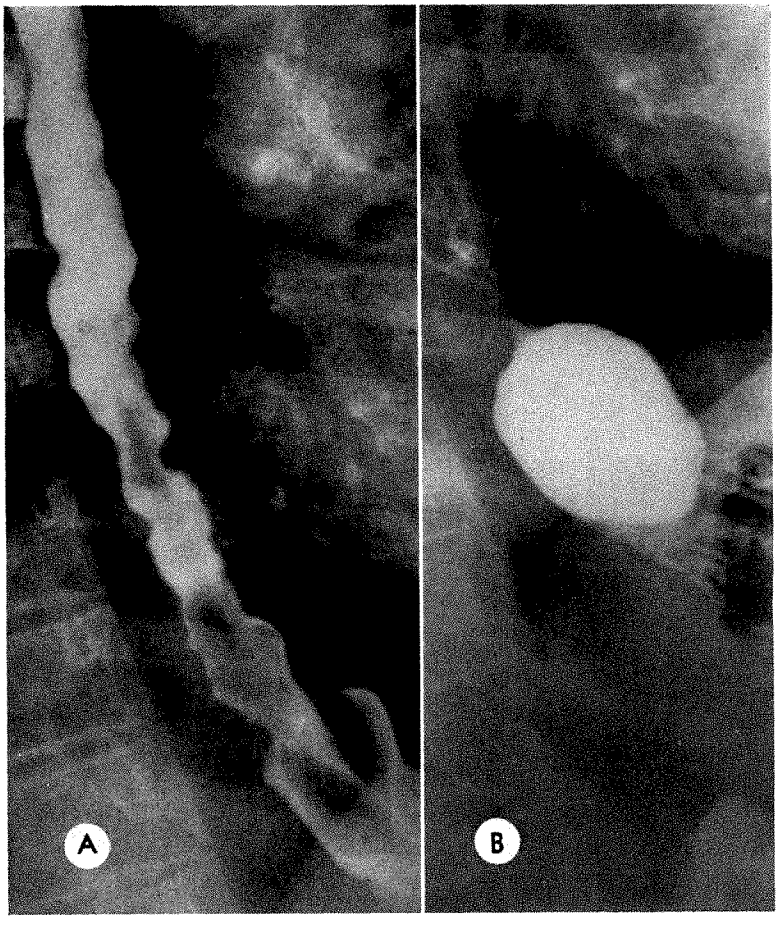



FIG. 4. *Sliding hiatal hernia.* (*A*) A barium swallow study reveals a sliding hiatal hernia with tertiary contractions of the esophagus. (*B*) The radiopaque marshmallow is seen within the hernial sac. The holdup of the bolus is due to the normal absence of peristalsis within the herniated cardia of the stomach. A holdup of the radiopaque marshmallow within the diaphragmatic hiatus without symptoms of dysphagia is diagnostic of a sliding hiatal hernia.

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# COTTON WRAPPING TECHNIQUE FOR REMOVING POINTED FOREIGN OBJECTS FROM THE GASTROINTESTINAL TRACT

## REPORT OF A CASE

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APPARENTLY few physicians are aware of the cotton wrapping or envelopment method for removing potentially dangerous pointed objects from the gastrointestinal tract. A review of the literature reveals a large number of papers on foreign objects in the gastrointestinal tract, but very few describe methods of assisting the passage of these objects. A majority of the authors advise waiting, either with or without the addition of bulky foods to the diet. A method of expediting the safe passage of the objects while reducing the risk of perforation should appeal to both patients and their physician.

### COTTON WRAPPING TECHNIQUE

The method or technique was outlined recently by Frohman.[1] Small pledgets of cotton are mixed thoroughly with banana or with moistened bread and are fed to the patient in small amounts at frequent intervals. The banana or bread simply facilitates the swallowing of the cotton. The food is digested, leaving the cotton fibers to move along with the intestinal contents.

The cotton fibers will adhere to the surfaces and catch on projections of the foreign object and enclose it completely as the fibers accumulate. The object is quickly wrapped or enclosed in a smooth, rounded mass of cotton which disengages the point or points from the mucosa and forms a smooth bolus which gastrointestinal peristalsis can propel easily.

The addition of very small amounts of barium sulfate suspension to the patient's intake will opacify the cotton as it accumulates, permitting roentgenographic observation of the progress.

Some authors have warned against the use of cotton for this purpose, citing possible intestinal obstruction as a theoretic reason against its use. Obviously, some degree of common sense should be exercised in determining a reasonable quantity of cotton to be fed the patient each day.

### REPORT OF CASE

An adult male, 68 years of age, unknowingly swallowed a dental bridge while at dinner. We suspect that the bridge was swallowed with a piece of meat which prevented the metal points from traumatizing the esophagus. The patient did not feel the object pass through the esophagus, and realized he had swallowed the bridge only when he discovered it was missing.

Twelve hours after ingestion, the bridge was in the stomach. After 36 hours, it was in the ileum, and at 60 hours was located in the ascending colon. After five days, the bridge had lodged in the sigmoid colon (Fig. 1). Mild distress and local tenderness developed in the left

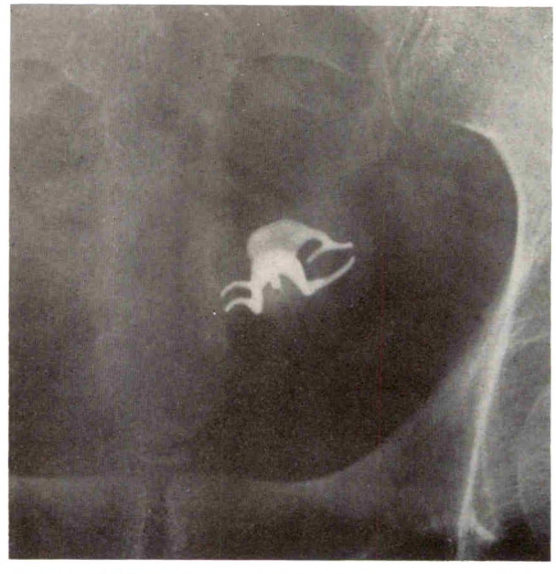


FIG. 1. Object before cotton wrapping is shown lodged in the sigmoid colon.

lower quadrant of the abdomen and the patient was admitted to the hospital for possible surgical removal. It was at this time that the cotton wrapping technique was initiated.

The object was beyond the reach of the sigmoidoscope. Seven days after ingestion, an attempt was made to disengage it from the sigmoid by means of an extremely viscous barium enema prepared by the Dietary Department. The enema was so thick it had to be injected with a hand syringe, rather than by gravity. We felt that if spasm could be overcome, and the colon distended, the bridge might be expelled along with the thick enema material. The colon spasm could not be overcome with reasonable pressure. A small amount of barium did pass the foreign object and proved helpful by visualizing the development of the cotton enclosure.

The day after attempted removal by the barium enema, a roentgenogram showed the object enclosed in cotton, faintly opacified by the barium (Fig. 2). At this time, the surgeon in charge was sufficiently concerned over the possibility of perforation of the colon by the long pointed metal prongs that the patient was scheduled for surgery the next day.

Early in the morning of the day of scheduled surgery, the foreign object was passed easily with no discomfort. Unfortunately, we did not anticipate the prompt disengagement and passage of the object and had not requested the nursing personnel to leave the cotton coating in place. Therefore, we did not obtain a photograph of the enclosed object as passed.

Apparently the colon suffered some injury, since the patient complained of left lower quadrant discomfort during the next 2 weeks. A barium enema colon study at that time demonstrated localized spasm of the sigmoid colon where the object had been lodged. Conservative management was followed and a repeat study 3 weeks later showed no spasm and a normal appearance of the sigmoid colon.

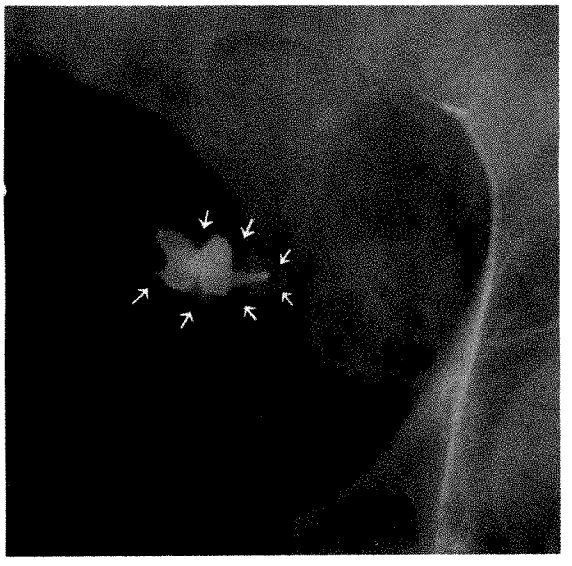


Fig. 2. Object with cotton wrapping. The cotton (arrows) is faintly opacified by the barium.

## CONCLUSION

Apprehension on the part of the patient, his relatives, and the surgeon or pediatrician is an important factor in the management of patients with foreign objects in the gastrointestinal tract. This is particularly true in the case of open safety pins, needles, straight pins, and multipointed objects, such as dental bridges. We recognize some risk of perforation of the intestinal tract, but there is also a risk inherent in surgical removal.

We believe that prompt initiation of the cotton wrapping technique will facilitate the quick and safe passage of the foreign objects to the satisfaction of all concerned.

This case is reported with the thought that all physicians should know of this technique. Radiologists, particularly, are in a position to suggest the method to physicians whose patients have ingested pointed foreign objects.

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*Liaison Representatives on the National Council on Radiation Protection and Measurements:* Herbert M. Parker, Richland, Wash.; Marvin M. D. Williams, Rochester, Minn.

*Representative on the Board of Chancellors of the American College of Radiology:* George G. Stetson.

The Fiftieth Annual Meeting of the Society will be held at the Hotel Fontainebleau in Miami Beach, Fla., April 7–11, 1968.

# EDITORIALS

## PROGRESS IN GASTROINTESTINAL ROENTGENOLOGY

BY CAREFUL selection of woods, with skill born from experience and with careful finishing and final polishing, Victorian and early American Cabinet makers hand-produced beautiful furniture, which today remains as a tribute to their craftsmanship. In a similar manner, our Editor, with the aid of the Editorial Policy Committee, "hand-produces" each issue of the AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE by careful selection from a large number of papers offered for publication. Scientific worth, grammar, composition, style, correct use of statistics and high-quality photographs for reproduction are some of the criteria for publication. Final polish is the assembly and publication of subject-related papers into a volume for continuity of thought and convenience of the reader.

This editorial "craftsmanship" is characteristic of each issue of the JOURNAL and the current "Hickey Volume" is an annual highlight. Thirty-three papers headed by the Hickey Lecture of Dr. Alexander Margulis present examples of the progress being made in diagnostic roentgenology of the gastrointestinal tract. All of the articles are worth careful study but the Hickey Lecture, in Margulis' fine lucid style, deserves special attention. Not only does he tell of some new very practical ways to improve our diagnostic abilities but he seems to open new horizons with his discussion of drug induced alterations of gastrointestinal physiology. Especially stimulating is the portion dealing with small bowel change in certain enzyme deficiencies and one does not have to be overly imaginative to visualize many new avenues of investigation as Margulis discusses challenging the intestinal tract with suspected allergens.

These suggestions, as well as the small bowel alterations in peripheral edema, as reported by Marshak and his co-workers, must be accepted with caution. For detection of gross neoplastic or inflammatory disease our examination of the esophagus, stomach and colon is more accurate than that of the small bowel. When we deal with the problems of alteration in *motility*, *mucosal pattern* and *absorptive function* of the small bowel we encounter difficulties. While it is relatively easy to recognize severe alteration of these 3 features, changes less marked must bring up the question, "Is this the normal roentgenologic appearance of the small bowel of this patient on this day?"

In the average healthy person the esophagus, stomach and colon will show very little, if any, change when examined at various intervals but small bowel motility, mucosal pattern and fluid content may vary from time to time and from person to person. Human emotions, dietary intake, state of hydration, previous disease, etc., make it necessary that we take full advantage of clinical and laboratory correlation when attempting to evaluate what we think may be minimal or moderate change in the small bowel.

Advances in gastrointestinal roentgenology have been very real and of benefit to the patient. Certainly, image intensification fluoroscopy and cine studies have increased our ability in diagnosis of disease of the esophagus. Scanning of radioactive isotope concentrations in the liver has proved to be a practical test of great value

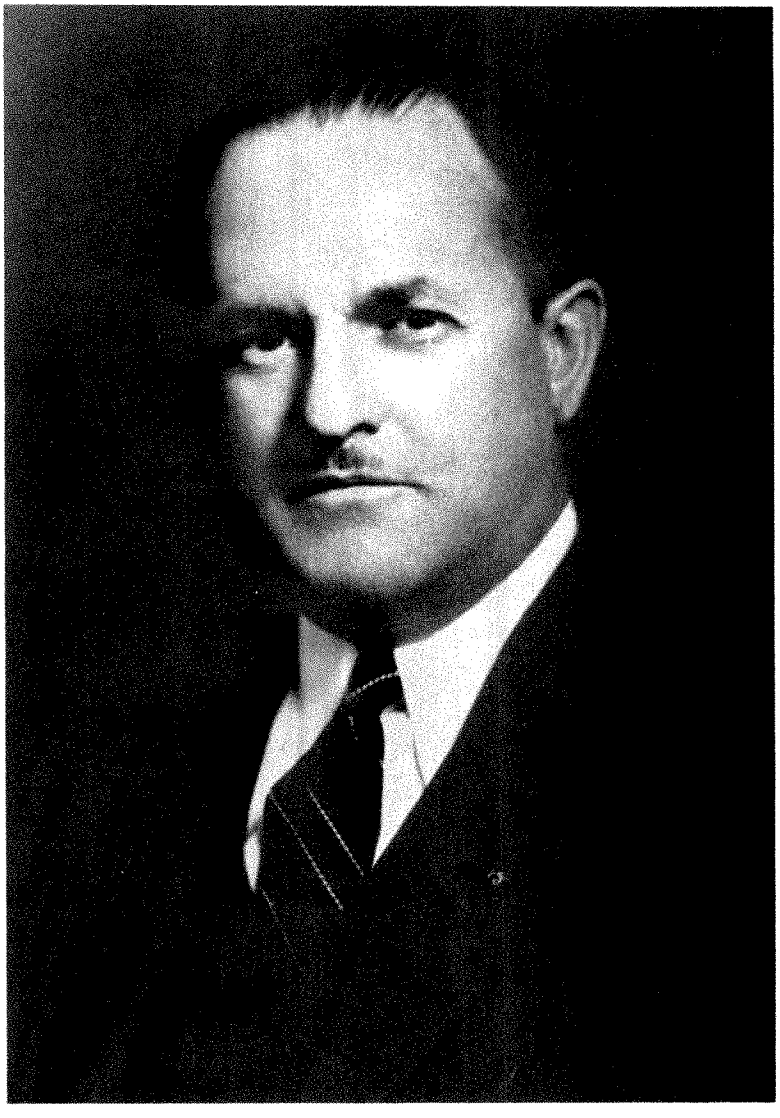
and, in cases of obscure bleeding from the gastrointestinal tract, isotopes can be useful in locating the point of origin. In selected cases, vascular opacification following selective catheterization can yield additional information but these studies are of more value in other body systems.

Image intensification, cine recording, general improvement in capacity and function of x-ray machines plus rapid film processing have produced benefits of convenience, reduced radiation exposure, increased speed of examination, provided better fluoroscopic and roentgenographic detail and enhanced teaching abilities. However, if some of our eminent predecessors such as Hickey, Cole, Carman, Sosman or Kirklin were with us today, I am not certain that we could convince them that we are making a better diagnosis of gastric and duodenal lesions than they did and, while knowledge of colon disease has increased, we have made no great advance in our detection ability since Weber's untimely death in 1958.

Gastrointestinal roentgenology presents a rich field for investigative work; many centers are doing excellent research and clinical studies and the usual closed-staff group has a well organized gastrointestinal service. However, over 50 per cent of the people in our country live in towns of less than 50,000 population and may not have the benefit of all of our current sophisticated diagnostic procedures.

In our enthusiasm for the new, we must not forget that for a large segment of our population real progress in gastrointestinal as well as in all diagnostic roentgenology can be made only if all physicians consider our work to be true *medical consultation.* The American Medical Association now officially supports this concept and we must make a serious effort to show that our consultation, like any other, would be of limited value without significant medical history and laboratory data. We must continue to emphasize that the patient may benefit if we have related previous roentgenograms for study before our examination, and, finally, report of our consultation can be of more value to a referring physician if we make an estimate of the accuracy of our findings.

Clyde A. Stevenson, M.D.
Sacred Heart Hospital
Spokane, Washington 99204



# CHRISTOPHER C. McCLURE, Sr., M.D.
## 1892–1967

DR. CHRISTOPHER C. McCLURE, Sr., pioneer radiologist of the South and first Chairman of the Radiology Department of Vanderbilt University, died April 19, 1967 following a cerebral thrombosis.

Dr. McClure was a native of Pennsylvania but moved to Mobile, Alabama in early childhood. He received the M.D. degree from Vanderbilt University in 1918 and obtained his radiology training at Post Graduate Hospital, New York City and Massachusetts General Hospital in Boston, Massachusetts in 1919 and 1920.

Dr. McClure was one of the original members of the Atomic Energy Commission and served on the Teletherapy Evaluation Board of the Oak Ridge Institute of

Nuclear Studies. He was also former Chief of the Radiological Section of the Veterans Administration for the eastern United States.

Dr. McClure was an outstanding lecturer from his carefully selected and meticulously documented museum of radiographs and a favorite of medical students for nearly four decades. He also promoted interest in medicine among undergraduate students by his lectures and demonstrations using a portable fluoroscope and was instrumental in organizing Skull and Bones, the Undergraduate Premedical Society. He retired from Vanderbilt University as Emeritus Professor in 1958.

He was a member of the American College of Radiology, American Roentgen Ray Society, Radiological Society of North America, Alpha Omega Alpha, and American Medical Association. He also was a Thirty-second Degree Mason, Shriner, and Exchange Club member. He served as an elder of the First Presbyterian Church.

He is survived by his wife, the former Miss Nelle Dunn, whom he married in 1917; a daughter, Miss Mary Eleanor McClure; a son, Dr. C. C. McClure, Jr., and four grandchildren.

Thomas R. Duncan, M.D.
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# NEWS ITEMS

## NINTH INTER-AMERICAN CONGRESS OF RADIOLOGY

Supplementing the announcements made in the November 1966, and March and June 1967, issues of this JOURNAL, the following additional information was obtained regarding the activities of the Ninth Inter-American Congress of Radiology.

The Preliminary Program lists some of the most outstanding radiologists as the Main Speakers in the 3 respective Sections of the Congress. They, and their topics, are: *Radiodiagnosis*: Dr. E. Cherigie (France), Indications and advantages of the different methods used to register the radiological image; Dr. H. Abrams (USA), The radiology of acquired heart diseases; Dr. J. Caffey (USA), The radiology of congenital bone dystrophies; Dr. M. Juster (France), Microradiography of bone; Dr. R. Leborgne (Uruguay), Mammography; and Dr. Torgny Greitz (Sweden), The radiology of insufficient brain circulation. *Radiotherapy*: Dr. V. Marcial (Puerto Rico-USA), Fractionation in radiotherapy; Dr. G. Fletcher (USA), The value of oxygenation in radiotherapy; and Dr. M. Roxo-Nobre (Brazil), The radiotherapy of tumors of the hypopharynx. *Nuclear Medicine*: Dr. A. Rodriguez Antunez (USA), The diagnosis by x-rays and radioisotopes of anatomic and functional alterations of the kidney, liver and brain.

The Congress will be held in the Hotel San Rafael, Punta del Este, Uruguay, December 6–12, 1967. The officers are: *President*, Dr. Leandro Zubiaurre; *Vice-Presidents*, Dr. Hector Bazzano and Dr. Agustin Gorlero Armas; *Secretaries*, Dr. Helmut Kasdorf and Dr. Roberto François; and *Treasurer*, Dr. Jose A. Soto.

The Official Delegation of the USA to this Congress is: Juan A. del Regato, M.D., *Chairman*, representing the Radiological Society of North America, The American Radium Society and the National Cancer Institute; F. J. Bonte, M.D., representing the American Roentgen Ray Society; William S. Maxfield, M.D., representing the Society of Nuclear Medicine; J. Paul Medelman, M.D., representing the Section on Radiology of the American Medical Association; J. E. Miller, M.D., representing the American College of Radiology; James J. Nickson, M.D., representing the American Society of Therapeutic Radiclogists; and Manuel Viamonte, Jr., M.D., representing the Association of University Radiologists.

The Alternate Delegates are: Herbert L. Abrams, M.D., Association of University Radiologists; George D. Davis, M.D., American Roentgen Ray Society; Philip J. Hodes, M.D., Radiological Society of North America; Juan A. del Regato, M.D., American Society of Therapeutic Radiologists; and W. T. Snow, M.D., American College of Radiology.

The address of the Secretariat of the Congress is Casilla de Correo No. 930, Montevideo, Uruguay.

## THIRD ANNUAL SAN FRANCISCO CANCER SYMPOSIUM

Claire Zellerbach Saroni Tumor Institute of San Francisco

The Third Annual San Francisco Cancer Symposium will be held Friday and Saturday, October 20, and 21, 1967, in the Mount Zion Sutter Building Auditorium.

The subject chosen for this year's symposium is "The Relationship of Time and Dose in the Radiation Therapy of Cancer."

The participants include: Dr. Charles Botstein, New York, New York; Miss Lucille DuSault, Detroit, Michigan; Dr. Mortimer Elkind, Bethesda, Maryland; Dr. Frank Ellis, Oxford, England; Dr. Edward Epp, New York, New York; Dr. Jack Fowler, London, England; Dr. Ralph Johnson, Bethesda, Maryland; Dr. Robert Kallman, Stanford, California; Dr. Seymour Levitt, Richmond, Virginia; Dr. Victor Marcial, San Juan, Puerto Rico; Dr.

Theodore Phillips, San Francisco, California; Dr. Juan A. del Regato, Colorado Springs, Colorado; Dr. Philip Rubin, Rochester, New York; Dr. D. K. Sambrook, Swansea, Wales, England; Dr. P. W. Scanlon, Rochester, Minnesota; Dr. Herman Suit, Houston, Texas; Dr. Jerome M. Vaeth, San Francisco, California; and Mr. Peter Wootton, Seattle, Washington.

For further information please write to: Jerome M. Vaeth, M.D., Saroni Tumor Institute, 1600 Divisadero Street, San Francisco, California 94115.

## RADIOISOTOPES AND THE KIDNEY

School of Medicine
State University of New York at Buffalo

A Symposium on Radioisotopes and the Kidney will be held as part of Continuing Medical Education Programs on Friday and Saturday, November 17 and 18, 1967.

The topics to be considered are the following: Principles of renal physiology; radio-pharmaceuticals and dosimetry; principles of the scintillation camera; principles of digital radio-autofluoroscopy; principles of multi-crystal scanners; principles of the Ter-Pogossian camera; rectilinear and non-rectilinear scanning; dynamic studies by external monitoring techniques; the mechanisms of renal excretion of isotopically-labelled compounds; renal hemodynamics; radio-renography and renal blood flow by $Xe^{133}$.

A distinguished Local and Guest Faculty will take part in the discussions.

For registration please contact: Associate Dean for Continuing Medical Education, Main Street Division, School of Medicine, State University of New York at Buffalo, 2211 Main Street, Buffalo, New York 14214.

## ELEVENTH ANNUAL POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY

University of California School of Medicine, San Francisco

The Eleventh Annual Postgraduate Course in Diagnostic Radiology will be presented by the Department of Radiology and Continuing Education in Health Sciences of the University of California San Francisco Medical Center, March 5–9, 1968.

This is an intensive 5 day course which includes lectures, panel discussions, symposia, exhibits, and a social hour. Registrants may enroll for the full 5 days or by the day.

The Program Chairman is Alexander R. Margulis, M.D., Professor and Chairman, Department of Radiology. The Program Director is Howard L. Steinbach, M.D., Professor of Radiology, and the Assistant Program Director is Charles D. Noonan, M.D., Assistant Professor of Radiology.

The course will be conducted by the Faculty of the Department of Radiology of the University of California School of Medicine, San Francisco, and the following Guest Speakers: Kurt Amplatz, M.D., Minneapolis, Minnesota; Arthur R. Clemett, M.D., New Haven, Connecticut; J. S. Dunbar, M.D., Montreal, Canada; Robert G. Fraser, M.D., Montreal, Canada; William Martel, M.D., Ann Arbor, Michigan; Professor P. E. S. Palmer, M.B., B.S., F.F.R., D.M.R., D.M.R.T., The Department of Radiology, Groote Schuur Hospital, University of Cape Town, Observatory Cape, Cape Town, South Africa; Gordon Potts, M.D., New York, New York; Charles E. Shopfner, M.D., Kansas City, Missouri; and Walter M. Whitehouse, M.D., Ann Arbor, Michigan.

For further information or application forms of enrollment please write to: Seymour M. Farber, M.D., Dean of Educational Services and Director of Continuing Education in Health Sciences, University of California Medical Center, San Francisco, California 94122.

## POSTGRADUATE COURSE IN PEDIATRIC RADIOLOGY-CONGENITAL HEART DISEASE

Cornell University Medical College-Memorial Hospital

A Postgraduate Course in Pediatric Radiology-Congenital Heart Disease will

be held at the Memorial Hospital Auditorium, 68th Street, New York, New York, April 25–27, 1968.

In addition to stressing diagnostic radiology, emphasis will be placed on developmental and pathologic anatomy, and on medical and surgical aspects of congenital heart disease.

Supplementing the Cornell Staff the following distinguished Guest Faculty will participate: Herbert L. Abrams, M.D., Boston, Massachusetts; Murray G. Baron, M.D., New York, New York; Sidney Blumenthal, M.D., New York, New York; John A. Campbell, M.D., Indianapolis, Indiana; Robert L. DeHaan, Ph.D., Carnegie Institution of Washington, Baltimore, Maryland; Jessee E. Edwards, M.D., Saint Paul, Minnesota; Larry P. Elliott, M.D., Gainesville, Florida; Kent Ellis, M.D., New York, New York; Owings W. Kincaid, M.D., Philadelphia, Pennsylvania; and Richard Van Praagh, M.D., Boston Massachusetts.

For further information please contact Herman Grossman, M.D., Department of Radiology, New York Hospital–Cornell Medical Center, 525 East 68th Street, New York, New York 10021.

## OAK RIDGE ASSOCIATED UNIVERSITIES

### Low Exposure Total Body Radiation Facility

Completion of a radiation therapy facility, embodying a new approach to the treatment of cancer and allied diseases, has been announced by Oak Ridge Associated Universities, Oak Ridge, Tennessee.

The new facility, called the *Low Exposure Total Body Radiation Facility* consists of a suite of rooms attached to the ORAU Medical Division Hospital on Vance Road in Oak Ridge. The suite contains a central living room-bedroom large enough for 2 patients to live in comfortably for several weeks, while they are treated with radiation at extremely low dosage rates. The radiation emanates from 10 sources, containing radioactive cobalt 60, arranged around the exterior of the room—2 sources at each of the 4 corners and 1 above and 1 below it.

The new low exposure facility is expected to demonstrate improvements over whole-body radiotherapy units which operate at higher radiation dosages.

While a patient is being treated in the new facility, his heart rate, pulse rate and other physiologic responses are carefully monitored by remote-controlled, closed-circuit television and electronic biologic monitoring equipment.

The other rooms of the facility house the physiologic monitoring equipment, the controls for the 10 cobalt 60 sources, and an on-line time-sharing computer that allows rapid data acquisition and processing so that the condition of the patients and a continuous record of their radiation exposure is known precisely at all times.

Since 1950, the ORAU Medical Division, under the Association's contract with the United States Atomic Energy Commission, has been carrying out a variety of research programs concerned with the use of radioactivity and radioactive materials in the diagnosis and treatment of cancer and other diseases. Oak Ridge Associated Universities is a nonprofit educational and research corporation of 41 Southern universities and colleges, operating under contract with the AEC and other agencies.

## SECOND MATTHEW J. WILSON PROFESSOR OF RESEARCH RADIOLOGY

A radiologist noted for his pioneer work in radiation hazard control has been named the second Matthew J. Wilson Professor of Research Radiology at the University of Pennsylvania School of Medicine, as announced by Dr. Richard H. Chamberlain, Chairman of the Department of Radiology.

Dr. James T. Brennan comes to Pennsylvania from the post of First Director of the Armed Forces Radiobiology Research Institute, Bethesda, Maryland and is a retired colonel from the U. S. Army Medical Corps after 22 years' service. He

began his career in radiation biology in 1947 when he studied radiation hazard control methods at the Hanford Plutonium Plant, Richland, Washington.

At the University of Pennsylvania, he ill explore the feasibility of another medical application of nuclear energy: the use of fast neutrons to treat cancer.

Working closely with Dr. Brennan will be Dr. Antolin I. Raventos, radiotherapist, and Dr. Mortimer L. Mendelsohn, radiobiologist, both of whom Dr. Brennan first knew at Walter Reed Army Hospital during the Korean War.

### VARIETY CHILDREN'S HOSPITAL ANNUAL PEDIATRIC POSTGRADUATE COURSE "INFECTIOUS DISEASE"

A Pediatric Postgraduate Course on "Infectious Disease" will be held January 28 to February 1, 1968, at Deauville Hotel, Miami Beach, Florida.

The factors influencing infectious disease as to epidemiology, diagnosis, immune response and treatment will be considered. Special attention will be given to urinary, respiratory and gastrointestinal system disorders. There will be two successive sessions each morning. Registrants and faculty will participate in question and answer periods following each session.

The Chairman is Donald H. Altman, M.D., Variety Children's Hospital, and the Guest Faculty, completing the Local Faculty, comprises: Heinz F. Eichenwald, M.D., Dallas, Texas; Robert A. Good, M.D., Ph.D., Minneapolis, Minnesota; Herman Grossman, M.D., New York, New York; Jorge E. Howard, M.D., Santiago, Chile; Saul Krugman, M.D., New York, New York; Mark M. Ravitch, M.D., Chicago, Illinois; Albert B. Sabin, M.D., Cincinnati, Ohio; John L. Sever, M.D., Washington, D.C.; Margaret H. D. Smith, M.D., New Orleans, Louisiana; and Warren E. Wheeler, M.D., Lexington, Kentucky.

For further information please contact Donald H. Altman, M.D., Chairman Postgraduate Course, Variety Children's Hospital, 6125 Southwest 31st Street, Miami, Florida 33155.

# BOOK REVIEWS

*Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.*

TRATTATO DI RADIODIAGNOSTICA. Edited by Luigi Turano, Direttore dell'Istituto di Radiologia dell'Università di Roma. Volume II. Cloth. Pp. 623, with 508 figures. Price, L. 15,000. Unione Tipografico, Editrice Torinese, 1967.

This is the second Volume (the first Volume appeared in 2 Parts) of the basic Italian Radiological Treatise edited and partially written by Professor Luigi Turano. As the previous two-part Volume, this Volume is quite impressive for its contents and for the beautiful illustrations.

The first chapter is written by A. Centi Colella, A. Crinelli, and E. Marzano. It is a review of the radiology of the liver and includes anatomic details, a discussion of congenital anomalies, abscesses, tumors, echinococcus cysts, displacements and calcifications.

The second chapter, regarding the radiology of the gallbladder and of the bile ducts, is written by Professor Turano and is excellent. In addition to the usual radiologic material, the author draws on his considerable experience in physioradiology to point out the conditions in which altered physiology may produce clinical symptoms which cannot be explained on a purely morphologic basis.

The chapter on the pancreas is written by L. Ciarpaglini and covers the anatomy, the anomalies of the pancreas, and the various pancreatic diseases. This chapter would have benefited if greater emphasis had been placed on the displacements caused by pancreatic enlargements; however, the section devoted to the pancreatic ducts and the discussion of laminagraphy with gas contrast is satisfactory.

The radiology of the kidneys and ureters, and of the bladder, urethra and prostate, is discussed by L. Turano in 2 chapters. These chapters are truly excellent and reflect the vast clinical experience of the senior author as well as his unique bent for applying physiologic methods to radiologic investigations.

The postoperative radiology of the urinary tract is discussed by E. Salomoni. This chapter is a welcome source of reference for those who have occasion to observe a variety of postoperative results.

The radiology of the female genital tract is thoroughly discussed by G. Bompiani, who has had a vast experience in this field. The section on obstetrics is especially good.

L. Dalla Palma describes the radiologic information obtainable through the use of pneumoperitoneum and of retroperitoneal insufflations. These examinations seem to be used more commonly in Italy than elsewhere and are expertly described.

The last section is written by I. Salomoni and is concerned with the radiology of the acute abdomen.

C. GIANTURCO, M.D.

CARDIOVASCULAR ROENTGENOLOGY: A VALIDATED PROGRAM. By Charles M. Nice, Jr., M.D., Ph.D., Professor and Chairman, Department of Radiology, Tulane University School of Medicine; Director of Department of Diagnostic Radiology, Charity Hospital of Louisiana at New Orleans; Consultant to U.S.P.H.S. and V.A. Hospitals, New Orleans, and to Mobile General Hospital, Mobile, Ala. Paper. Pp. 272, with 98 illustrations. Price, $9.35. Hoeber Medical Division, Harper & Row, Publishers, 49 East 33rd Street, New York, N. Y. 10017, 1967.

Intense interest in teaching programs, self learning devices and in "teaching machines" has gripped educational circles during the last several years. Only more recently, however, have these new teaching modalities been extended into medical education. Dr. Nice's book represents one of the pioneer efforts in this direction for medical students. As an experiment in medical education, as a text designed for learning in a systematic fashion and as an attempt toward self instruction for medical students, it is of real interest and value.

As with any pioneer effort, a number of criticisms of the program can be made. For this reviewer, and for two medical students at Duke University who took this program, there

appeared not to be enough information on basic physiologic processes as reflected in the roentgenograms. While some of the reproductions are excellent, others are not clearly illustrative of the processes described in the text. The blocks of information covered by individual segments of the program are occasionally too large to be absorbed or understood by the students.

In a way, then, the program text provides a better summary than a learning program. It well may be that this particular program will be found more satisfactory as a "quick review" for residents in Radiology than for medical students.

Perhaps the most important aspect of this book will be its use by teachers of Radiology as a model for programmed texts. There can be little doubt that the creation of programs of this sort will become more and more useful in the instruction of residents, medical students and as refresher programs for practicing radiologists.

Richard G. Lester, M.D.

The Physical Aspects of Diagnostic Radiology. By Michel M. Ter-Pogossian, Ph.D., Professor of Radiation Physics, The Edward Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Mo. Cloth. Pp. 426. Price, $13.50. Hoeber Medical Division, Harper & Row, Publishers, 49 East 33rd Street, New York, N. Y. 10017, 1967.

Many radiological physicists will agree with Dr. Ter-Pogossian's "conviction that the study of the physical aspects of diagnostic radiology is insufficiently represented in the teaching curriculum of radiologists. . . . " Certain parts of Dr. Ter-Pogossian's text may help remedy this deficiency. Particularly pertinent to radiology physics training are chapters on X-ray Apparatus, The Radiologic Image, The Radiographic Image, X-Ray Image Intensification, and Cinefluorography-Photofluorography.

Chapters on x-ray apparatus, image intensification and cinefluorography should be required reading in all diagnostic radiology training programs. Excellent photographs and line drawings are included in these chapters, together with good discussions of 3 phase circuitry and energy conversion efficiencies at various stages of different image intensification and cinefluorographic systems. Physical interpretation is difficult of the unit watt/(mm.$^2$-sec.) found on page 112.

Discussion of radiologic images includes illustration of the usefulness of concepts such as subject contrast, film contrast, unsharpness, resolution, etc. One wonders whether " . . . the Poisson distributions of the statistical fluctuations of the number of photons . . . " on page 190 is really what the author is trying to say. On the same page the meaning of the following is anyone's guess: "The influence on the image of the statistical fluctuations in the number of photons forming the radiologic image on the quality of the latter can be demonstrated. . . ."

Excellent discussions of modulation transfer and line spread functions are included in the chapter on radiographic images. In the same chapter are found descriptions of parameters such as contrast improvement factor and grid selectivity used to describe characteristics of radiographic grids.

A chapter on radiation dosimetry serves little purpose in its location at the end of the book. No discussion of problems in measuring high exposure rates from pulsed x-ray generators is included during explanation of ion chamber saturation voltages. Perhaps omission of diagnostic x-ray calibration procedures is explained by the author's statement "Other subjects, such as radiation protection, were excluded because they are very well treated in other texts." However, what texts cover calibration and protection survey procedures for diagnostic x-ray generators? Other topics which probably should have been included in Dr. Ter-Pogossian's book are: physical aspects of tomography and color roentgenography and methods of kilovoltage measurement.

Chapters on The Discovery of X-Rays, Classification of Physical Manifestations and Interaction of X-Rays with Matter repeat material already covered well in many texts. These topics are not so well covered in Dr. Ter-Pogossian's book; for example, electric fields measured in volts (p. 10), matter manifesting itself as fundamental particles (p. 11), intra-atomic centrifugal forces (p. 14), corpuscular radiations defined as accelerated particles (p. 20), and a 9 page discussion of exponential absorption of x-rays (pp. 64–72). Even derivations of equations can be derived (p. 64). And just how relevant to the subject is a 24 page discussion of x-ray spectrometry?

There is good material in parts of Dr. Ter-

Pogossian's book. These parts, however, must be excised with great care from the rest of the text.

WILLIAM R. HENDEE, Ph.D.

## BOOKS RECEIVED

ATLAS OF ROENTGENOGRAPHIC MEASUREMENT. Second edition. By Lee B. Lusted, M.D., Professor of Radiology, University of Oregon Medical School; and Theodore E. Keats, M.D., Professor and Chairman, Department of Radiology, University of Virginia School of Medicine. Cloth. Pp. 250, with many illustrations. Price, $11.00. Year Book Medical Publishers, 35 East Wacker Drive, Chicago, Ill. 60601, 1967.

THE PHYSICAL ASPECTS OF DIAGNOSTIC RADIOLOGY. By Michel M. Ter-Pogossian, Ph.D., Professor of Radiation Physics, The Edward Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Mo. Cloth. Pp. 426, with 257 illustrations. Price, $13.50. Hoeber Medical Division, Harper & Row, Publishers, 49 East 33rd Street, New York, N. Y. 10017, 1967.

MUSCLES ALIVE: THEIR FUNCTIONS REVEALED BY ELECTROMYOGRAPHY. Second edition. By J. V. Basmajian, M.D., Professor and Head of the Department of Anatomy, Queen's University, Kingston, Ontario, Canada. Cloth. Pp. 422, with many illustrations. The Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, 1967.

TECHNIQUES OF RADIOBIOCHEMISTRY. By Sam Aronoff, Professor, Institute for Atomic Research; and Departments of Botany and Plant Pathology and Chemistry, Iowa State University. Cloth. Pp. 228, with many illustrations. Price, $8.50. Hafner Publishing Company, 31 East 10th Street, New York, N. Y. 10003, 1967.

NEOPLASMS OF THE STOMACH. By Gordon McNeer, M.D., Late Attending Surgeon and Chief, Gastric and Mixed Tumor Services, Memorial Hospital for Cancer and Allied Diseases and James Ewing Hospital, New York, N. Y.; and George T. Pack, M.D., LL.D. (Hon.), Attending Surgeon, Emeritus, Memorial Hospital for Cancer and Allied Diseases, and Director, Pack Medical Foundation, New York, N. Y. Cloth. Pp. 555, with many illustrations. Price, $28.50. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1967.

HISTOPATHOLOGY OF THE SKIN. Fourth edition. By Walter F. Lever, M.D., Professor of Dermatology and Chairman of the Department, Tufts University; Lecturer on Dermatology, Harvard University; Director, Dermatology Service, Boston City Hospital; Physician-in-Chief, Dermatology Clinic, Boston Dispensary; Physician (Dermatology), Boston Floating Hospital for Children; Member of the Board of Consultation, Massachusetts General Hospital; Consultant in Dermatology, Peter Bent Brigham Hospital and Robert Breck Brigham Hospital. Cloth. Pp. 794, with 338 figures. Price, $20.00. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1967.

SYMPOSIA IN RADIOLOGY FOR THE ORTHOPEDIC SURGEON. Reprinted from the Radiologic Clinics of North America. I. Radiology and the Skeletal System, Edited by Gwilym S. Lodwick, M.D. II. The Radiology of Trauma, edited by Sidney W. Nelson, M.D. Cloth. Pp. 454, with many illustrations. Price, $16.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

NOVAK'S GYNECOLOGIC AND OBSTETRIC PATHOLOGY: WITH CLINICAL AND ENDOCRINE RELATIONS. Sixth edition. By Edmund R. Novak, A.B., M.D., Assistant Professor of Gynecology, The Johns Hopkins University School of Medicine; Gynecologist, Johns Hopkins and Union Memorial Hospitals, and Greater Baltimore Medical Center, Baltimore, Md.; and J. Donald Woodruff, B.S., M.D., Associate Professor of Gynecology, The Johns Hopkins University School of Medicine; Associate Professor of Pathology, The Johns Hopkins University School of Medicine, Baltimore, Md. Cloth. Pp. 696, with 865 illustrations. Price, $20.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

HYPOPHYSENTUMOREN HYPOPHYSEKTOMIE: KLINIK, THERAPIE, ERGEBNISSE. By Professor Dr. F. Mundinger, Wissenschaftlicher Rat an der Neurochirurgischen Klinik der Universität Freiburg/Br.; and Professor Dr. T. Riechert, Direktor der Neurochirurgischen Klinik der Universität Freiburg/Br. Cloth. Pp. 467, with many illustrations. Price, Ganzleinen DM 178.-. Georg Thieme Verlag, 7 Stuttgart 1, P. O. Box 732, Herdweg 63, Germany, 1967.

CURRENT PERSPECTIVES IN GASTROENTEROLOGY. Edited by William S. Blakemore, M.D., Professor of Surgery, University of Pennsylvania School of Medicine, and Chairman of the Department of Surgery, University of Pennsylvania, Division of Graduate Medicine, School of Medicine, Philadelphia, Pa.; and L. Kraeer Ferguson, M.D., Professor of Surgery, University of Pennsylvania School of Medicine, Philadelphia, Pa. Cloth. Pp. 333, with 120 illustrations. Price, $13.50. Hoeber Medical Division, Harper & Row, Publishers, 49 East 33rd Street, New York, N. Y. 10016, 1967.

SYMPOSIUM ON TUMORS OF THE ALIMENTARY TRACT IN AFRICANS. Symposium held at Geneva, Switzerland, Nov. 29–Dec. 5, 1965. Edited by J. F. Murray, Deputy Director, The South African Institute for Medical Research, Johannesburg, South

Africa. Sponsored by the International Union Against Cancer, and supported by the Calouste Gulbenkian Foundation, Lisbon, Portugal. National Cancer Institute Monograph 25, July, 1967. Cloth. Pp. 316, with many illustrations. Price, $3.25. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, 1967.

RADIATION THERAPY. Second edition. By Walter T. Murphy, M.D., Chief, Department of Radiation Therapy, The Buffalo General Hospital, Buffalo, N. Y. Cloth. Pp. 1020, with many illustrations. Price, $45.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

ERGEBNISSE DER GESAMTEN LUNGEN-UND TUBERKULOSEFORSCHUNG. Band XVI. By R. Amgwerd, St. Gallen; St. Engel, London; A. Kluge, Münster (West.); and W. Pagel, London. Paper. Pp. 144, with many illustrations. Price, kartoniert DM 49.60. Georg Thieme Verlag, 7 Stuttgart 1, P. O. Box 732, Herdweg 63, Germany, 1967.

CERVICAL SPONDYLOSIS: AND OTHER DISORDERS OF THE CERVICAL SPINE. Edited by Lord Brain, D.M., LL.D., D.C.L., D.Sc., F.R.C.P., F.R.C.S., F.R.C.O.G., F.R.S., F.F.R., Consulting Neurologist to the London Hospital and Consulting Physician to the Maida Vale Hospital; and Marcia Wilkinson, D.M., F.R.C.P., Consultant Neurologist, Elizabeth Garrett Anderson Hospital and the Hackney and Queen Elizabeth Group of Hospital; Director, Regional Neurological Unit, Eastern Hospital, Hackney, E.9, England. Cloth. Pp. 232, with many illustrations. Price, $10.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

THE YEAR BOOK OF NUCLEAR MEDICINE. Volume II, 1967. Edited by James L. Quinn, III, M.D., Associate Professor of Radiology, Northwestern University; Director of Nuclear Medicine, Chicago Wesley Memorial Hospital; Consultant in Nuclear Medicine, Veterans Administration Research Hospital, Chicago and Great Lakes Naval Hospital, Great Lakes, Ill. Cloth. Pp. 384, with 101 figures. Price, $10.00. Year Book Medical Publishers, Inc., 35 E. Wacker Drive, Chicago, Ill. 60601, 1967.

END RESULTS IN THE TREATMENT OF SCOLIOSIS: A SURVEY OF 57 CASES. By Erik B. Riska, The Orthopaedic Hospital of the Invalid Foundation, Helsinki, Finland. Paper. Pp. 22. Acta Orthopaedica Scandinavica, Supplementum No. 102. Munksgaard, Copenhagen, 1967.

SOME ASPECTS OF LONGITUDINAL BONE GROWTH: AN EXPERIMENTAL STUDY OF THE RABBIT TIBIA By Göran Sundén, Departments of Anatomy and Orthopaedics, University of Lund, Sweden. Paper. Pp. 134. Acta Orthopaedica Scandinavica, Supplementum No. 103. Munksgaard, Copenhagen, 1967.

THE RADIOLOGIC CLINICS OF NORTH AMERICA. Symposium on Radiology and the Endocrine System. Guest Editor, E. James Potchen, M.D. Cloth. Pp. 360, with many illustrations. August, 1967, Volume V, Number 2. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

CLINICAL STUDIES OF PERIPHERAL CIRCULATION. An International Symposium held in Copenhagen, May 6–7, 1966. Edited by I. Dahn, Department of Surgery, University of Lund, Lund, Sweden; and N. A. Lassen, Department of Clinical Physiology, Bispebjerg Hospital, Copenhagen, Denmark. Paper. Pp. 248, with many illustrations. The Scandinavian Journal of Clinical & Laboratory Investigation, Volume 19, Supplementum 99. Munksgaard, Copenhagen, 1967.

TRANSACTIONS OF THE XI MEETING OF THE SCANDINAVIAN SOCIETY FOR CLINICAL CHEMISTRY AND CLINICAL PHYSIOLOGY, Aalborg, Denmark, Aug. 29–31, 1967. Paper. Pp. 150. The Scandinavian Journal of Clinical & Laboratory Investigation. Volume 19, Supplementum 100. Universitetsforlaget, Oslo, Norway, 1967.

ANGIOGRAPHIE DER UTERUSTUMOREN UND IHRER REZIDIVE. By Dr. med. Alfred Breit, Priv.-Doz. an der Universität München; Chefarzt der Radiologischen Abteilung des Städtischen Krankenhauses Passau. Cloth. Pp. 54, with some illustrations. Price, Ganzleinen DM 39.-. Georg Thieme Verlag, 7 Stuttgart 1, P. O. Box 732, Herdweg 63, Germany, 1967.

GOLDEN'S DIAGNOSTIC ROENTGENOLOGY. Edited by Laurence L. Robbins, M.D., Clinical Professor of Radiology, Harvard Medical School; Radiologist-in-Chief, Massachusetts General Hospital, Boston, Mass. Cloth loose-leaf binder. Price, $39.00. The Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, 1967.

ATLAS OF STEREOTAXIC ANATOMY OF THE TELENCEPHALON: ANATOMO-RADIOLOGICAL STUDIES. By J. Talairach, G. Szikla, P. Tournoux, A. Prossalentis, M. Bordas-Ferrer, L. Covello, M. Iacob, and E. Mempel. French-English text. Cloth. Pp. 326, with many figures. Price, 260 F. Masson & Cie, Editeurs, 120 Boulevard Saint-Germain, Paris, France, 1967.

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# ABSTRACTS OF RADIOLOGICAL LITERATURE

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## ROENTGEN DIAGNOSIS

### Head

Dodge, J. A., and Kernohan, D. C. Oral-facial-digital syndrome. *Arch. Dis. Childhood*, April, 1967, *42*, 214–219. (From: Departments of Child Health, and Child and Preventive Dentistry, Queen's University of Belfast, and Royal Belfast Hospital for Sick Children, Belfast, Ireland.)

Five patients with oral facial digital (OFD) syndrome (4 were members of one family) are reported, with a discussion of the clinical and genetic features of this condition. An excellent table-summary of the clinical features derived from the authors' material as well as material of other authors is presented.

The oral findings include a lobulated tongue with hamartomata between lobules, a short frenulum, and incomplete differentiation of the floor of the mouth. The gums show a cleft by an abnormal supernumerary frenulum. The palate is cleft; (often in the soft palate) this may be bilateral or asymmetric. There is a high vault with lateral ridges. The teeth show malposition. There may be supernumerary or absent teeth, often with enamel hypoplasia. The lips show a midline notch "pseudocleft" in the upper lip.

The facial features include hypertelorism, epicanthic folds, hypoplastic alar cartilages, and flattened hypoplastic maxillae.

The fingers and toes show polydactyly, syndactyly, camptodactyly, clinodactyly, brachydactyly, and the roentgenograms may also show osteoporosis.

Other findings include frontal bossing of the cranium and a wide nasion-sella-basion angle. The skin may be dry or seborrheic and there may be alopecia. Between 30 and 50 per cent have mental retardation. Neurologic findings include familial trembling, hydrocephalus with porencephalic cyst, hydranencephaly, and questionable agenesis of the corpus callosum. In addition, there may be polycystic kidneys.

The inheritance of the OFD syndrome is not clear. The multisystem involvement suggests, however, that a chromosomal aberration involving more than one gene is also a possibility. On the present evidence an X-linked dominant gene, lethal when hemizygous, appears to be the most likely cause of the condition.—*E. Nicholas Sargent, M.D.*

Kubin, Doris A., Amundsen, Per, and Newton, Thomas H. Orbital venography in the diagnosis of intermittent exophthalmos. *Radiology*, May, 1967, *88*, 930–934. (From: Department of Radiology, University of California School of Medicine, San Francisco, Calif.)

Intermittent exophthalmos is a condition characterized by unilateral proptosis which rapidly occurs when the head is lowered, caused by a venous abnormality within the orbital cavity, above or behind the globe. Increasing venous pressure by the Valsalva maneuver or ipsilateral neck compression may also cause proptosis and pain in the orbital area. Enophthalmos may eventually result from orbital fat atrophy due to pressure from these dilated retrobulbar veins.

Two cases of this entity are reported, in which the diagnosis was proven by opacification of the retrobulbar veins, and the extent of the varices defined. In the first case the veins were filled by catheterization of the inferior petrosal sinus via the internal jugular vein, and in the second case by injection of contrast material into the frontal vein of the forehead.

The anatomy of the ophthalmic veins and adjoining facial and intracranial venous pathways is reviewed, and different techniques of ophthalmic venography are discussed. It is pointed out that carotid arteriography is not successful in opacifying these vessels.—*James A. Cunyus, M.D.*

New, Paul F. J. True aneurysm of the middle meningeal artery, cranial Paget's disease and hypertension: a triad. *Clin. Radiol.*, April, 1967, *18*, 154–157. (From: Department of Radiology, Harvard Medical School, Massachusetts General Hospital, Boston, Mass.)

A very detailed case history is given of an older female with Paget's disease, aneurysm of the middle meningeal artery and hypertension. This brings the total number of cases of this triad reported in the literature to 3.

The interesting aspect of the case reported by the author was the presence of a pulsating mass over the right frontotemporal area with a bruit. On skull roentgenograms this could not be demonstrated to be communicating intracranially until angiography was done. It was felt that the reason of not seeing the defect was that the variation in density due to Paget's disease obscured the defect. This patient also had syphilis but it was proven that the aneurysm was due to atherosclerosis rather than luetic involvement.

The case demonstrated the difficulty in differentiating by clinical means between an aneurysm of the middle meningeal artery which has eroded through the vault and an aneurysm of the superficial temporal artery. It also helped to support the hypothesis previously presented that an aneurysm is unlikely to develop in this rare location unless predisposed by weakening of the arterial wall by intrinsic factors (developmental defects of the media and atherosclerotic changes). There was, furthermore, a loss of osseous support due to softening of overlying bone.

Paget's disease, by producing not only bone

softening but increased arterial stress due to the greatly increased meningeal arterial blood flow, seems particularly likely to predispose to aneurysm formation. The atherosclerotic involvement, as a significant factor, is indicated by consistent finding of hypertension and the age of the individual.—*Samuel G. Henderson, M.D.*

Passerini, A., and Tagliabue, G. Aneurysms of the vertebrobasilar system. *Radiol. clin. et biol.*, 1966, *35*, 257–273. (Address: Dr. Angelo Passerini, Istituto Neurologico, Via Celoria, 11, Milano, Italy.)

In this article, the authors present a brief and concise résumé of the clinical and roentgenologic features found in aneurysms of the vertebral and basilar arteries.

The most common roentgenologic findings are bone erosion and calcification within the walls of the aneurysm. The major clinical features are those of a subarachnoid hemorrhage or of a space-occupying lesion.

The authors conclude that a full exploration of the cerebral circulation is needed in those patients who have a subarachnoid hemorrhage.—*Richard P. Taylor, M.D.*

Brown, R. E. A standardized procedure for echoencephalography and for analyzing echoencephalograms. *Canad. M.A.J.*, May 20, 1967, *96*, 1349–1354. (From: Department of Diagnostic Ultrasound, Winnipeg General Hospital, and Department of Radiology, University of Manitoba, Winnipeg, Manitoba.)

Echoencephalography is a simple, convenient and atraumatic diagnostic technique for the detection of diencephalic midline displacement caused by space occupying lesions. The accuracy and reliability of this test and whether it is reproducible can be greatly influenced by the way in which the examination is performed and subsequently analyzed.

The author states that echoencephalography on 850 patients has led him to develop a standardized technique which takes into account the positioning of the patient, the application of the transducer, the identification of the midline-echo-complex, and even the elimination of observer bias.

Once performed, the test must meet certain rigid standards of acceptability as designed by the author. If it does not meet these criteria, the echoencephalogram is discarded and the examination is repeated. After it is accepted, the test is analyzed according to an established format which is described in detail.

Although the accuracy of echoencephalography has been shown to exceed 90 per cent and significant results can be effected by the methods used for making the examination and analyzing its results, there still are further limitations to echoencephalography. The examination cannot reliably detect or rule out space occupying lesions in the posterior fossa, or the frontal or occipital lobes. Bilateral lesions which do not cause midline displacement may also escape detection even though they lie in the supratentorial area. However, these restrictions may be overcome in the future as new techniques and equipment are developed.—*E. Nicholas Sargent, M.D.*

## Neck and Chest

Pfuetze, Karl H. Pulmonary disease caused by photochromogenic mycobacteria. *G.P.*, May, 1967, *35*, 85–92. (From: Suburban Cook County Tuberculosis Sanitarium, Hinsdale, Ill.)

Pulmonary disease due to *Mycobacterium kansasii* has been reported with increasing frequency. In contrast to tuberculosis, *M. kansasii* seldom occurs in more than one member of a family. The disease is three times more prevalent in men than women; however, occupation plays no role in the incidence. Chicago, Dallas, Houston, and New Orleans have reported the most cases, although it has appeared in all parts of the world. The organisms have not been isolated in soil nor in animals.

*M. kansasii* can be positively identified only by laboratory tests, although certain roentgen findings are strongly suggestive of the disease. Generally, the picture is similar to tuberculosis with the following exceptions: cavitation is more common, and cavity walls are thinner in *M. kansasii;* exudative infiltration is less frequent and less extensive than in tuberculosis; dissemination is rare; granulomatous nodules are fewer; and retraction, contraction, and pleural effusion are infrequent.

An aggressive therapy should be instituted as soon as possible—home treatment can be disastrous. A combined chemotherapy regimen should be carried out longer in this disease than in tuberculosis because *M. kansasii* has a strong tendency to reactivate.

Chemotherapy by itself often fails to provide long-term control of the disease. Surgical extirpation of all cavities provides the best protection against relapse. In this series of 82 patients, 90 per cent achieved "inactive status" through this surgical approach.—*Lois Cowan Collins, M.D.*

Tscherne, H., Schreyer, H., and Magerl, F. Pulmonale und kardiale Röntgenbefunde bei traumatischer Fettembolie. (Pulmonary and cardiac roentgen findings due to traumatic fat embolism.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1967, *106*, 703–710. (Address: Dr. H. Tscherne, Chirurgische Universitätsklinik Graz, A-8036 Graz, Austria.)

Fat emboli may occur after serious fractures, extensive thermal damage to the soft tissues, serious degree of poisoning or infection. Although fat emboli are quite common after severe fractures, they are recognized clinically in only about 1–3.4 per cent of the cases.

Various theories exist to explain the occurrence of fat emboli: (a) fat particles filter into the capillaries; (b) lipase enters the blood stream; (c) the stimulation for these activities comes from the hypothalamic center; (d) in shock, alteration of plasma proteins permits admixture with fat resulting in an accumulation of fat particles in the vascular system.

Fat embolization should be suspected, especially with an appropriate history of trauma, if the following symptoms are present: (1) cardio-pulmonary—restlessness, fear, tachycardia, dyspnea, cyanosis, hemoptysis, and chest pain; (2) cerebral—delirium, drowsiness, coma, cramps and neurologic defects. Electrocardiographic and electroencephalographic changes may be significant; also, petechiae may be abundant and changes may be seen in the optic fundi.

The roentgenologic changes in the lungs consist of areas of edema characterized by small or medium sized soft tissue densities in the middle and lower lobes but occasionally also in the upper lobes. They may become confluent and reach lobular proportions. Under proper management, the changes last approximately 6 days. The pulmonary edema may be associated with tiny hemorrhages and atelectasis, but inflammation is uncommon. The edema is probably due to obstruction of the terminal arteries in the lungs with a resultant increase in pressure in the lesser circulation. This may cause vagal stimulation which produces a disturbance in capillary permeability, especially if the brain stem is also involved. The fatty acids produce a toxic effect upon the pulmonary capillaries.

The cardiac changes are probably due to fat emboli in the coronary vessels. As a result of the circulatory changes, the myocardium shows signs of focal degeneration and necrosis. All these manifestations are reversible. A previously normal heart may become dilated (particularly the left ventricle) or resemble a typical cor pulmonale. Dilatation of the heart is certainly to be expected if there is a preexisting coronary insufficiency, cor pulmonale or valvular disease. In such cases, the changes in the cardiac configuration are less likely to be reversible; on the contrary, they may become more pronounced.—*J. Zausner, M.D.*

Fleischner, Felix G. Recurrent pulmonary embolism and cor pulmonale. *New England J. Med.*, June 1, 1967, *276*, 1213–1220. (From: Department of Radiology, Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts 02114.)

It is now well established that pulmonary embolism is a very common disease; however, the frequency of chronic embolization resulting in pulmonary hypertension and cor pulmonale, is less broadly appreciated.

Pulmonary arterial hypertension was formerly regarded as being caused by primary pathologic changes in the pulmonary vessels. Many of the changes in the pulmonary arteries are now being recognized by pathologists as organized thrombi secondary to repeated "silent" pulmonary embolism. In 1965, Freiman and co-workers carefully examined lungs in 61 consecutive autopsies, found traces of previous embolization in two-thirds of all cases, and commented that half of these would have been overlooked in routine autopsy work.

The classic clinical picture of pulmonary embolism has been much revised, for the episode may be disguised as other pulmonary, cardiac, pleural, neurologic or abdominal disease. The majority of embolic episodes occur without infarct formation, or the infarct can be incomplete, reversible and fleeting. Infarct formation can occur in the absence of pre-existing heart failure. The myth of the triangular shadow should be dismissed, and replaced with the appreciation that any pleura-based pulmonary consolidation is potentially an infarct.

Since the clinical picture and pulmonary parenchymal appearance may be of little help, the radiologist must be alert to other features of embolization. Pleural effusion, basal atelectasis and inhibited diaphragmatic motion are nonspecific, early hints. An important and specific sign is increased radiolucency and diminished vascularity denoting regional underperfusion. Depending on the extent of the occlusion, there may be signs of pulmonary hypertension with pulmonary trunk and hilar dilatation. Before discernible dilatation of the right ventricle and right atrium occurs, there may be evidence of right inflow stasis by dilatation of the superior vena cava and azygos vein.

The more advanced stages of chronic cor pulmonale present a very characteristic roentgenologic picture, with dilatation of the right ventricle, pulmonary artery trunk and hilar arteries, and oligemia of the peripheral lung fields. Other causes of cor pulmonale such as emphysema, fibrosis or kyphoscoliosis may be evident roentgenologically, much as Pickwickian syndrome, carcinomatosis, schistosomiasis or reversal of an intracardiac shunt might be suspected clinically. By maintaining a high index of suspicion for embolization, the radiologist can alert the clinician and direct confirmative examinations.

A group of cases is presented illustrating instances and variations of the clinical and roentgenologic pattern of chronic embolization.—*Rosalind H. Troupin, M. D.*

Tow, Donald E., and Wagner, Henry N., Jr. Recovery of pulmonary arterial blood flow in

patients with pulmonary embolism. *New England J. Med.*, May 11, 1967, *276*, 1153–1159. (From: Division of Nuclear Medicine, Johns Hopkins Medical Institutions, Baltimore, Md.)

Lung scanning with $I^{131}$ labelled macroaggregated human albumin was performed on 69 patients with pulmonary embolism. Scans were then obtained at varying intervals following the initial scanning. The degree of impairment of blood flow was determined by the regional concentration of radioactivity as seen on these scans. Twenty-two patients had thrombophlebitis; 29 patients had associated cardiovascular disease. In 10 the pulmonary embolism followed surgery. All patients were treated initially with heparin followed by anticoagulants by mouth unless such therapy was contraindicated. Fourteen patients had plication of the inferior vena cava.

The most frequently involved region was the right lower lung field (60 per cent) followed by the left lower lung field (52 per cent). Sixty-five per cent of patients had emboli to more than one region. Within 4 months after pulmonary embolism many patients had complete or nearly complete return of pulmonary blood flow. In the 27 patients with minimal involvement 67 per cent had complete return of flow and 75 per cent had improvement. In the group of 31 patients with intermediate involvement 38 per cent had complete return and 51 per cent had improvement. In the group of 9 patients with severe involvement 20 per cent had complete return and 70 per cent had improvement of pulmonary blood flow.

A major characteristic of the serial scans in patients with pulmonary embolism was a changing scanning pattern over a short time. Improvement was frequently observed in one region of the lung, while worsening was observed in another. This finding aided in the differential diagnosis of pulmonary embolism.—*David Morse, M.D.*

Arkoff, R. S., De Vries, P. A., Leonards, R., and Stevens, L. E. Cor triatriatum: a case report. *Radiol. clin. et biol.*, 1966, *35*, 274–280. (Address: Dr. Robert S. Arkoff, Department of Radiology, Children's Hospital, 3700 California Street, San Francisco, Calif. 94119.)

The authors report a case of a 19 month old male afflicted with cor triatriatum.

The clinical, physical and radiologic findings are described with an emphasis on the angiocardiographic diagnosis of cor triatriatum.—*Richard P. Taylor, M.D.*

Raphael, M. J., Steiner, R. E., and Raftery, E. B. Acute mitral incompetence. *Clin. Radiol.*, April, 1967, *18*, 126–136. (From: Departments of Radiodiagnosis and Clinical Cardiology, Postgraduate Medical School of London, Hammersmith Hospital, Duncane Road, London, W.12, England.)

Acute mitral incompetence due to sudden dysfunction of a previously normal mitral valve is characterized by a sudden onset of severe and progressiv exertional dyspnea and acute pulmonary eder

The roentgenographic findings are different fron. those of rheumatic mitral incompetence, in that the sudden onset is usually the result of a rupture of the chordae tendineae, the myocardium and valve are normal, and there has not been time for atrial enlargement.

The article deals with angiocardiographic, as well as plain roentgenographic, examination of 23 patients; the diagnosis was confirmed in 13 either at operation or autopsy.

The authors give a very good comparative study of the plain roentgenograms at the onset and during progressive followup as regards chamber enlargement and lung edema. They also give angiocardiographic data, as to degree of incompetence, the position of jet, and whether the anterior or posterior part of the valve is involved. These data are then compared with the findings in rheumatic mitral incompetence.

The most striking feature in plain roentgenograms is lack of abnormality. There is no atrial enlargement and the lungs are clear. As the disease progresses changes occur. There may be slight overfilling of the upper lung vessels in contrast to the lower lung vessels.

In angiocardiograms the difference between rheumatic and acute incompetence depends on the pathology. In rheumatic incompetence the onset is slow, the valve thickens with calcification, and the heart muscle is also involved. In acute incompetence the onset is sudden; therefore, the valve is not thickened, there is no calcification and the muscle is healthy.

It is important to establish accurate diagnosis since in the acute cases the prognosis after surgical treatment is good. Most of these patients have a normal heart and are good operative risks. Radiology contributes largely to the diagnosis by demonstrating the typical small heart and left atrium with normal lungs. Left ventricular cineangiocardiography confirms the presence of mitral incompetence and shows a marginal jet which suggests that the cause of the incompetence is disruption of the subvalvular apparatus rather than chronic destructive changes in the valve itself.

The roentgenograms demonstrating all of the changes are reproduced and are of excellent quality.—*Samuel G. Henderson, M. D.*

Chang, C. H. (Joseph). Holt-Oram syndrome. *Radiology*, March, 1967, *88*, 479–483. (From: Department of Radiology, West Virginia University School of Medicine, Morgantown, W. Va.)

Association of extracardiac anomalies in congenital ... disease is not infrequent and was reported as ...s 1664. The Holt-Oram syndrome was re... in 1960 describing a familial transmission of ...tal cardiac disease and upper limb deform... This report included 4 members of a family. ...hen several reports of multiple cases in a ...y or several generations of a family have ap... eared. This paper concerns 4 members of a family, a father and 3 children (2 sisters and 1 brother), presenting this syndrome. The mother and the 2 oldest children were normal. Clinically a secundum type of atrial septal defect was present in 3 cases but only proven in 1 case and skeletal anomalies limited to the thumbs and thenar muscles were present in all 4 cases. The Duane syndrome was present in 2 cases.

Chromosome studies in both parents and 2 of the 3 children with the Holt-Oram syndrome were normal. This syndrome is transmitted as an autosomal dominant trait.

The author states in a review of reported cases that atrial septal defect is the most common anomaly but it may be a ventricular septal defect. The upper extremity, usually the thumbs, are most common sites of musculoskeletal anomalies.—*C. T. Edmondson, M. D.*

Sanders, Charles A., Austen, W. Gerald, Harthorne, J. Warren, Dinsmore, Robert E., and Scannell, J. Gordon. Diagnosis and surgical treatment of mitral regurgitation secondary to ruptured chordae tendineae. *New England J. Med.*, April 27, 1967, *276*, 943–949. (From: The General Medical and Surgical Services and the Department of Radiology, Massachusetts General Hospital; and the Department of Medicine, Surgery and Radiology, Harvard Medical School, Boston, Mass.)

Preoperative diagnosis of ruptured chordae tendineae was made in only 6 of the first 15 of the 39 patients included in the study reported. However, in the remainder of the patients, the correct diagnosis was made consistently.

The criteria for diagnosis were: (1) paroxysmal cardiac symptoms; (2) abrupt appearance of a loud apical systolic murmur; and (3) radiologic demonstration of a small left atrium with clinically severe mitral regurgitation. The third criterion is the most reliable diagnostic sign. Another useful sign is the disproportionate small heart size as compared to the degree of pulmonary congestion.

In 3 patients in whom the left auricle was enlarged, the degree of mitral regurgitation was not appreciated until demonstrated by ventriculography. In general, however, angiography was useful only in confirming the fluoroscopic evaluation of chamber size. Catheterization of the left side of the heart proved most valuable in the 10 patients in whom aortic stenosis was suspected. Evaluation of the aortic valve function is important in planning the surgical approach.

Although rheumatic valvulitis was the etiology most commonly encountered, almost half of the patients had no obvious cause. Possibly, both aortic regurgitation and systemic hypertension are involved in rupture of chordae tendineae.

The authors suggest replacement of chordae *only* if the left ventricle is of normal size. In 25 patients mitral-valve replacement resulted in marked improvement in 21, indicating that this surgical approach is safe and effective, and is the treatment of choice for severe mitral regurgitation secondary to ruptured chordae tendineae.—*Lois Cowan Collins, M. D.*

Delahaye, R. P., Pannier, R., Laaban, J., and Canicave, J. C. Les aspects radiologiques des localisations thoraciques de l'amibiase: 17 observations métropolitaines. (Radiologic aspects of thoracic amebiasis; 17 metropolitan observations.) *J. de radiol., d'électrol. et de méd. nucléaire*, March–April, 1967, *48*, 173–182. (From: Services d'Electroradiologie, Service clinique de Médecine aéronautique, and Service de Médecine de l'H. M. I. Dominique-Larrey, 78—Versailles, France.)

The authors record 17 cases of thoracic amebiasis in persons who contracted the disease in the far Orient or in northern Africa. There were 16 males and 1 female ranging in age from 20 to 48 years. All the patients complained of pain referable to the liver, had positive clinical and roentgenologic findings in the chest, and some degree of fever.

It is believed that the initial infection starts in the colon before it reaches the liver, whence it is transmitted to the thoracic cavity via the portal vein or the lymphatics and occasionally by direct extension through the diaphragm. The right lung is most often involved but a rare case is found in the left lung.

Roentgenologic manifestations are most often characterized by a band-like density, chiefly in the right lower lobe with fan-shaped radiation from the hilus. The area of involvement is usually separated from the diaphragm by a fairly clear space unless a pleural effusion develops. Very often the densities are quite similar to the Fleischner lines. Multiple nodular opacities have also been observed mainly in the right lower lobe but also in the middle lobe and less frequently in the upper lobe. With suppurative involvement of the liver, pulmonary abscesses are more likely to occur. Most of these contain air-fluid levels similar to other pyogenic abscesses. The pleural reactions are usually sero-fibrinous effusions, but empyema has also been found and requires decortication. Pericardial reactions, described in the literature, were not encountered in this series.

The differential diagnosis of pulmonary and pleural amebiasis includes pneumococcal and staphylococcal pneumonia, viral and Friedländer's pneumonia, Rickettsia and neoplasms.—*J. Zausner, M. D.*

## Abdomen

Bayless, Theodore M., Kapelowitz, Robert F., Shelley, William M., Ballinger, Walter F., II, and Hendrix, Thomas R. Intestinal ulceration: a complication of celiac disease. *New England J. Med.*, May 4, 1967, *276*, 996–1002. (From: Departments of Medicine, Pathology and Surgery, Johns Hopkins University School of Medicine, Baltimore, Md.)

A patient with well documented celiac disease with a 6 year complete remission on a strict gluten-free diet is reported. The patient developed multiple intestinal ulcerations and died ultimately of perforation and abscess formation. Microscopic examination revealed normal intestinal mucosa without evidence of celiac disease up to the edge of the ulcers. These ulcerations were entirely nonspecific. Fungal, bacterial and acid-fast stains were negative. There was no evidence of regional enteritis, lymphoma or arteritis. There was no history of ingestion of enteric coated potassium tablets or diuretics.

The reports of 15 patients in the literature are added to the authors' case report. These all had well documented celiac disease or impressive presumptive evidence for this diagnosis and complications due to multiple acute ulcerations of the small intestine developed. It seems probable that ulcerations of the small intestine represent a complication of celiac disease itself.

No effective medical therapy for this ulcerative complication of celiac disease is known; it is observed despite a gluten-free diet. Corticosteroids and broad spectrum antibiotics have not been effective. Thirteen of the 16 patients have died, usually after perforation of an intestinal ulcer. Two patients had been treated with gluten-free diet and surgical resection of the multiple ulcerated areas. Both are alive and well on a gluten-free diet 2 and 6 years later. It is, therefore, possible that early resection of the ulcers and obstructed areas may alter the course of the disease.—*David Morse, M. D.*

Sargent, E. Nicholas, Guth, Paul H., and Graffman, Martin. Rectal cholecystography. *Radiology*, May, 1967, *88*, 997–1001. (From: Departments of Radiology and Gastroenterology, Orange County General Hospital, Orange, Calif.)

Rectal cholecystography is a procedure which may be of considerable diagnostic value when the oral or intravenous route cannot be used. The technique is simple, does not require special preparation, is without side-effects, and resulted in visualization of gallbladder in most of the patients studied in series reported by the authors. All patients in study were free of hepatobiliary disease.

Ipodate sodium and ipodate calcium salts used in the 43 patients examined by this technique. Each enema package contained 6 gm. of the dry ipodate salt and this was reconstituted into a solution or suspension with 60 cc. of tap water just before use. A double-blind analysis was employed in this study with neither the patient nor the radiologist knowing which salt had been administered. Diagnostically adequate visualization of the gallbladder was obtained in 58 per cent of the patients who retained the rectally administered media. Visualization of the gallbladder, however faint, was obtained in 87 per cent of the patients. No difference in adequacy of visualization between the sodium and calcium salts was observed, but a higher percentage of patients who received the sodium salt had difficulty in retaining the enema. Optimum visualization of the gallbladder was obtained in most instances 6 hours after the rectal instillation of the medium, but in several patients, the study had to be continued for an additional 6 hours to obtain adequate visualization.

The procedure used by the authors is as follows: (1) the patient should fast overnight; (2) a cleansing soapsuds enema should be given prior to the rectal instillation of the ipodate solution; no laxative should be used; (3) roentgenograms of the right upper quadrant should be taken at 4 and 6 hours after the rectal instillation of the medium. If adequate visualization of the gallbladder is obtained, roentgenograms should be taken every 2 hours up to an additional 6 hours.

The authors state that rectal cholecystography is not a substitute for oral or intravenous cholecystography but may be useful in patients with vomiting, ileus, inflammatory gastrointestinal disease, and the occasional reactions in intravenous cholecystography.—*Donald N. Dysart, M.D.*

Amberg, John R., Cholestanol-induced gallstones: radiologic observations. *Radiology*, March, 1967, *88*, 584–587. (From: Department of Radiology, University of California School of Medicine, and the Veterans Administration Hospital, Radiology Service, San Francisco, Calif.)

The author reports the results of a study in which he demonstrated that gallstones can be reliably induced in rabbits by the addition of 3-beta-cholestanol to an otherwise normal diet.

All the rabbits had normal gallbladders prior to the addition of cholestanol to the diet, demonstrated by intravenous injections of 2 cc. of 52 per cent methylglucamine iodipamide (cholegrafin). The formation of gallstones was observed by subsequent intravenous cholecystography twice a week after

the lithogenic diet was started until the time of sacrifice. All rabbits that received 1 gm. of cholesterol per day for 28 days and those that received 1 gm. per day for 14 days developed gallstones. The transition from normal to faint visualization occurred within 2 weeks and by the end of 4 weeks when the animals were sacrificed all but one had nonvisualization. Pathologic study revealed that the gallbladders were completely filled with stones. Many of the stones were adherent to the gallbladder wall, held there apparently by the thick mucin coating the mucosa.

The roentgenograms on the rabbits obtained before sacrifice showed at no time a layering phenomenon to permit diagnosis of stones. Only occasionally could distinct aggregates of stones be seen. The progressive nonvisualization of the gallbladder was thought to be due to the increasing number of the small stones which physically diminished the space and consequently the amount of contrast-laden bile that could occupy the gallbladder.

It is the author's opinion that initial stone formation appears to occur at the gallbladder wall and stones then become free in the gallbladder lumen.—*Donald N. Dysart, M.D.*

Gelford, Gerald J., Wilets, Arthur J., Nelson, Donald, and Kroll, Larry L. Retroperitoneal fibrosis and methysergide: report of three cases. *Radiology*, May, 1967, *88*, 976–981. (From: Washington Sanitarium and Hospital, Takoma Park, Md.)

Three additional cases of retroperitoneal fibrosis after methysergide therapy are reported, and several interesting features of each case are cited.

The first case presented with urinary tract obstruction bilaterally, with onset of symptoms only 5 months after initiation of methysergide (sansert) therapy for migraine. The patient also had symptoms of left iliac artery obstruction. Pyelography demonstrated bilateral pelvocaliectasis and medial deviation of the mid ureters—findings which are suggestive of retroperitoneal fibrosis. At surgery, a fibrotic mass surrounding the left ureter and common iliac artery was excised, with relief of symptoms. The right side was subsequently treated, and the patient has had no recurrence after 20 months, although she continued taking sansert for 7 months after surgery.

Cases II and III showed bilateral hydronephrosis, which resolved after methysergide was discontinued. Case III showed residual renal damage, and the patient developed angina pectoris while on this medication.

The authors point out that none of the 3 patients exceeded the recommended dosage schedule for this drug, and that the mechanism for these serious side effects is unknown. The subsidence of hydronephrosis when the drug is discontinued suggests that the ureteral obstruction is partly due to inflammation, with pressure from retroperitoneal edema, in addition to the fibrotic component. They recommend discontinuance of the medication for 1 month out of every 6, careful urologic followup, and discontinuing the medication if retroperitoneal fibrosis is detected.—*James A. Cunyus, M.D.*

## Gynecology and Obstetrics

Rubin, E. L., and Francis, H. Limitations of lateral pelvimetry. *Clin. Radiol.*, April, 1967, *18*, 213–217. (From: Departments of Radiology and Obstetrics, Liverpool Maternity Hospital, Liverpool, England.)

This article brings out again the importance of two views in pelvimetry in spite of many using only a lateral view to cut down on radiation exposure.

Roentgenograms are shown of what appears to be a normal pelvis in the lateral view, yet in the anteroposterior view there are gross oblique contractions.

The authors state that if an examination is deemed necessary it should be complete and informative regardless of the small amount of radiation to mother and fetus. These examinations are usually done near term and the exposure risk is minimal.—*Samuel G. Henderson, M.D.*

Friesen, Rhinehart F. Obstetrical problems coincidental to intrauterine transfusion for erythroblastosis. *Canad. M. A. J.*, April 15, 1967, *96*, 1079–1083. (From: Department of Obstetrics and Gynecology, University of Manitoba, and the Rh Laboratory, 735 Notre Dame Ave., Winnipeg 3, Manitoba, Canada.)

The records of 49 patients given 100 intrauterine transfusions for erythroblastosis were reviewed.

It was the purpose of the study to learn about the problems encountered.

It was found that the difficulties were not always directly related to the procedure.

Because of the wide variety of complications associated with transfusions, the author recommends that this procedure should be attempted only in centers equipped to deal with all possible obstetrical complications.—*Richard P. Taylor, M.D.*

Kane, Rosamond, Erez, Selcuk, and O'Leary, James A. Symptomatic symphyseal separation in pregnancy. *Surg., Gynec. & Obst.*, May, 1967, *124*, 1032–1036. (From: New York Orthopedic Hospital, Sloane Hospital for Women, and Columbia-Presbyterian Medical Center, New York, N. Y.)

Early recognition of symphyseal separation in pregnancy is often overlooked or misinterpreted; increased awareness of this condition will hopefully bring about prompt treatment providing relief to the

patient and shortening her distress. In 16 instances of symphyseal separation recognized in 54,376 pregnant women, roentgenograms revealed separations ranging from 0.5 to 2 cm. The degree of separation did not correlate with the severity of symptoms.

Discomfort and disability are characteristic. Pain is most frequently experienced in the pubic symphysis, sacroiliac joints, and thighs. A slow pace and ducklike waddle often result. The degree of diastasis can sometimes be determined by bidigital palpation, but can be demonstrated consistently by adequate roentgenographic examination. To demonstrate separation the patient stands on two 6 inch boxes. Pelvic roentgenograms should be taken while the patient bears weight on one foot and the other leg is allowed to hang free (one box is removed).

Histologically, there is progressive degeneration of the fibrocartilaginous tissues between the bony end plates, as marked thickening of the posterior ligament, and hypertrophy of the posterior aspects of the pubic rami. Anatomic variations, hormonal factors, and obstetric conditions (cephalopelvic disproportion, abnormal presentation, misdirection of traction with forceps) may all be implicated as contributing factors.

A combination of bedrest, trochanteric belt, and vitamin-mineral therapy has been the most effective form of management. Surgical intervention is seldom necessary.—*Lois Cowan Collins, M.D.*

## Genitourinary System

Kilpatrick, F. R. Horseshoe kidneys. *Proc. Roy. Soc. Med.*, May, 1967, *60*, 433–438. (From: Guy's and St. Peter's Hospitals, London, England.)

This article is an abridged version of an address to the Section of Urology, Royal Society of Medicine. The horseshoe kidney, originally regarded as a rare anatomic curiosity, actually occurs with an incidence of 1 in 200–400 individuals. The defect is thought to originate in the 4th–8th fetal week, with abnormal fusion of the renal blastomata interfering with independent renal rotation and cephalad migration. This accounts for the anterior relationship of the renal pelvis, as well as the frequency of low ectopic location of the horseshoe kidney.

The commonest complication is that of hydronephrosis. Ureteral obstruction may be related to a high ureteropelvic take off, or to compression by the renal isthmus or bulky lower pole. The usual potential sequelae of infection and stone formation may ensue.

On rare occasions the renal bridge may cause distal aortic compression. The author briefly reports one such instance in a patient presenting with the Leriche syndrome.

Surgery should be performed if complications supervene. Symphysiotomy (division of the isthmus) may be sufficient; however, in specific instances may be supplemented by excision of the isthmus, nephropexy or ureteropelvic plastic procedures.—*Rosalin H. Troupin, M.D.*

Hodson, C. J. The radiological contribut toward the diagnosis of chronic pyelonepl tis. *Radiology*, May, 1967, *88*, 857–8 (From: University College Hospital, London, England.)

From a radiologic point of view, changes due to chronic pyelonephritis are focal changes in the kidney which are the direct result of bacterial infection. Generalized changes resulting from bacterial action have not been described unless they follow the confluence of focal lesions. The late infective process is a localized area of fibrosis or scar which is usually large and affects the thickness of renal substance. The outer surface is pulled inward, and the renal papilla outward with secondary distortion of the calyx. The stem of the affected calyx may not be dilated unless the scar is very large. The calyx is no longer cup-shaped but presents a clubbed appearance with smooth margins. There may be numerous scars, unevenly distributed, with healthy tissue between the scars. The affected kidney is smaller than normal and the pelvis and ureter may show wall thickening and dilatation. The output of urine from the affected kidney is reduced, but no difference in the concentration is discernible between the two sides. This is in contradistinction to the Goldblatt kidney due to renal ischemia, with its diminished flow but increased concentration, and the back-pressure kidney where there is diminished flow and often diminished concentration.

Chronic pyelonephritis is reported to be the most common disease of the kidney in children. It is believed that most of the cases start in childhood. In this age group, 85 per cent of the cases with scars had demonstrable vesicoureteric reflux. The small occasional backflow into the lower third of the ureter is of doubtful importance. The more severe reflux is an obvious method of infection from below.

Leading authorities on this subject are now adopting the view that a completely reliable diagnosis of chronic pyelonephritis cannot be made on a biopsy sample or a histologic slide alone. All morphologic changes throughout the kidney, pelvicalycine system, and ureter must be taken into account, together with clinical and bacterial data and the results of radiologic studies.

The earliest radiologic sign of chronic pyelonephritis is localized narrowing of the renal substance between a calyx and the surface of the kidney. A single scar may occur anywhere but is most often found in the upper or lower poles. Bipolar scarring is a frequent finding. In bilateral disease, one kidney is always affected more than the other. It is essentially a disease of children and young adults. In the series of 100 cases of scarred kidneys reported by the author

the peak incidence of scarring was found to be in the early teen-age group. It is rarely found in middle-age people.

The coarse scarring due to chronic pyelonephritis may be simulated by fetal lobulation, infarction, tuberculosis, localized back-pressure effect, some forms of papillary necrosis, external pressure effects, and deformity of a calyx from cyst or tumor. —*A. W. Sommer, M.D.*

Dautrebande, Jacques, Duckett, Guy, and Roy, Paul. The claw sign of cortical cysts in renal arteriography: a contribution to the differential diagnosis of renal space-occupying lesions. *J. Canad. A. Radiologists*, March, 1967, *18*, 240–250. (From: Section of Angioradiology, Hôtel-Dieu Hospital, and Department of Radiology, Hôpital Jean-Talon, Montréal, Quebec, Canada.)

The appearance of renal parenchymal displacement by peripherally located cysts in the nephrographic phase during arteriography was first described by Olsson in 1961. This displacement produces a pathognomonic finding of localized protrusion of the cortex at the junction of the cortex of the kidney with the portion of the cyst protruding beyond the kidney (claw or spur).

Out of 51 cases of simple cyst in the authors' series, 41 showed this sign in the anteroposterior projection. In 10 cases it was necessary to obtain oblique views as well for conclusive demonstration of the claw sign. No case of proven malignant renal tumor was found to have a claw sign.

In the authors' series, it was felt that the claw sign was of more diagnostic value than the displacement of the peripheral vessels seen during the arterial phase in peripheral cysts.

Excellent illustrations are presented with the article.—*George A. Miller, M.D.*

Kelly, Daniel G. Renal arteriovenous fistula: a report of four cases and review of the literature. *Brit. J. Urol.*, April, 1967, *39*, 162–169. (From: Department of Surgery of the Cornell University Medical College; and the Department of Surgery, James Buchanan Brady Foundation, of the New York Hospital, New York, N. Y.)

The author reports 4 new cases of intrarenal arteriovenous fistula and reviews a case of extrarenal arteriovenous fistula by another author. Two cases were secondary to trauma, two cases were secondary to renal carcinoma and the extrarenal fistula occured post nephrectomy, as a complication in the renal pedicle in the fifth case.

The theories of origin of the idiopathic or "congenital" arteriovenous fistula are briefly discussed.

Certain specific clinical features are noted in respect to arteriovenous fistula such as decrease in circulation time, tachycardia, and high output cardiac failure. Cardiomegaly is a common finding. The bruit, however, is the pathognomonic physical sign. In some cases, where renal ischemia results from the fistula, hypertension may appear.

Roentgenographic demonstration of the fistula by means of arteriography is helpful both in establishing the diagnosis and in outlining the extent of the lesion for subsequent surgery.

The method of management is surgical and usually consists of nephrectomy. Some reported cases have been treated by partial nephrectomy or ligation of feeding vessels followed by direct excision of the lesion. The prognosis depends largely upon the nature of the underlying condition.—*Captain Edward Savolaine, MC*

Veiga-Pires, J. A., and Elebute, E. A. Urethrocystography in the male. *Brit. J. Urol.*, April, 1967, *39*, 194–120. (From: Departments of Radiodiagnosis and Surgery, Lagos University Medical School, Nigeria, Africa.)

Urethrocystography is presented by the authors as a valuable diagnostic method in diseases of the lower urinary tract in the male patient. It is easily performed and safe, and in some situations such as severe stricture or obstruction may be the only diagnostic method available.

The technique is described and is divided into three steps: (1) retrograde injection of 60 per cent triiodide contrast agent mixed with 5 per cent dextran in saline into the urethra under fluoroscopic control; (2) filling of bladder with a more dilute solution of the contrast agent; and (3) evaluation of the urethra and bladder during micturition. Asepsis is ensured in all steps. Urethrocystography is not performed during the acute stage of urethritis or immediately following instrumentation. The method is useful in investigating traumatic lesions of the urethra and bladder and is safe when carried out under fluoroscopic control.

A new anatomic-roentgenographic classification is offered in which the urethra is divided by the following demarcations: (1) cavernous urethra divided into distal and proximal parts by the genu; (2) external sphincter; (3) prostatic urethra; and (4) bladder neck. The authors feel that this classification is more in keeping with the roentgenographic image than some previous classifications.

Some roentgenographic findings in disease are described beginning with a classification and description of strictures. The clinical relevance of site and etiology of the strictures is mentioned. The retrograde injection defines the site and extent of strictures well; however, the degree of physiologic interference and obstruction is best appreciated on examination during micturition.

Filling of the prostatic and paraurethral ducts is observed as a result of inflammatory process in the urethra or prostate. Reflux into the cavernous spaces and intravasation of contrast material into the urethral venous plexus is felt to be secondary to a loss of integrity of the urethral mucosa. The incidence of this is high following instrumentation. The clinical significance is seen in that intravasation of infected urine may occur while straining during micturition. This may result in thrombophlebitis and formation of pyemic emboli. Also, sinus tracts may form in this fashion.

The impression of prostatic enlargement and external sphincter spasm are well demonstrated by this technique. The sphincter spasm is thought to result from acute urethritis and responds successfully to antibiotics. The extent and location of fistulae are also well demonstrated with the retrograde injection. Most fistulae originate in the proximal portion of the cavernous urethra but may extend in any direction from that point.

The "urethrovesical injection stream effect" is described as a phenomenon associated with bladder neck constriction. A spray-like jet of contrast medium is seen emanating from the bladder neck into the bladder in cases of constriction, while a turbulent flow is noted in the absence of constriction at this site. Roentgenograms of a bladder model with a simulated neck constriction were obtained experimentally and reproduce strikingly the phenomenon seen in patients.—*Captain Edward Savolaine, MC*

Craft, L. L., and Swales, J. D. Renal failure after cholangiography. *Brit. M. J.*, June 17, 1967, *2*, 736–738. (From: Westminister Hospital, London S. W.1., England.)

The authors report 2 cases of renal failure after intravenous cholangiography. In each case there was evidence that the patient was predisposed to develop renal failure. The first patient, who had unsuspected chronic pyelonephritis and a mild degree of jaundice, became anuric immediately after cholangiography. The second, who had only one kidney, developed oliguric renal failure after cholangiography performed within 24 hours of a laparotomy.

The first patient was a 77 year old woman who was jaundiced. She was given an intravenous injection of 35 ml. of 50 per cent solution of iodipamide (biligrafin forte). The patient collapsed with diarrhea and vomiting and later became breathless with cold cyanosed extremities. Her blood pressure was 110/70. She recovered spontaneously but anuria developed. Five days after the cholangiography she passed no urine at all and in 3 days her blood urea was 249 mg./100 ml. Her death occurred 5 weeks after the cholangiography. An autopsy of the kidneys which were not contracted showed an irregular cortex with adherent capsule showing changes of a chronic pyelonephritis. Because of postmortem autolysis a histologic diagnosis of acute tubular necrosis could not be definitely established.

The second patient was a 42 year old man. After intravenous injections of 20 ml. of 50 per cent iodipamide he collapsed and remained hypotensive for 10 minutes. He had an operation for a large pancreatic cyst on the next day and following the operation he became oliguric and passed 300 ml. of urine the day of operation and 100 ml. the day after. An intravenous injection of 20 gm. mannitol failed to produce a diuresis. The oliguria persisted and the blood urea rose to a maximum of 287 mg./100 ml. on the tenth postoperative day. Fortunately, his renal failure was not severe enough to require dialysis. Diuresis ensued, and 6 weeks after the operation his blood urea and urine output were normal. His health since has been excellent.—*Peter C. Truog, M.D.*

Lange, H., and Gossmann, H. H. Metastatic calcification during peritoneal dialysis for terminal chronic glomerulonephritis. *German Med. Monthly*, May, 1967, *12*, 220–222. (Address: Dr. H. Lange, Medizinische Universitätsklinik, Mannkopffstr. 1, Marburg/Lahn, Germany.)

The appearance of soft tissue calcifications in chronic uremia may be related to acidosis, secondary hyperparathyroidism or administration of vitamin D. It has recently been noticed that metastatic calcifications occur after repeated dialyses.

The authors report a case of a 36 year old woman in a terminal stage of glomerulonephritis who was treated by peritoneal dialysis. Three weeks after starting treatment metastatic plaque-like calcifications appeared in the skin overlying pressure points. Calcification of the medium-sized arteries of the extremities were seen on roentgenograms. At postmortem examination this was found to be in the media of the vessels. Nephrocalcinosis, a calcified cardiac infarct and deposits of calcium in the Kupffer cells, secondary hyperplasia of the parathyroids and evidence of hyperparathyroidism in the skeleton were also demonstrated at postmortem study.

The relationship between secondary hyperparathyroidism, the alteration of minerals during dialysis and the appearance of calcifications is discussed.

Aluminum hydroxide blocks the absorption of phosphate and may be considered the treatment of choice of such calcifications. The raised calcium-phosphate ratio is thus lowered and this in turn diminishes secondary hyperparathyroidism. The authors therefore advise the administration of aluminum hydroxide to patients in chronic renal failure, even before instituting dialysis.

Treatment with the aluminum hydroxide is still justified as a therapeutic measure, in view of the fact that others as well as the authors have observed regression of the calcifications. Furthermore, de-

creasing the calcium concentration of the dialysis solution from 9 to 6 mg. per cent seems to be a more reliable way of preventing metastatic calcifications. —*Peter C. Truog, M.D.*

KAUFMAN, JOSEPH J., DIGNAM, WILLIAM, GOODWIN, WILLARD E., MARTIN, DONALD C., GOLDMAN, RALPH, and MAXWELL, MORTON H. Successful, normal childbirth after kidney homotransplantation. *J.A.M.A.*, April 24, 1967, *200*, 338–341. (Address: Dr. Kaufman, Department of Surgery, University of California School of Medicine, Los Angeles, Calif. 90024.)

The first normal delivery at term after a kidney homotransplantation occurred at the Medical College of Virginia. The authors present a second case which was delivered at the University of California, Los Angeles, Center for the Health Sciences.

Both cases received transplants from a living relative a little more than a year earlier. Renal function remained excellent throughout pregnancy. Normal vaginal deliveries at term occurred in each case. The newborn children appear to be normal despite maintenance of immunosuppressive therapy during pregnancy.

The authors suggest that there may be an enhanced graft tolerance during pregnancy.—*George A. Miller, M.D.*

## SKELETAL SYSTEM

VAN DER HEUL, R. O., and VON RONNEN, J. R. Juxtacortical osteosarcoma: diagnosis, differential diagnosis, treatment, and an analysis of eighty cases. *J. Bone & Joint Surg.*, April, 1967, *49A*, 415–439. (From: Department of Pathology of The Netherlands Cancer Institute, Amsterdam, and the Department of Radiology, University Hospital, Leiden, The Netherlands.)

Sixteen cases of juxtacortical osteosarcoma from the archives of the Netherlands Committee on Bone Tumors were analyzed and compared with 64 cases from the literature. The clinical, roentgenologic and pathologic features were studied in order to shed light on the natural history and prognosis of this tumor, evaluate the results of treatment, and study the relation of this entity to intra-osseous osteosarcoma.

In contradistinction to the clinical setting of intra-osseous osteosarcoma, four-fifths of the patients were over 20 years of age, and there was a 3:2 female to male predominance. The duration of the symptoms was longer, and the presenting complaint was more often mass than pain.

Roentgenographically, the tumor was found at the metaphyseal region of a long bone, appearing as a lobulated, densely calcified mass attached to bone by a sessile base. It has a tendency to encircle the shaft of the bone, and except at its sessile origin, remains separated from the bone by a narrow radiolucent zone. In a few cases the underlying cortex was invaded. Osteogenic sarcoma is usually readily differentiated by extensive bone destruction, sunburst striations, Codman's triangle and secondary soft tissue extension. Differentiation from myositis ossificans may pose more of a problem; however, prior trauma, angular borders and a more distinct trabecular pattern are characteristics of myositis ossificans. Subperiosteal hematoma, osteochondroma and extra-osseous osteosarcoma can also be considered in the roentgenologic differential diagnosis.

The histologic picture is quite variable, with areas of cellular connective tissue, giant cells, osseous tissue, and cartilage. The basic pattern of the connective tissue was fibrosarcomatous with only moderate pleomorphism. Only in one-fifth of tumors was the histology clearly malignant. The pattern of bone deposition was variable, but lamellar features of cortical bone were frequent, as opposed to the disordered atypical pattern seen in intra-osseous osteosarcomas.

As is now appreciated, the prognosis of juxtacortical osteosarcoma is far better than that of the intra-osseous tumor, although recurrence following local excision is common. If the histology is low grade malignancy and the tumor of limited size, a wide en block local removal is justified. However, for more extensive growths or treatment of a recurrence, amputation or disarticulation is recommended. Radiation therapy does not appear to affect the growth of this tumor.—*Rosalind H. Troupin, M.D.*

## RADIOISOTOPES

BECK, R. N., CHARLESTON, D. B., EIDELBERG, P., and HARPER, P. V. The Argonne Cancer Research Hospital brain scanning system. *J. Nuclear Med.*, Jan., 1967, *8*, 1–24. (From: The Argonne Cancer Research Hospital operated by the University of Chicago for the United States Atomic Energy Commission, Chicago, Ill.)

The authors describe a multidetector brain scanner consisting of 4 collimated scintillation detectors arranged in opposing pairs and capable of scanning the head simultaneously.

The recording system consists of light projectors coupled with the detectors and produces bell shaped spots on film for each selected photopeak pulse.

Count rate meters are provided for each channel, but not used for background erase or contrast enhancement.

Ideal imagining systems are discussed and it is stated that, in practice, we do not observe a perfect image of the projected distribution. However, attempts are being made to minimize artifacts in the

ACRH brain scanner without increasing time and radiation exposure to the patient.

Artifacts due to statistical structure, reduced contrast, spot structure, line structure, variable line spacing, scalloping, etc. are described in great detail.

Special features of this scanner, such as calibration system, electronic circuits, scan control system, etc. are also described.

Eight hundred patients were scanned with $Tc^{99m}$ and the clinical results on the first 182 scans are analyzed.—*Abbas M. Rejali, M.D.*

Bonte, Frederick J., and Curry, Thomas S. Technetium-99m HSA blood pool scan diagnosis of an intracardiac myxoma. *J. Nuclear Med.*, Jan., 1967, *8*, 35–39. (From: Department of Radiology, The University of Texas Southwestern Medical School, Parkland Memorial Hospital.)

The authors summarize the uses of the radioisotope blood pool scan and report 1 patient with an enlarged liver, pulsating neck, and progressive enlargement of the cardiac silhouette without any enlargement of the pulmonary vessels. A blood pool scan with $Tc^{99m}$ tagged human serum albumin failed to show suspected pericardial effusion, but demonstrated a filling defect within the right side of the cardiac blood pool which proved to be due to an intracardiac myxoma.

It is suggested to scan the patient with progressive increase in cardiac silhouette, unaccompanied by an increase in the pulmonary vascular volume, to rule out not only pericardial effusion but occasional intracardiac neoplasm.—*Abbas M. Rejali, M.D.*

## CHEMOTHERAPY

Park, Wallace. Clinical choriomatosis. *Proc. Roy. Soc. Med.*, March, 1967, *60*, 235–237. (From: University Department of Pathology, Queen's College, Dundee, Scotland.)

The author discusses the cryptic character of chorion carcinoma and proposes classification and analysis of trophoblastic disease that might be more helpful in view of the increasing use of chemotherapy, the earlier diagnosis of trophoblastic disease and its wider investigation. In view of the diagnostic and therapeutic campaign organized by the Union Internationale Contre le Cancer throughout the Pacific and Far Eastern areas, the comparison of different regimes of therapy must be made accurately. Therefore, there is an urgent need for a comprehensive scheme of classification and analysis of trophoblastic disease.

The author contends that this could be achieved to some extent by adding supplementary data to the diagnosis (a pointer to the preceding pregnancy, the anatomic extent of the lesion, the interval since the apparently causative pregnancy and the means whereby the diagnosis was made).—*Luther W. Brady, M.D.*

Cope, E., and Robinson, A. W. The follow up of hydatidiform mole. *Proc. Roy. Soc. Med.*, March, 1967, *60*, 237–239. (From: Dudley Road Hospital, Birmingham, England.)

The new diagnostic techniques and powerful modes of therapy being used for trophoblastic disease have yet to be satisfactorily evaluated.

The authors present the hazards of drug resistance and iatrogenic disease as being especially great and as regression is the rule rather than the exception, the argument for conservative management of the borderline cases is very strong. They discuss the value of serial angiograms and quantitative gonadotrophin estimations in the conservative management of borderline cases of trophoblastic disease and point out that these new diagnostic methods in the study of the natural history of the disease must be utilized.

Four cases are reported illustrating the value of the procedures in the management of hydatidiform mole.—*Luther W. Brady, M.D.*

Tow, S. H. The pulmonary lesion in chorion carcinoma. *Proc. Roy. Soc. Med.*, March, 1967, *60*, 239–240. (From: University Department of Obstetrics and Gynaecology, Kandang Kerbau Hospital, Singapore, Singapore.)

The author reports his experiences in the treatment of 55 cases of pulmonary chorion carcinoma admitted to the University Unit at the Kandang Kerbau Hospital in Singapore from 1959 to 1966. Pathologic material from 14 autopsies and 4 lung resections were available for study.

In the diagnosis, the hemagglutination-inhibition test for chorionic gonadotrophin and chest roentgenography were of great help. The roentgenographic appearances varied from "normal" to "heavy vascular markings," "diffuse mottlings," "multiple nodular opacities," "cannon ball opacities," "cotton wool appearance," and "hemothorax." The typical lesion on macroscopic examination was a hemorrhagic nodule and microscopically revealed trophoblast lodgements in arteries with thrombus formation and invasion of the vessel wall, hemorrhagic nodules infiltrated with chorion carcinoma cells, chorion carcinoma invading the lung parenchyma, lung infarction or villus formation. The healed lesion disappeared completely even though in a few instances roentgenographic opacities persisted.

Treatment consisted of the use of prophylactic hysterectomy in the treatment of hydatidiform mole in certain "risk" groups and has shown its effectiveness in preventing malignant sequelae, pulmonary disease and death. Therapeutic measures were surgery and chemotherapy, the chemotherapy being the use of methotrexate and 6-mercaptopurine.

Of the 55 patients, 30 have died with an over-all survival rate of 45 per cent. Seven patients were eated by prophylactic hysterectomy for 100 per nt survival, 13 patients by chemotherapy alone for 54 per cent survival, 24 patients by hysterectomy d chemotherapy with a 46 per cent survival.

Chemotherapy has increased the chances of survival from zero to nearly 60 per cent and prophylactic hysterectomy to 100 per cent.

The causes of death in the 30 patients were cerebral hemorrhage and pulmonary insufficiency. The prognosis was largely determined by the extent of pulmonary involvement.—*Luther W. Brady, M.D.*

BAGSHAWE, K. D. Gonadotrophin excretion, pelvic arteriography and treatment in post-molar trophoblastic disease. *Proc. Roy. Soc. Med.*, March, 1967, *60*, 240–242. (From: Charing Cross Group of Hospitals, Edgar Research Laboratory, Fulham Hospital, London, England.)

It is unusual to find evidence of trophoblast surviving after term delivery or early termination of normal pregnancy. By contrast, trophoblast commonly survives after evacuation of hydatidiform mole, its location being in the uterus, pelvic vasculature or lungs but not well defined. Its presence would be indicated by continued production of chorionic gonadotrophin. This persisting trophoblast usually dies out spontaneously but bioassays may be positive more than 60 days after evacuation.

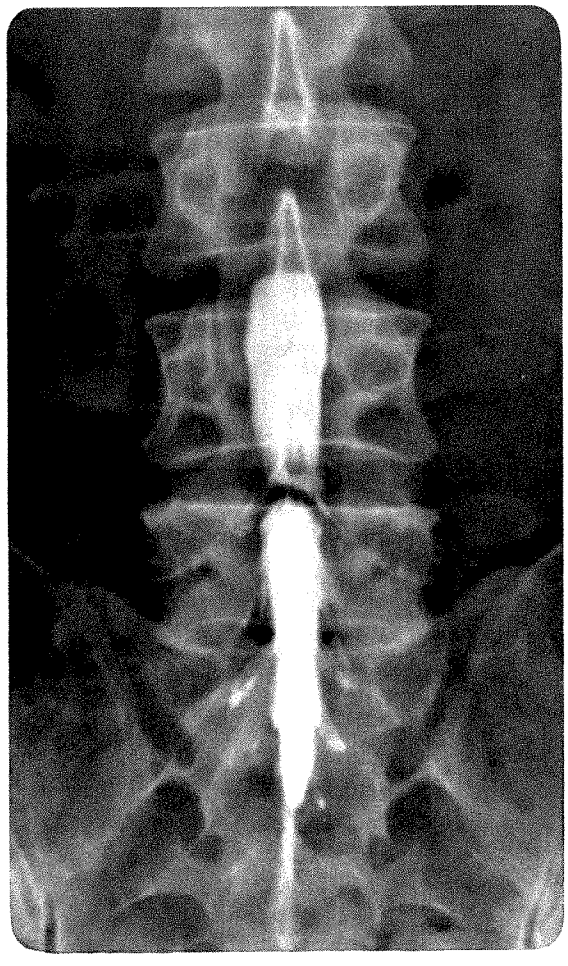
Arteriographic examination of patients with persisting post-molar trophoblast shows a high incidence of gross abnormalities. It is a study that can be used to follow the course of the disease and its response to therapy.

Several factors must be taken into account when deciding upon treatment. The longer the time interval after evacuation, the greater is the risk of overt malignancy and of cerebral metastases and the less certain the response to chemotherapy. Treatment may be precipitated by symptoms such as uterine or vaginal hemorrhage, abdominal or chest pain or dyspnea. It may be acceptable to withhold treatment in the presence of a falling titer. Patients with lung lesions run a considerable risk of intracranial spread. Precise quantitative HCG (hemagglutination—inhibition test for chorionic gonadotrophin) assays may provide information relative to treatment; follow-up examination indicates in many instances those patients who should be treated.

The author reports his experience with 21 patients with nonmetastatic post-molar trophoblastic disease who have been treated in the 40 months prior to this report. Only 3 of them had children, and only 1 had more than 2 children. In 15 patients the mole was the first pregnancy. All patients had arteriographic abnormalities, and all had HCG titers.

The author describes the treatment of the lesion by use of an infusion technique with methotrexate combined with folinic acid and 6-mercaptopurine, given orally. The treatment was continued for 4 to 8 courses untill gonadotrophin values had been in the normal range for 4 to 6 weeks. All of the patients responded fully to the treatment and relapse has not occurred. Toxicity was negligible and transient. The complications to the treatment were minimal. Four patients have subsequently had normal pregnancies, and 4 others are currently pregnant.

The author suggests that carefully controlled chemotherapy of post-molar patients who are demonstrably at great risk is preferable to the indiscriminate use of prophylactic chemotherapy for all patients with mole.—*Luther W. Brady, M.D.*



The patient, a 46-year-old male, had persistent low back pain radiating down both lower extremities posteriorly, and numbness and pain over both heels. X-ray examinations of back and hips, made 18 months before, following an automobile accident, were reported as normal.

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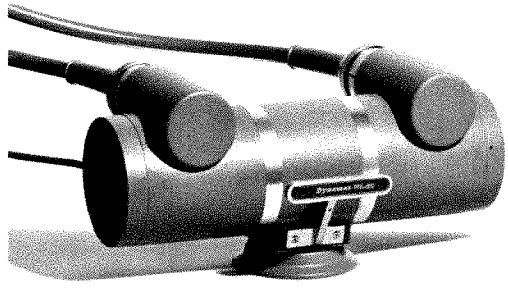
For detailed specifications on this new ultrasonic echograph and for more information on the clinical utility of these techniques, please request file 120.

References: (1) Ford and Ambrose, Brain 86 (1963). (2) Feigenbaum, Annals of Internal Medicine 65 (1966). (3) Gordon, Bio-Medical Engineering Dec. (1965).

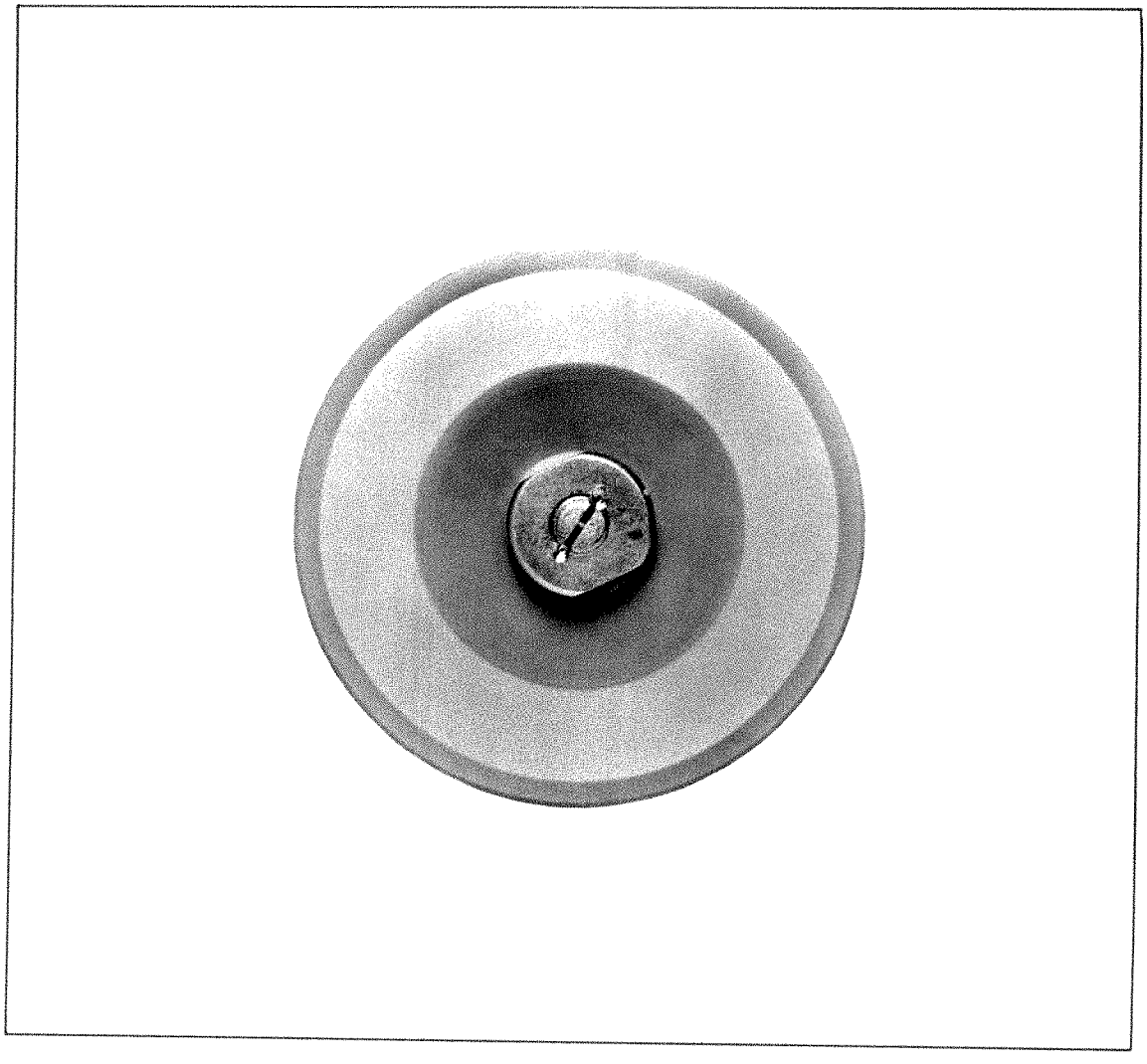


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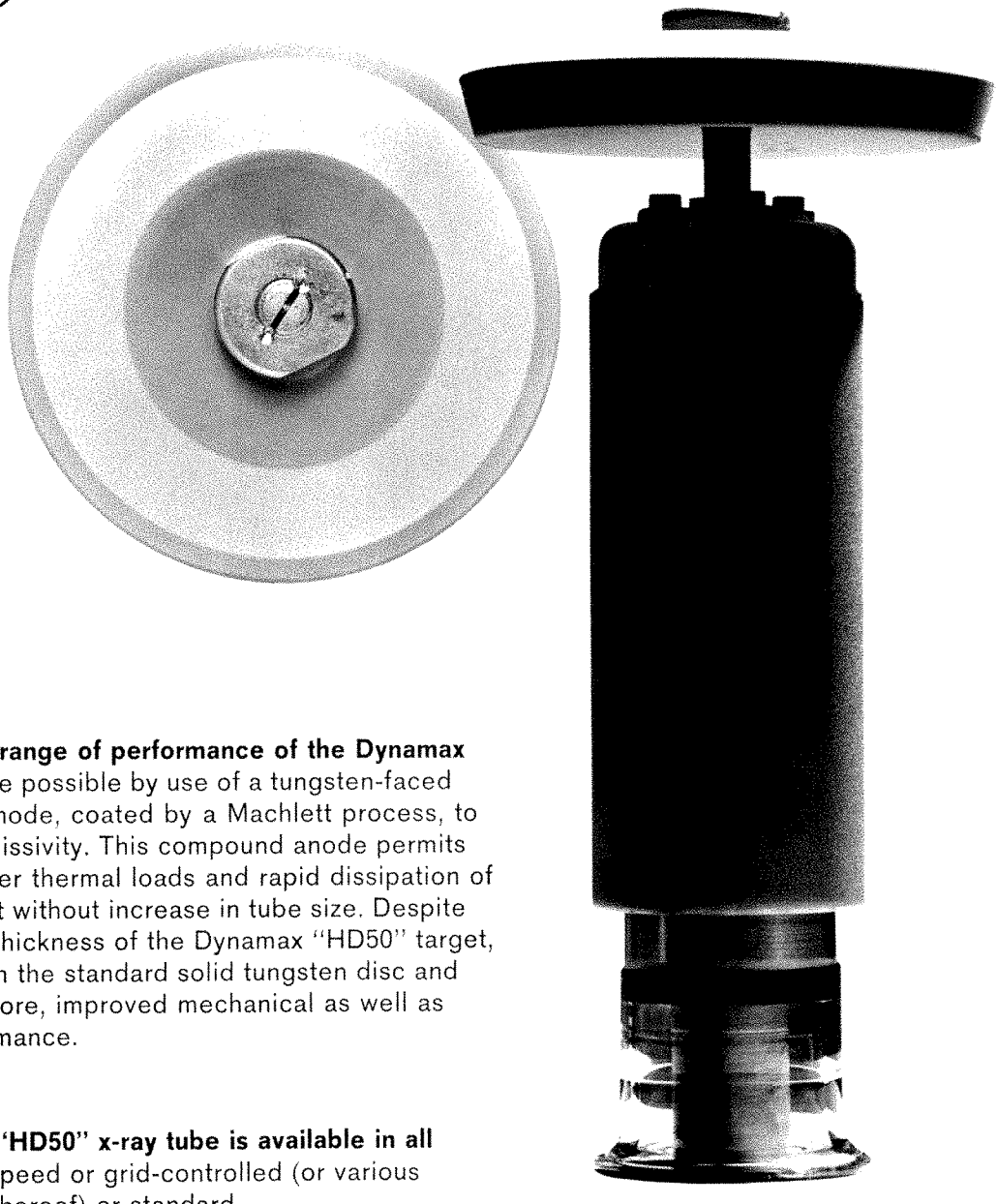
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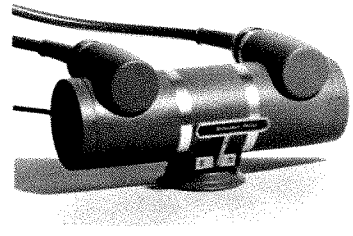


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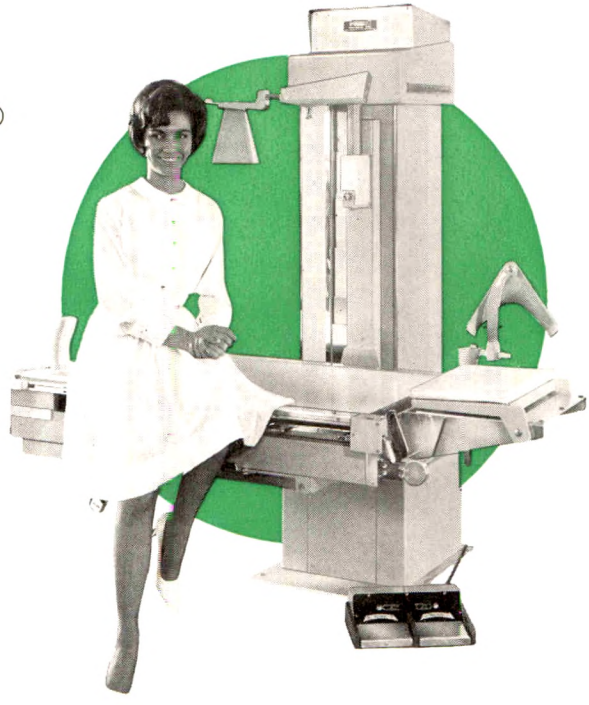
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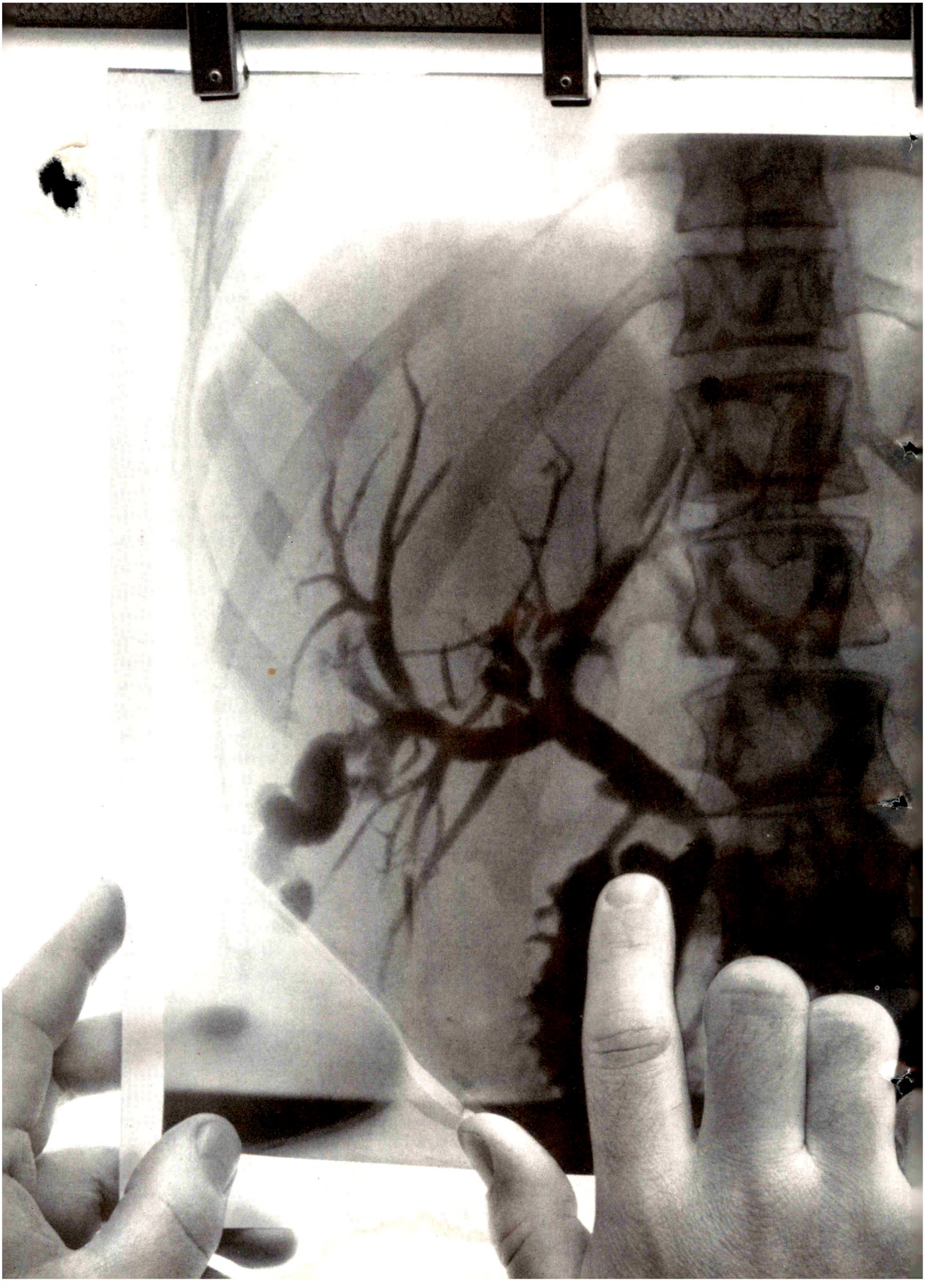
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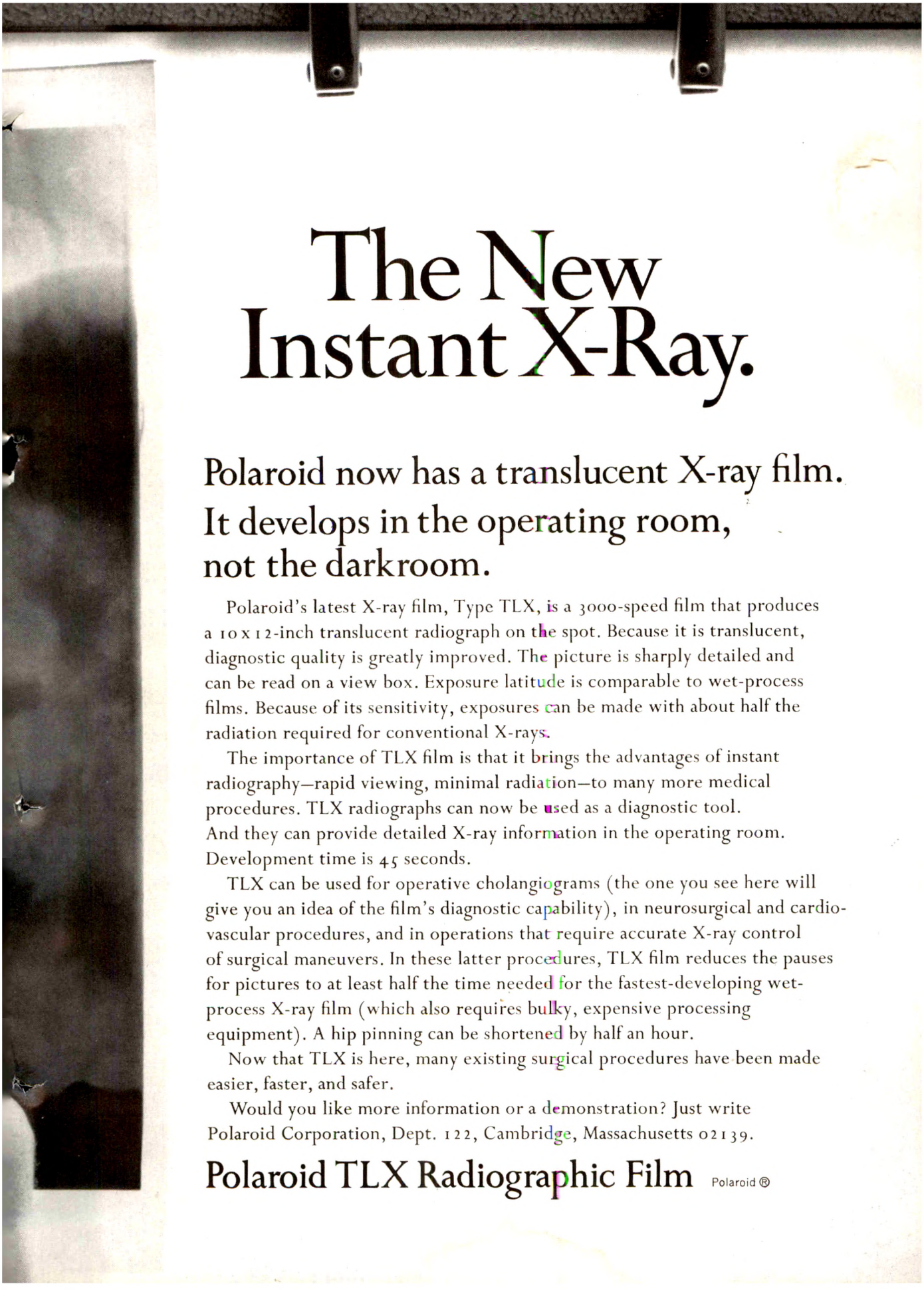
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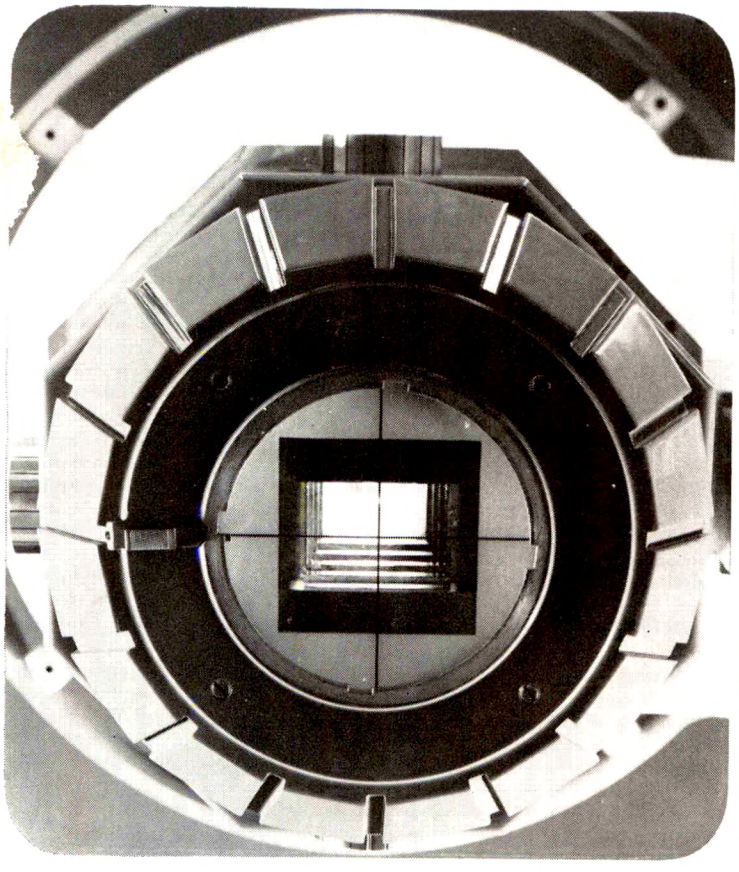
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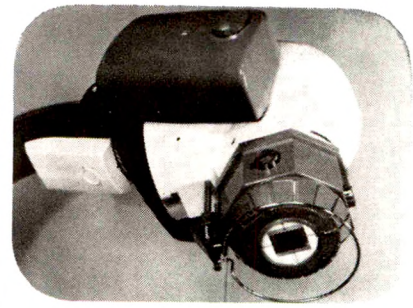
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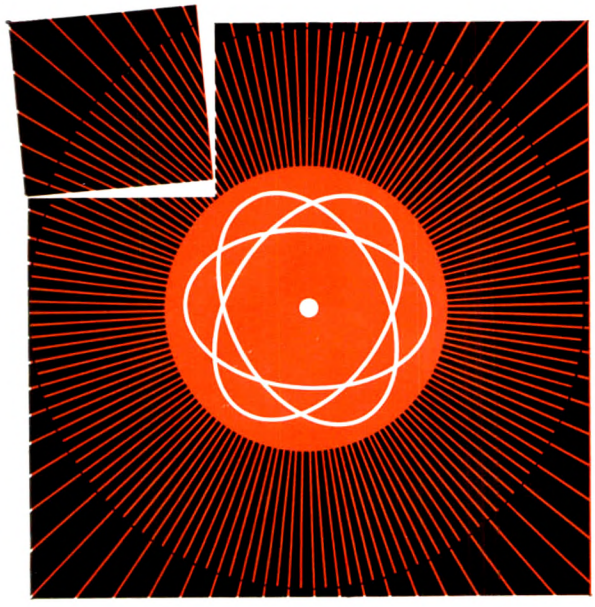
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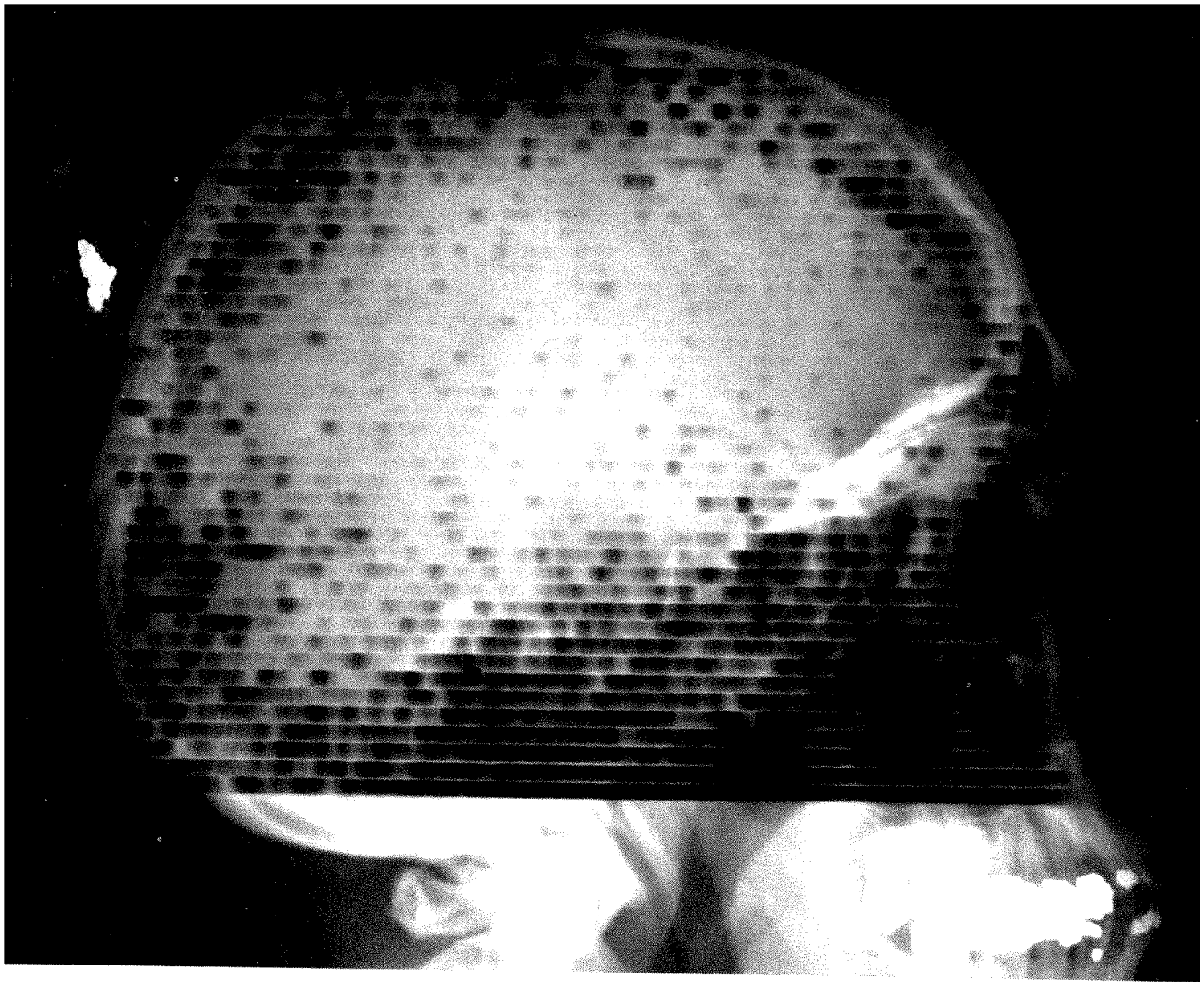
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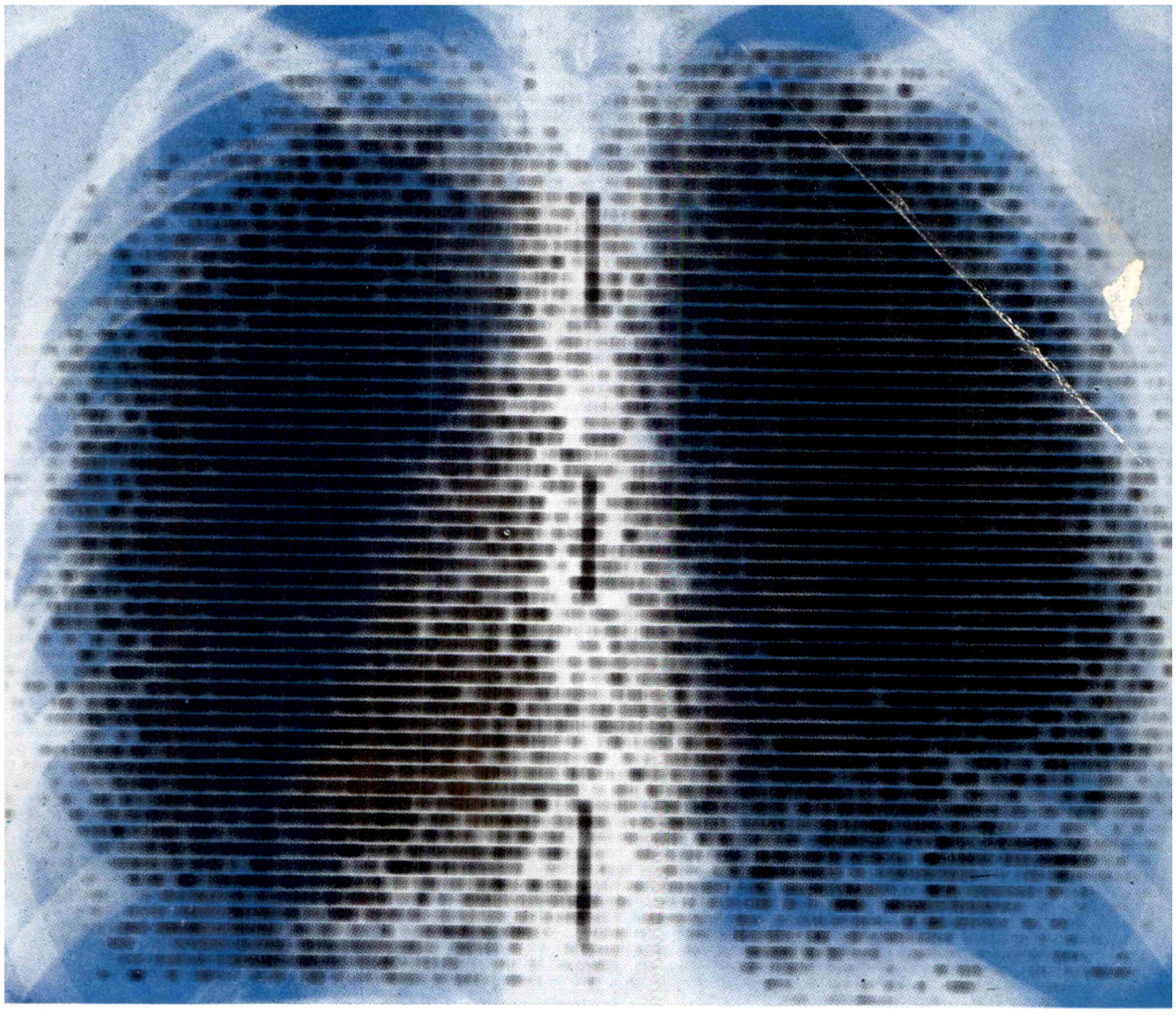
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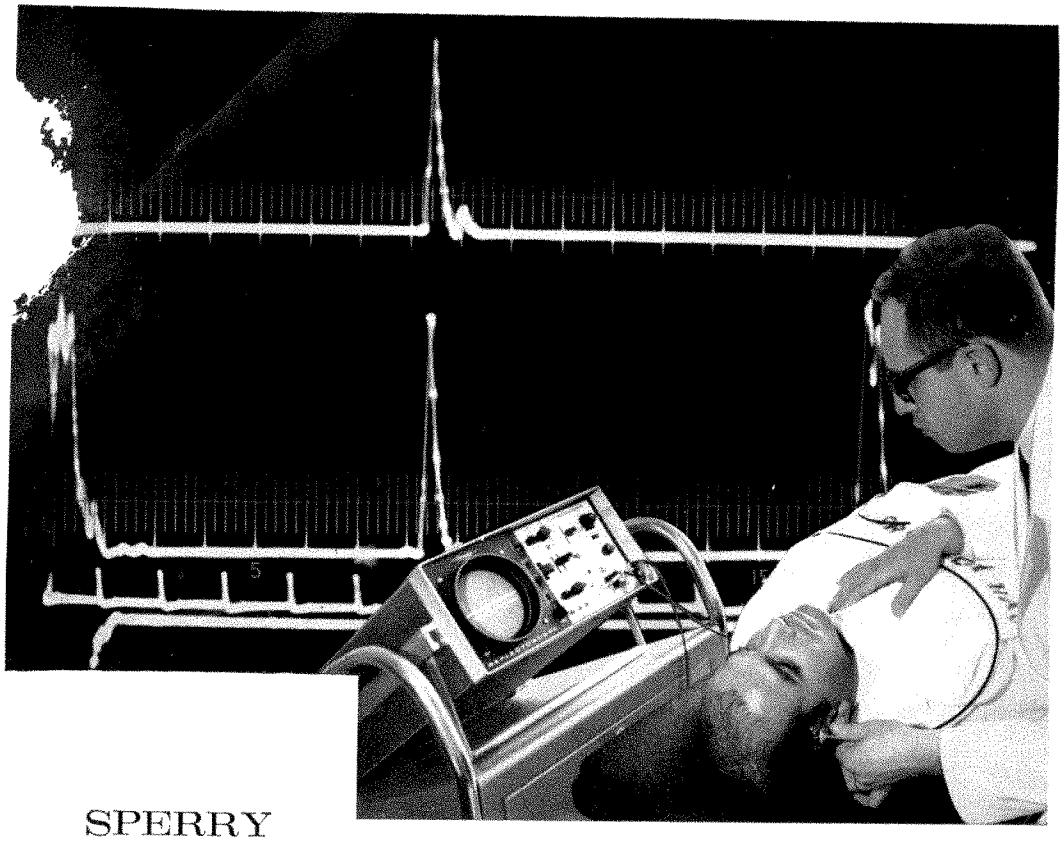
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1. S. Jeppsson, Acta, Chir. Scand 119 (1960) 445-462; Suppl 272(1961)
2. A. Jefferson, Acta Neurochirurgica, x4(1962) 392-409
3. R. Ford and J. Ambrose, Brain 86(1963) 189-196


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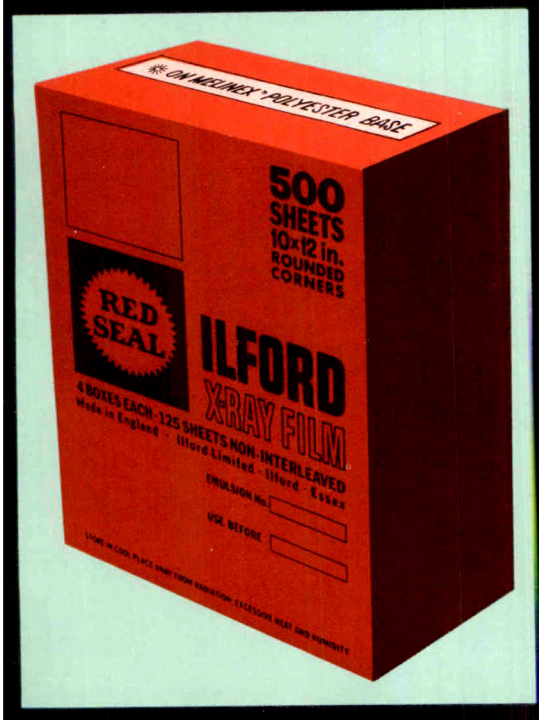
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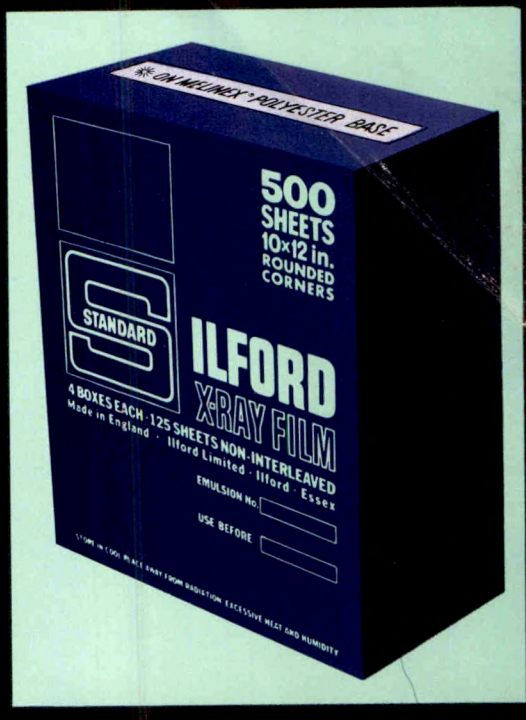
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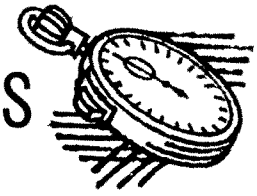
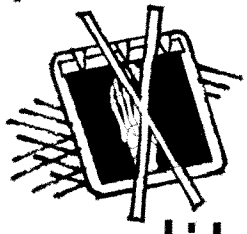
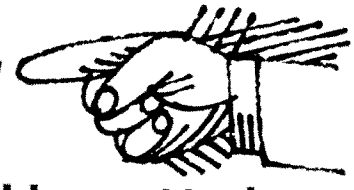
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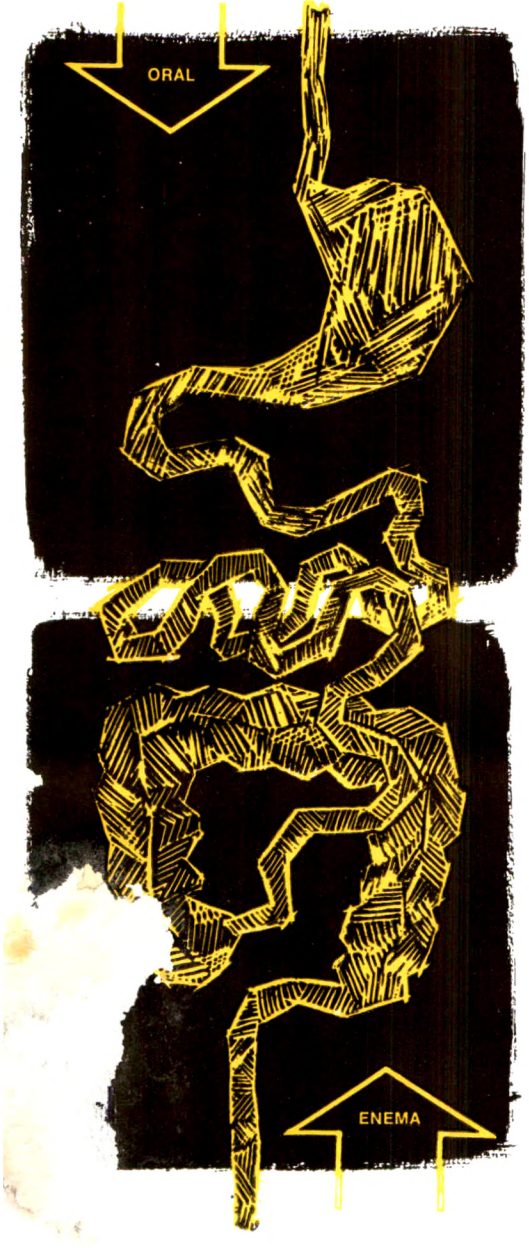
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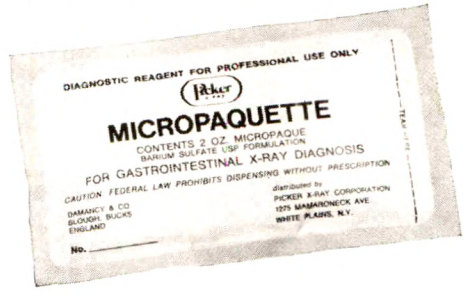
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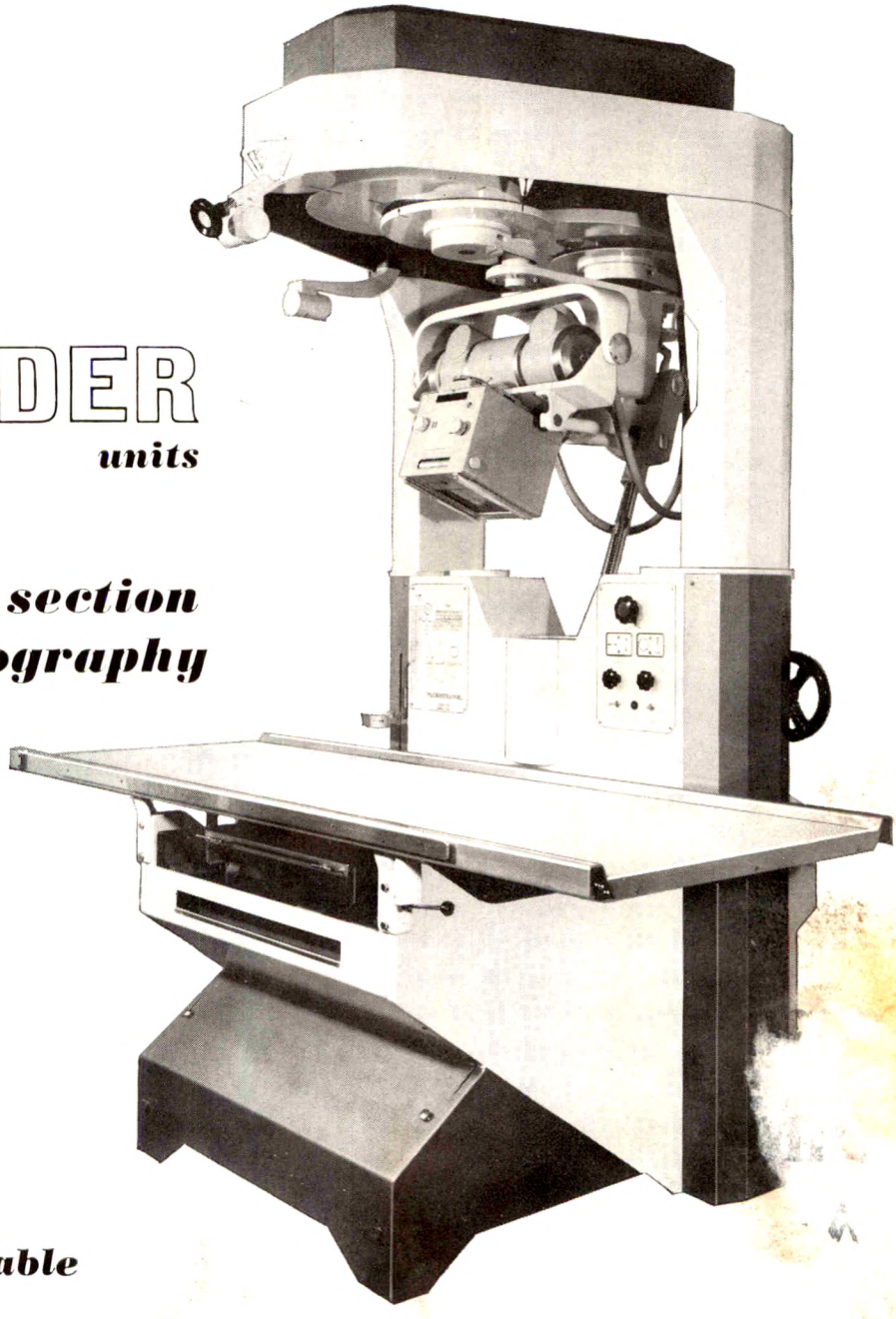
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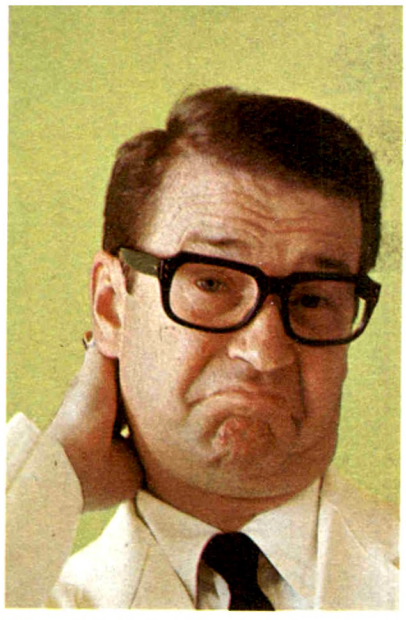
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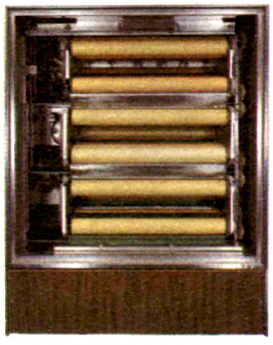
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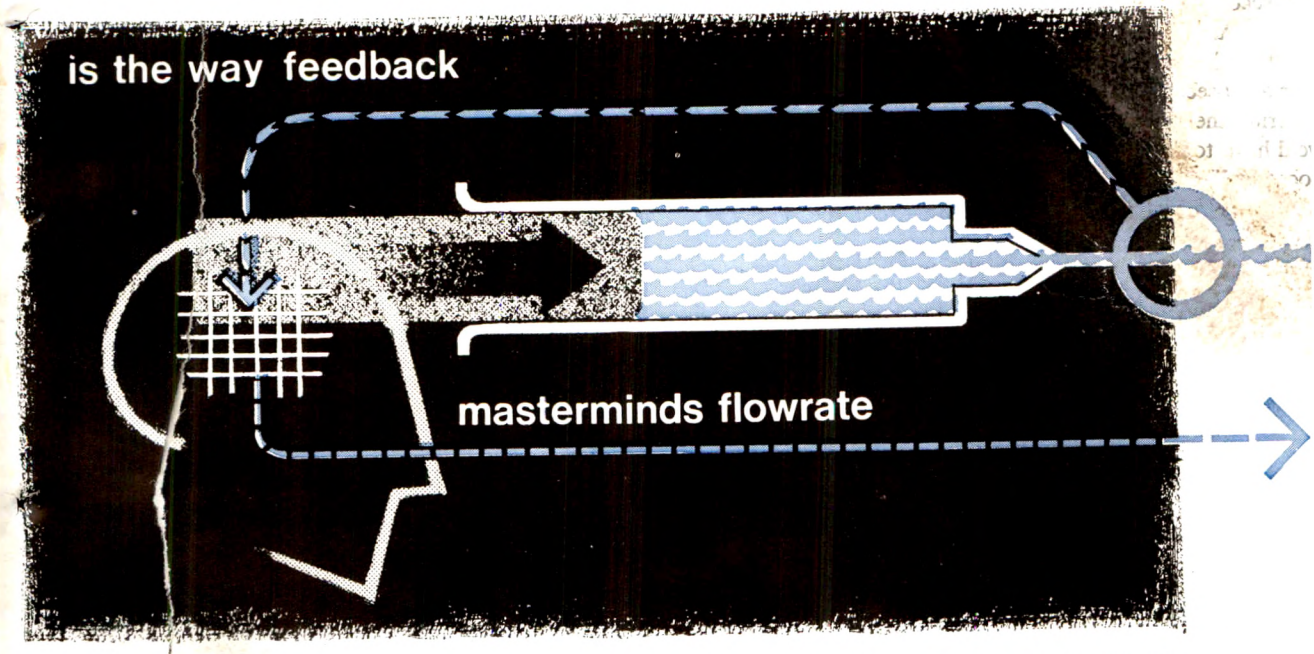
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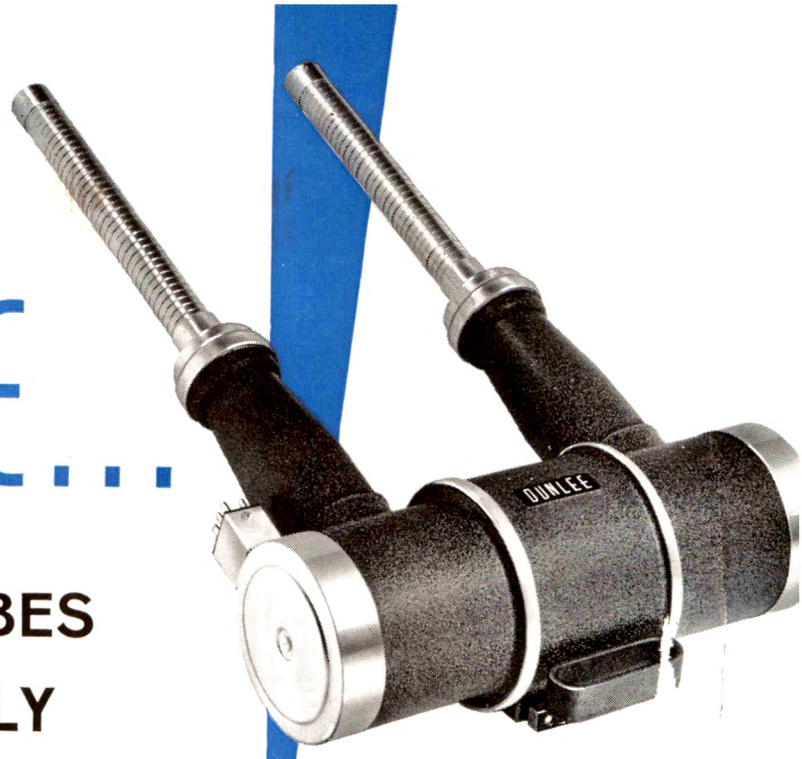
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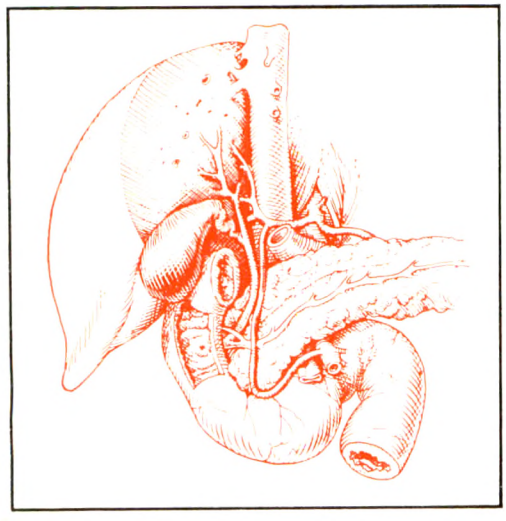
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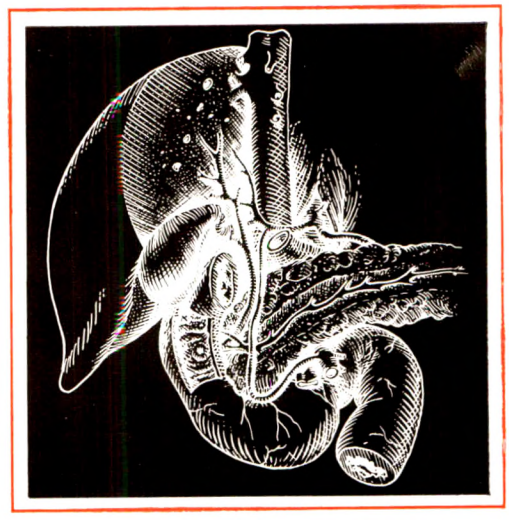
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**definition of better toleration**

**low incidence of untoward reactions**—In contrast to findings with other cholecystographic agents, Lewitan and Garcia[5] reported in their 246 patient study: "With

ipodate calcium...there was no clinical evidence of immediate or delayed nephrotoxicity in any of the patients given it...."[5] To study possible renal toxicity, "Creatinine clearance tests were also done before and after administration of multiple doses in excess of 6 gm. in 8 cases, and showed no significant alterations."[5] However, "Multiple doses beyond 6 gm. are not recommended."[5] In their study of 120 patients, Glenn and O'Brien reported "...no reactions in this series of patients attributable to the administration of [Oragrafin]...."[4] And, McCrory reported that Oragrafin was "...used routinely in cholecystographic studies in approximately 2000 patients with excellent diagnostic films and only rare and mild reactions."[6]

**side effects compared to iopanoic acid**

side effects encountered in administration of 3 Gm. of Oragrafin Sodium Capsules and 3 Gm. iopanoic acid *

| | Oragrafin Sodium Capsules (Squibb Sodium Ipodate) | iopanoic acid |
|---|---|---|
| no. of cases | 99 | 105 |
| no. of side effects | | |
| nausea | | |
| slight | 10 | 14 |
| severe | 2 | 6 |
| vomiting | 0 | 1 |
| diarrhea | | |
| mild | 7 | 22 |
| severe | 3 | 16 |
| cramps | 5 | 15 |
| dysuria | 9 | 13 |
| total/per cent | 36 (36.3%) | 87 (82.9%) |

*Adapted from White, W. W., and Fischer, H. W.[2]

Juhl,[3] in his study of 200 patients (100 on each agent), found no significant difference between iopanoic acid and sodium ipodate in the incidence of nausea; vomiting occurred in an equal number of cases.

## definition of convenience:

# Oragrafin® Calcium Granules

## Squibb Calcium Ipodate

**visualization of poorly functioning gallbladder reduces the need for I.V. studies**—In a concentrating gallbladder, Oragrafin Calcium Granules (Squibb Calcium Ipodate) will show storage, concentration, delivery or stones. A patent biliary duct can generally be expected to visualize. Provided the cystic duct is patent, Oragrafin will visualize the normal gallbladder, the abnormal gallbladder containing papillomata, nonopaque stones or radiodense calculi, and gallbladders where concentrating power is diminished.

**patient convenience**—Routine cholecystography night-before procedure is easy for patients to follow; palatable granules further enhance patient acceptability. Rapid clearance of medium permits same-day administration and gallbladder and ductal films, and, if necessary, same-day re-examination; reduces need for I.V. studies.

Optimal concentration in the he atic and biliary ducts usually occurs with hours. Although the gallbla opacified 10 hours after agent, diagnostically valu can often be obtained with

**rapid absorption permits same-day re-examination**— cause of nonopacification af lecystography, most physic examination by repeating t a later date (sometimes dou





by administering more agent the evening of the first unsuccessful examination (again sometimes doubling dose), and repeating the study the next day. "The advantage of the calcium ipodate method is that the examination can be completed in five additional hours with a limited dose of contrast agent."[7]

**a valuable medium for peroral cholegraphy[5]** — Rapidly absorbed from the gastrointestinal tract, calcium ipodate has been reported by some investigators to be diagnostically superior to other oral cholangiographic contrast agents. With careful timing of the examination and the use of tomograms or laminograms, the frequency of good results can approximate that obtained with intravenously administered agents. According to Lewitan and Garcia,[5] the medium's relative safety makes it a valuable medium for peroral cholegraphy. Timesaving and economical Oragrafin Calcium Granules may be particularly useful in certain patients for whom I.V. radiography presents potential hazards, such as elderly patients, those with cardiovascular disease, or patients who may exhibit sensitivity to the test dose of an intravenous agent.

Unique among oral media, Oragrafin Calcium Granules permits same-day films of the gallbladder and ductal system.

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...32. **2.** White, W. W., and
...mer. J. Roentgen. *87*:745
...uhl, J. H., *et al.*: Radiology
...3. **4.** Glenn, J. C., Jr., and
Southern Med. J. *56*:167 (Feb.) 1963. **5.** Lewitan, A., and Garcia, J. F.: Amer. J. Dig. Dis. *10*:219 (March) 1965. **6.** McCrory, E.: J. Tenn. Med. Ass. *58*:258 (Aug.) 1965. **7.** Crummy, A. B.: Wisconsin Med. J. *65*:84 (Feb.) 1966.

**Contraindications:** Contraindicated for persons sensitive to oral iodine compounds or for patients with combined renal and hepatic disease or severe kidney impairment. Gastrointestinal disorders, which may interfere with absorption, or liver dysfunction, which may result in inadequate biliary secretion of medium, are likely to result in unsatisfactory visualization.

**Precautions and Side Effects:** Mild and transient nausea, vomiting, or diarrhea sometimes occur; but the incidence can be reduced by using the calcium granules and restricting the dosage to 3 Gm. Transient headache, dysuria, or abdominal pains may occur.

Hypersensitivity reactions may include urticaria, serum sickness-like reactions (fever, rash, arthralgia), other skin reactions, and rarely anaphylactoid shock. They are more likely to occur in the individual with a history of allergy, asthma, hay fever, or urticaria and in the individual who is known to be hypersensitive to iodine compounds. Antihistamines and corticosteroids are used to control hypersensitivity reactions; but the occasional serious anaphylactoid reactions require the immediate use of epinephrine or phenylephrine, oxygen, and intravenous corticosteroids.

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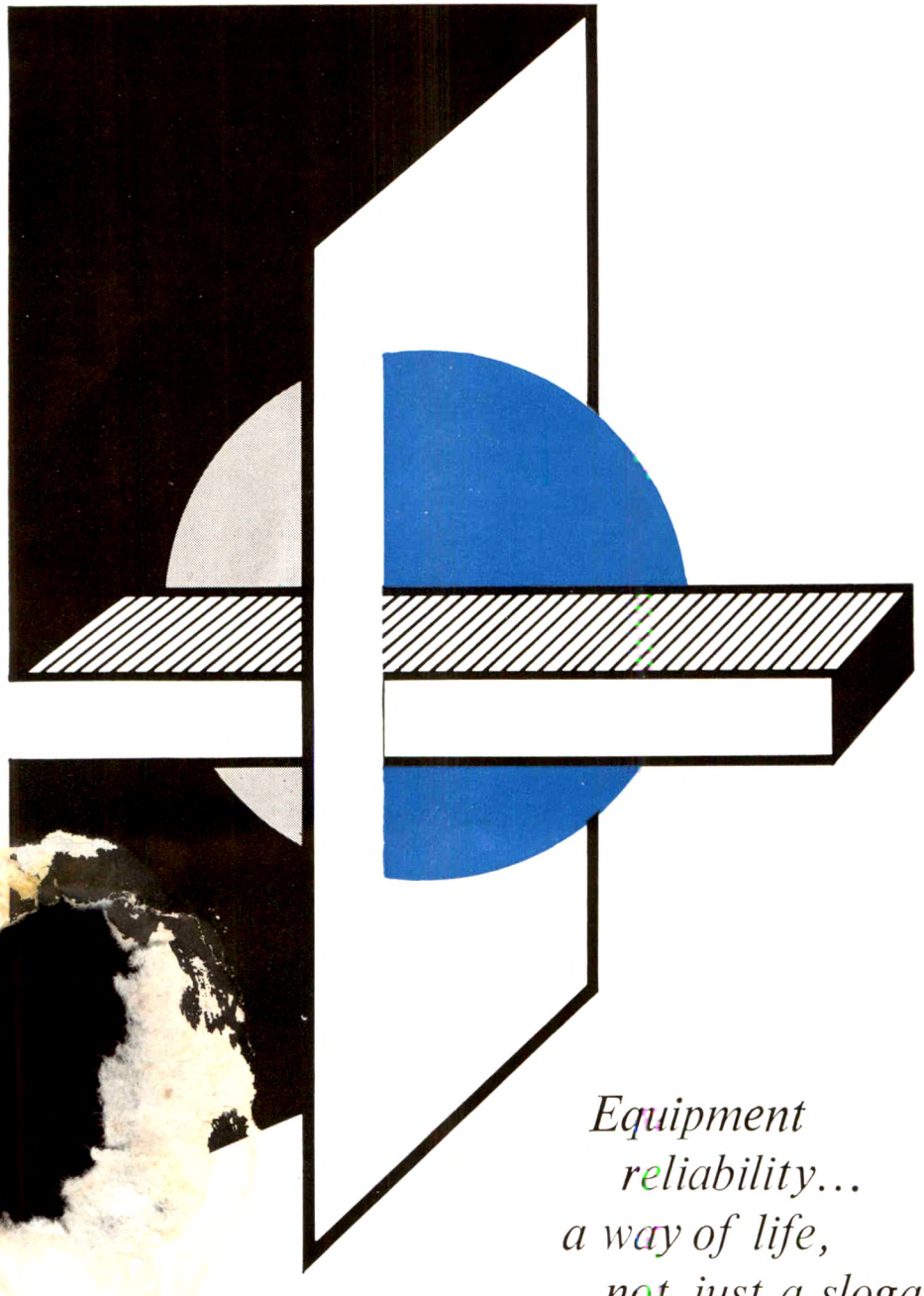
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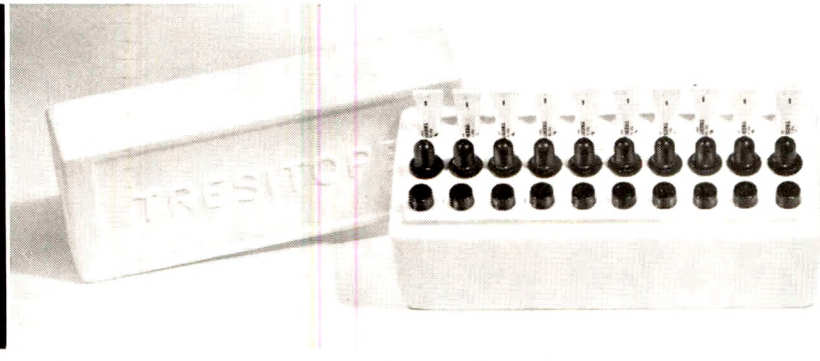
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**$I^{125}$ versus $I^{131}$**

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**NOTE:** While the resin uptake test is a very useful aid in the evaluation of thyroid function, it should not be used as the sole basis for such an evaluation. In any patient, the clinical state is probably the best indication of thyroid status, and *any* laboratory test must be interpreted with caution when test results do not agree with clinical evidence.

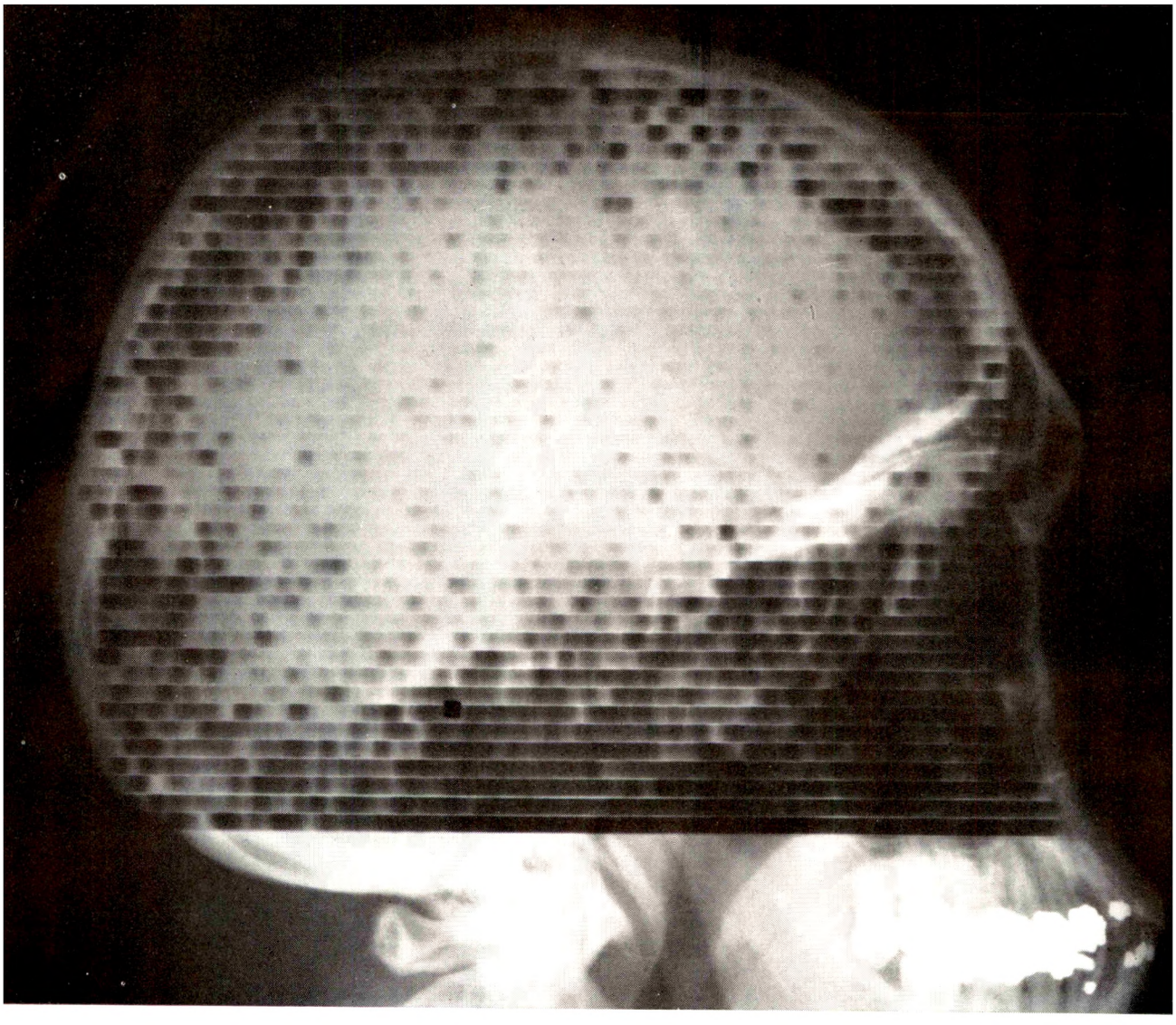
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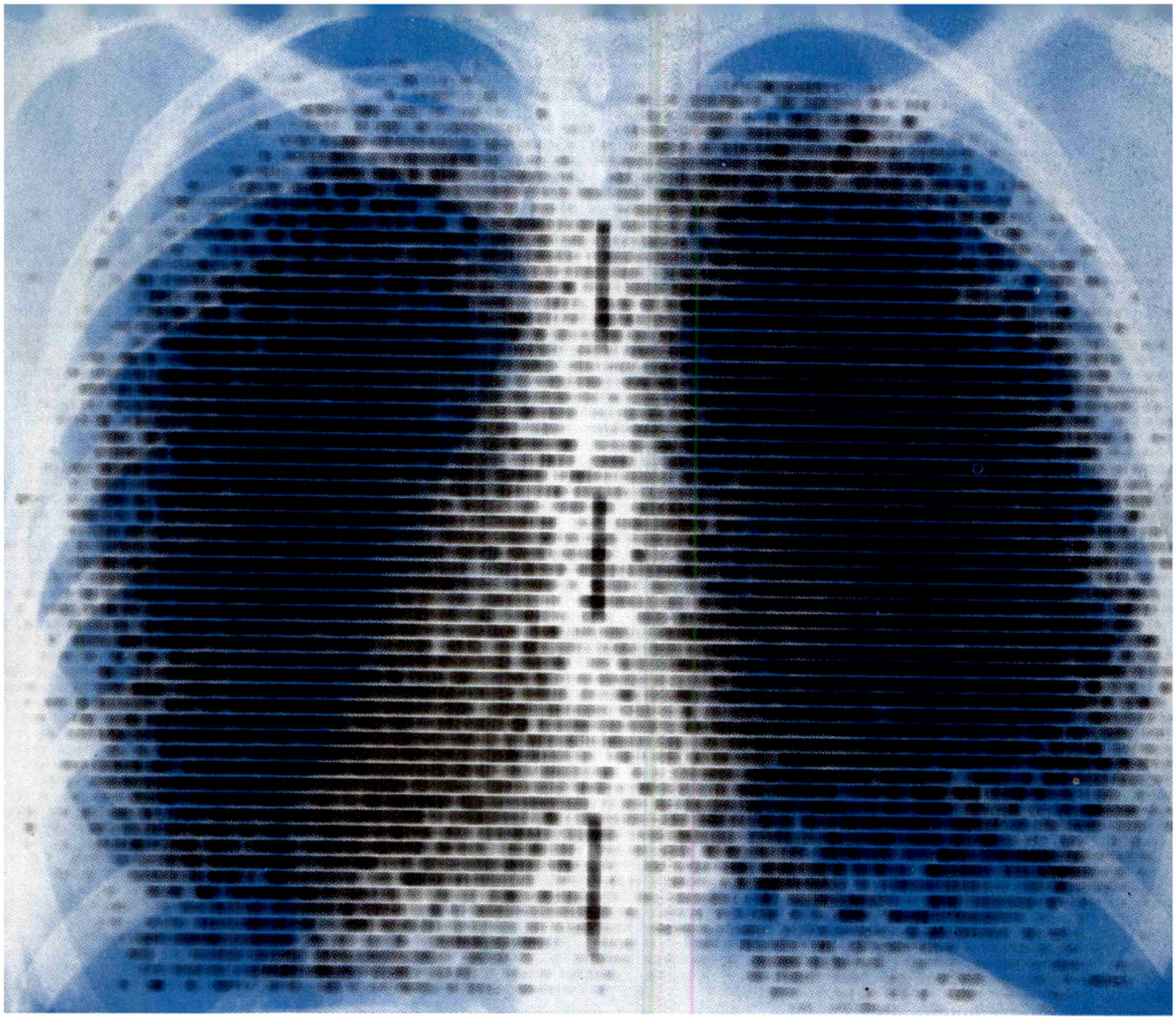
SHIPMENTS: Monday through Friday—and Sunday . . . allows scheduling of brain scans 6 days a week—Monday through Saturday.

INDICATIONS: Adjunctive diagnostic aid in detecting and localizing intracranial neoplastic (primary or metastatic) and non-neoplastic lesions.

CONTRAINDICATION: Radio-pharmaceutical agents should not be administered to pregnant women or to persons less than 18 years old unless the indications are very exceptional.

PRECAUTIONS: Care should be taken to ensure minimum radiation exposure to the patient as well as all personnel; to prevent extracranial contamination because this can lead to erroneous interpretation; and to differentiate areas of abnormal activity from areas of normal vascular activity. 711391

TM-TRADEMARK



# Abbott announces

# Macroscan™-131

## AGGREGATED RADIO-IODINATED ($I^{131}$) ALBUMIN (HUMAN)

## If it's a pulmonary problem, Macroscan-131 pictures it!

**Pulmonary embolism, suspected:** To confirm (or rule out) its occurrence.

**Chronic pulmonary tuberculosis:** To estimate unilateral and regional function and perfusion of the lungs.

**Emphysema:** To evaluate the degree of focal lack of perfusion.

**Pneumonitis:** To evaluate the decreased regional blood flow that occurs without obstruction of vessels.

**Lung tumors:** To evaluate the regional ischemia resulting from compression or obstruction of pulmonary arteries.

**Surgery and/or other therapy for lung disorders:** To evaluate the effectiveness of therapeutic measures.

Macroscan-131 is sterile and non-pyrogenic. It is ready to use and should not be heated prior to use.

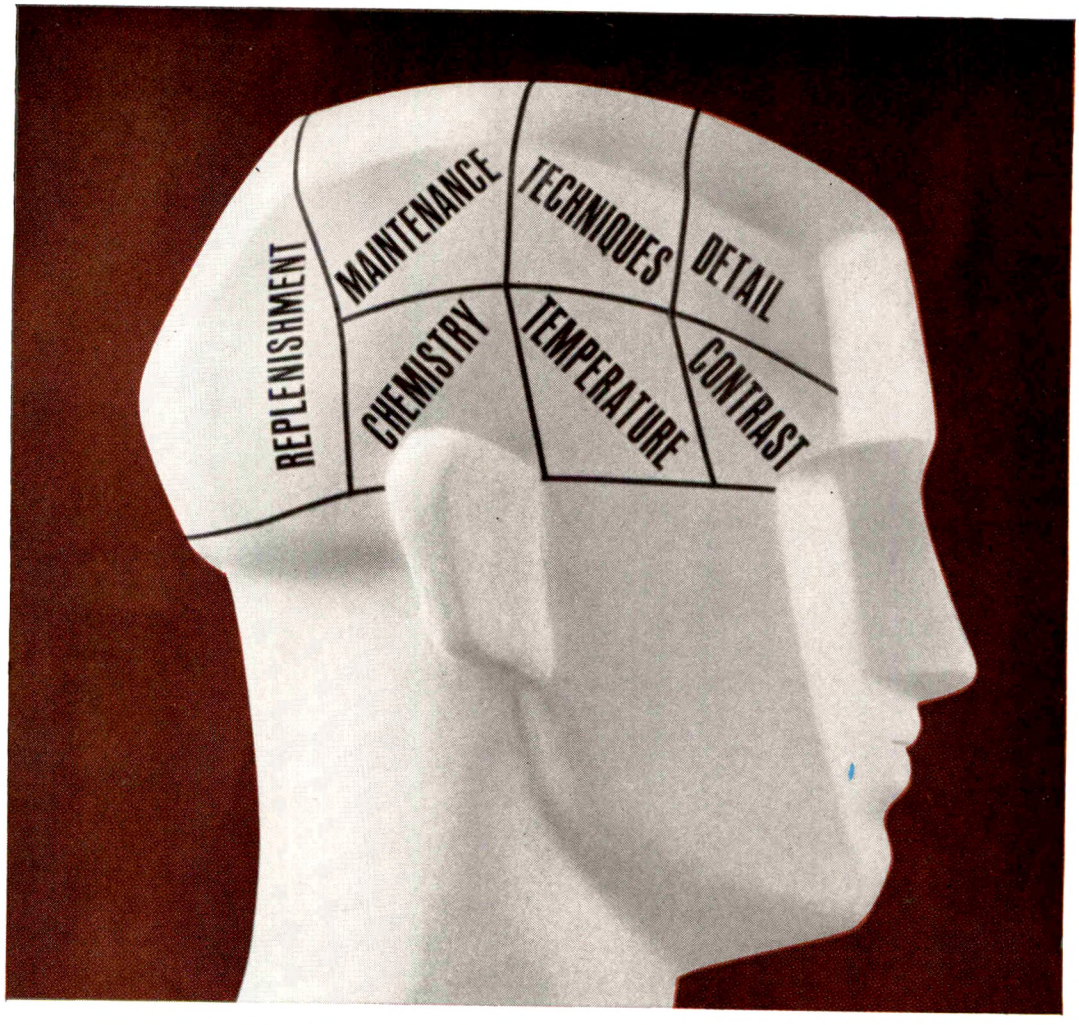
**INDICATIONS:** For scintillation scanning of the lungs to evaluate total, unilateral, and regional arterial perfusion to the lungs.

**CONTRAINDICATION:** Radio-pharmaceutical agents should not be administered to pregnant women, nursing mothers, or to persons less than 18 years old unless the indications are very exceptional.

**PRECAUTIONS, SIDE EFFECTS:** Care should be taken to administer the minimum dose consistent with safety and validity of data. The possibility of an immunological response to albumin should be kept in mind when serial scans are performed. There is a theoretical hazard in acute cor pulmonale, because of the temporary small additional mechanical impediment to pulmonary blood flow. A possible case of urticara has been related to a similar preparation. The thyroid gland should be protected by prophylactic administration of concentrated iodide solution.

TM–TRADEMARK

711400




## Pyne's expert technical representatives stand behind every delivery of New FUJI KX

Even though New Fuji KX provides consistently superior quality (compatible with half-cycle systems) in every film, complications in processing do occur regardless of the brand used. Pyne X-Ray is well aware of this and provides expert technical advice as part of its continuing service policy. Whether your problem is mechanical or chemical,




Pyne's tech reps are ready anytime, anywhere to give expert assistance.
You sacrifice neither quality nor service when you switch to Fuji KX polyester or acetate base film, because Fuji KX is equal in all ways to any quality x-ray film on the market.

PYNE X-RAY CORPORATION

FUJI FILM

U.S. Distributors of FUJI KX Film
Offices in New York, Chicago, and Los Angeles
Warehoused throughout the Country

# *Now...* Sterile, Pyrogen-Free $^{99m}$Tc in your own laboratory

## ASSAYED, READY-FOR-INJECTION from the

# TechneKow®-CS

## COMPLETE SYSTEM

The new Mallinckrodt Nuclear **TechneKow-CS** System includes *complete* laboratory equipment for daily production and immediate assay of injectable sodium pertechnetate Tc 99m, to meet the exacting specifications of the USAEC and agreement states. The exclusive MOLYTECH™ Assay Kit provides an easy, rapid method for daily $^{99m}$Tc and $^{99}$Mo assay of the milked solution. Call or write today for complete information.



New sterile, pyrogen-free TechneKow-CS Generator; also supplied in dual purpose shipping shield

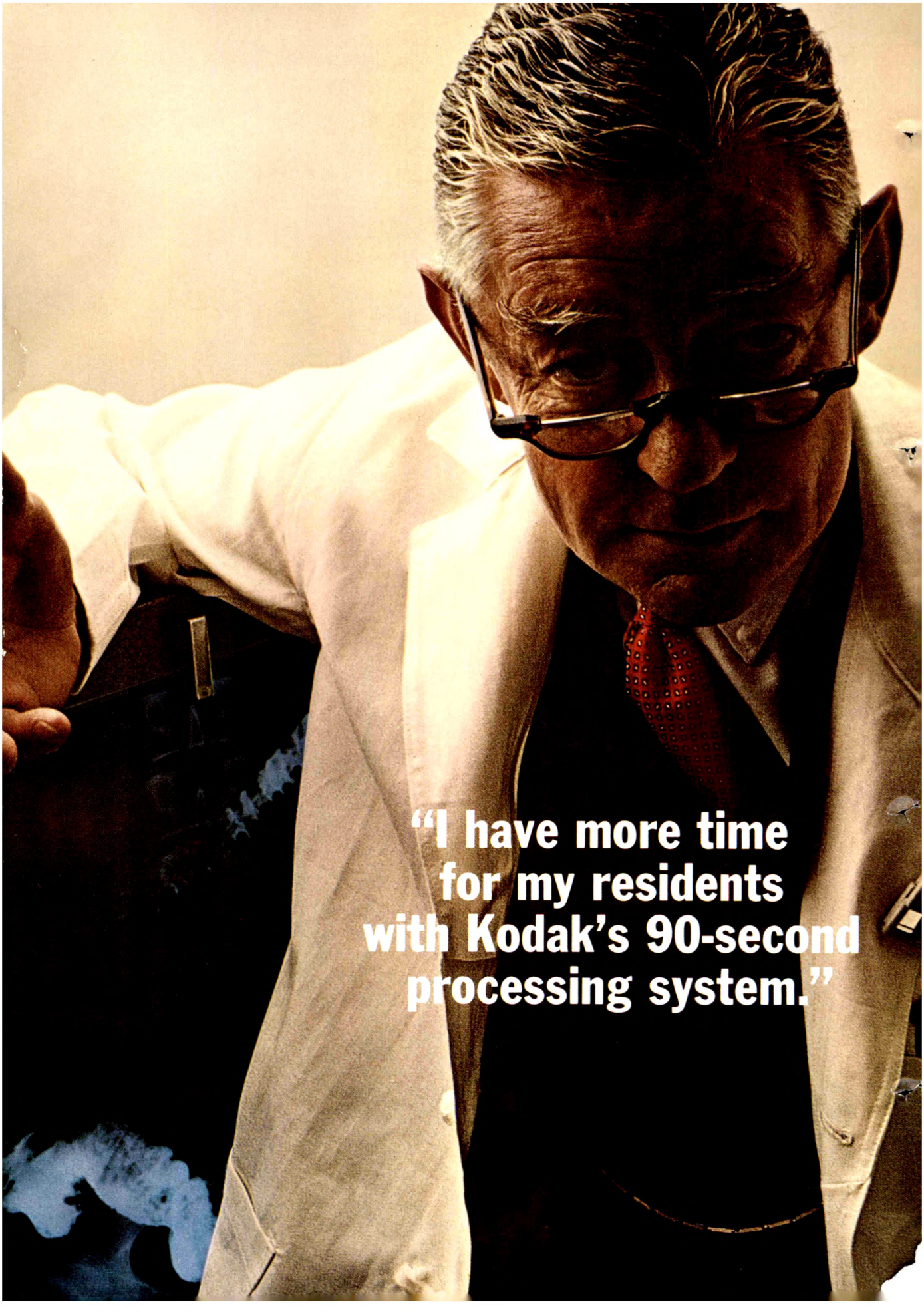
New MOLYTECH™ Calibrator Assay Kit for rapid assay of 99mTc and 99Mo

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"I have more time
for my residents
with Kodak's 90-second
processing system."





This is what converting to the KODAK RP X-OMAT® System meant to the chief radiologist in a large teaching hospital. "There is less confusion, faster service, smoother patient flow, and greater capacity," he told us. "Staff and facilities can be employed more efficiently. . . patients are now being scheduled with little or no waiting time."

Two new 90-second rapid-processing films yield clean, brilliant radiographs of high diagnostic readability time after time. Your Kodak Technical Sales Representative will be happy to demonstrate the distinct characteristics of each. Ask him or your KODAK X-OMAT dealer about the new KODAK RP X-OMAT Processor, Model M6—the first processor designed exclusively for 90-second operation. They'll be able to describe the efficiencies of Rapid Processing to you.

**EASTMAN KODAK COMPANY**
**Radiography Markets Division**
**Rochester, N.Y.**

Kodak
TRADEMARK

# New, low-priced, portable TLD System designed for clinical radiation therapy

EG&G's new solid-state TL-6, Thermoluminescent Dosimeter Reading System uses small encapsulated LiF dosimeters for accurate radiation measurements over wide dose and energy ranges. The operator has only to insert an exposed dosimeter and holder into the reader and press one button. Within seven seconds, the radiation dose to the patient has been determined accurately and reliably. Since the reader is a peak readout instrument the dosimeters do not require annealing for doses in the therapy range.

The TL-6 not only provides the needed performance, plus portability . . . but is priced to put it within the reach of every hospital and clinic, (under $3,000)

Just send the coupon below, if you'd like more complete details or a demonstration.

SPECIFICATIONS

Dose Range: ..... 0.1 R-10,000 R
Data Presentation: ..... Digital
Standardization: .... $C^{14}$ Reference Light
Automatic Ranging: ..... 4 Decades
Outputs: ..... Analog & Digital
Power Requirements: ..... 115 Vac, 50-60 cps, 1.0 A
Size: ..... 5" x 10" x 16"
Weight: ..... 8 lbs.

EG&G



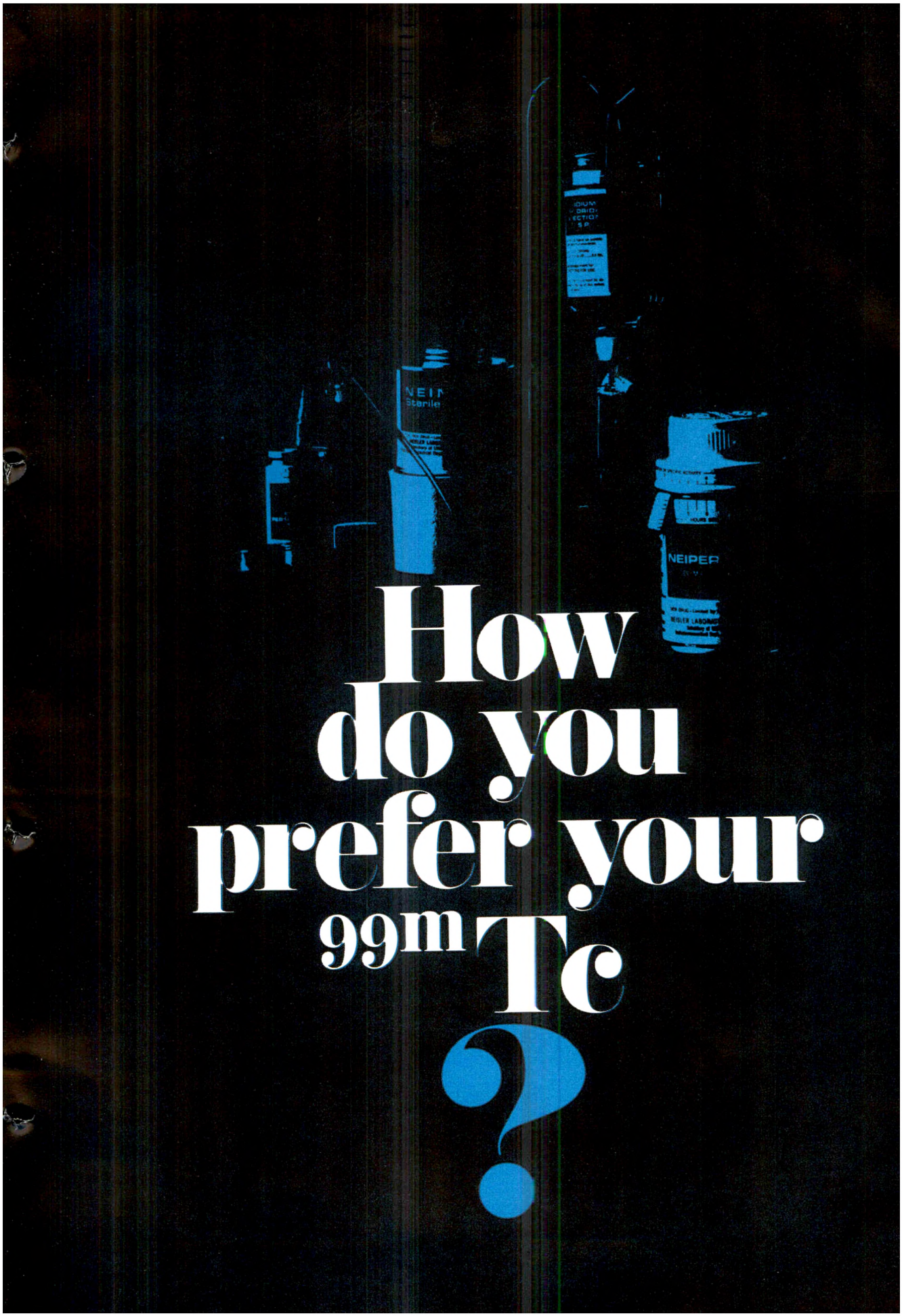

**EG&G, Inc.,** Santa Barbara Division, P.O. Box 98, Goleta, Calif. 93017

☐ Send additional information on your low-cost TL-6 system.
☐ Please have representative call to arrange a demonstration.

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TITLE _____ CITY _____

INSTITUTION _____ STATE _____ ZIP _____




# How do you prefer your $^{99m}Tc$ ?

# now from Neisler...

## STERILE, PYROGEN-FREE SODIUM PERTECHNETATE Tc 99m AS YOU NEED IT, ALL WEEK LONG

# NEIMOTEC® Sterile Generator

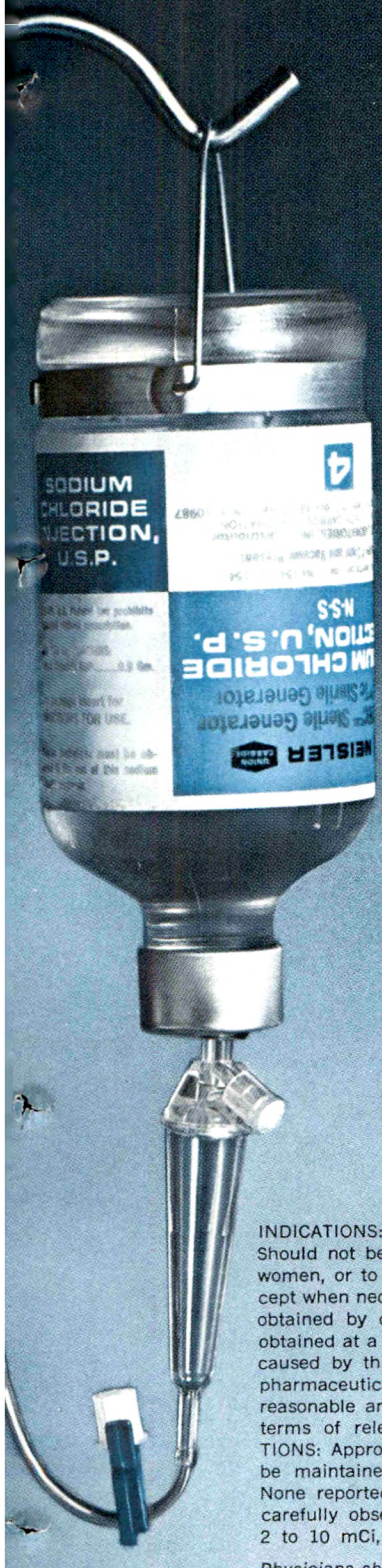
$^{99}Mo/^{99m}Tc$ Sterile Generator

**FAST... EASY... ECONOMICAL**

- simple vacuum elution system for maximum dependability
- entire system sterile...one-time entry to easily accessible septa
- high-yield...high chemical purity
- multiple daily elutions possible

SUPPLIED: 100, 200 or 300 mCi at noon, New York time, on Mondays following shipment; in nonreturnable lead container, with complete eluting accessories.






# PRECALIBRATED READY-TO-USE

## NEIPERTEC™ 99m

sodium pertechnetate Tc 99m

SUPPLIED: In lead-shielded vials in convenient COMPUTERCAP™ packaging; 10 or 15 mCi at the time of calibration.




INDICATIONS: Brain scanning. CONTRAINDICATIONS: Should not be administered to pregnant or lactating women, or to patients under the age of 18 years, except when necessary diagnostic information cannot be obtained by other types of studies or can only be obtained at a risk greater than the radiation exposure caused by this drug. WARNINGS: As with all radiopharmaceuticals, dose should be limited to smallest reasonable amount consistent with greatest value in terms of relevant diagnostic information. PRECAUTIONS: Approved radiation safety precautions should be maintained at all times. ADVERSE REACTIONS: None reported to date; however, patients should be carefully observed. DOSAGE AND ADMINISTRATION: 2 to 10 mCi, administered by intravenous injection.

*Physicians should consult product package insert before administering.*

**NEISLER LABORATORIES, INC.**
Subsidiary of
UNION CARBIDE CORPORATION
Radiopharmaceutical Dept.
P.O. Box 433, Tuxedo, New York 10987

# What they're saying about the



**"Gives us better contrast at higher kv."**

**"Visibility of detail is better."**

These are the comments that speak for the Diagnostic Clarity of CRONEX 4 X-ray Film. They came from radiologists around the country who have tried our new film at 90 seconds, at 3½ minutes and at 7 minutes.

CRONEX 4 is perfectly matched to Du Pont's high-energy Multi-Process chemicals, which function at temperatures in the 90° F. to 96° F. range. These lower temperatures mean simplified conversion of existing equipment to a fast-access system and less wear and tear on processors.

If you're considering converting one or more of your processors to fast

# Diagnostic Clarity of new CRONEX*4:



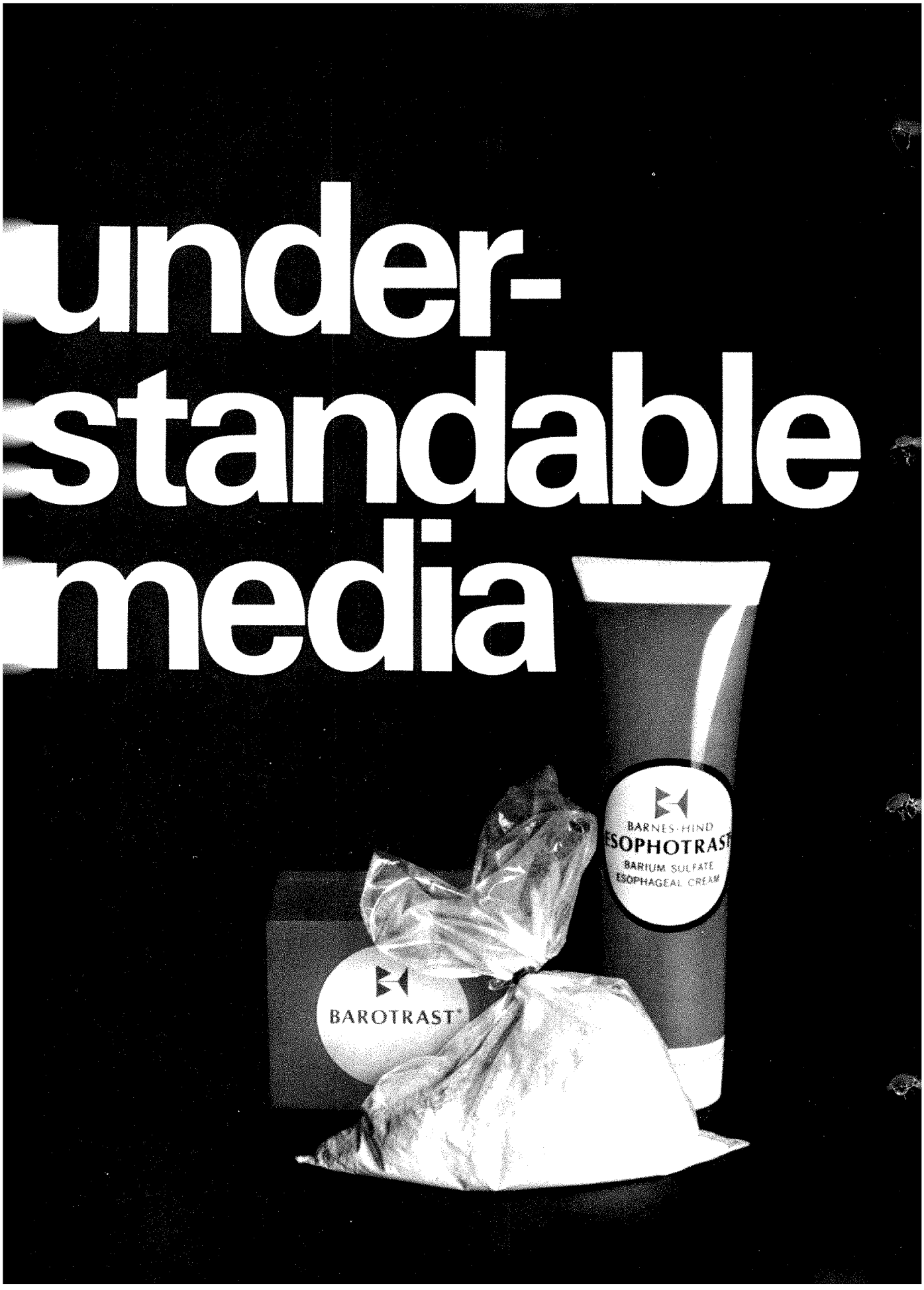
**"Stays diagnostic under varying exposure conditions."**

**"Image sharpness is improved."**

access, call on your Du Pont Technical Representative for a fuller explanation of the CRONEX 4 system.

*Reg. U. S. Pat. Off. for Du Pont's medical X-ray film.

DU PONT CRONEX X-ray Products


under-
standable
media
BARNES-HIND
ESOPHOTRAST
BARIUM SULFATE
ESOPHAGEAL CREAM
BAROTRAST

If the barium preparations you've used up to now have all seemed pretty much alike, then it's time to compare the contrasts with ESOPHOTRAST® (the ready-to-use esophageal cream) and BAROTRAST® (a micronized form of barium sulfate).

One of the prominent problems with most contrast media is no small matter. Their large particles have a tendency to precipitate and to flocculate out of suspension with the slightest change in pH, mucin concentration, or enzyme activity. Not so with ESOPHOTRAST and BAROTRAST, both of which are formulated to ensure an easy suspension that remains in suspension even during routine storage.

Then too, a common feature of many other barium sulfates suggests a sticky problem. The varying size of the particles reduces their adhesive properties since not enough particles come into contact with the esophageal and/or intestinal lining to provide complete coverage. With micronized ESOPHOTRAST and BAROTRAST, a maximum number of particles are available to produce a tenacious elastic coating that lasts for the time it takes to "get the picture."

ESOPHOTRAST, because of its specially formulated viscosity, provides a consistent, prolonged, residual coat that permits an excellent outline of the esophagus as well as an evaluation of cardiac size. Since it is premixed, ESOPHOTRAST is ready for instant use, which means longer visualization time and shorter preparation time. BAROTRAST, because of its natural viscosity, flows in a steady consistent column at a speed that can cut the upper G.I. series and small bowel examination time almost in half.

Both formulations are creamy, nongritty and pleasantly flavored to help your patient help you make the examination that much easier.

Considering the barium sulfates in the light of this inside information, it would seem that there is a marked contrast. And all it takes is a little understanding to get the clear picture.

# Esophotrast® · Barotrast®

**BARNES-HIND BARIUM PRODUCTS**

Subsidiary of Barnes-Hind Pharmaceuticals, Inc.
Sunnyvale, Calif. 94086

# 'or a clear picture
# 'rep" the colon
# 'ith pleasant-tasting
# -PREP Liquid

ndardized extract of senna fruit)

cifically designed for preradiographic bowel evacua-
. X-PREP Liquid permits excellent visualization in
. and urologic roentgenography.

significant gas shadows. No residual oil droplets.

PREP Liquid reduces or eliminates the need for enemas
or to radiography.

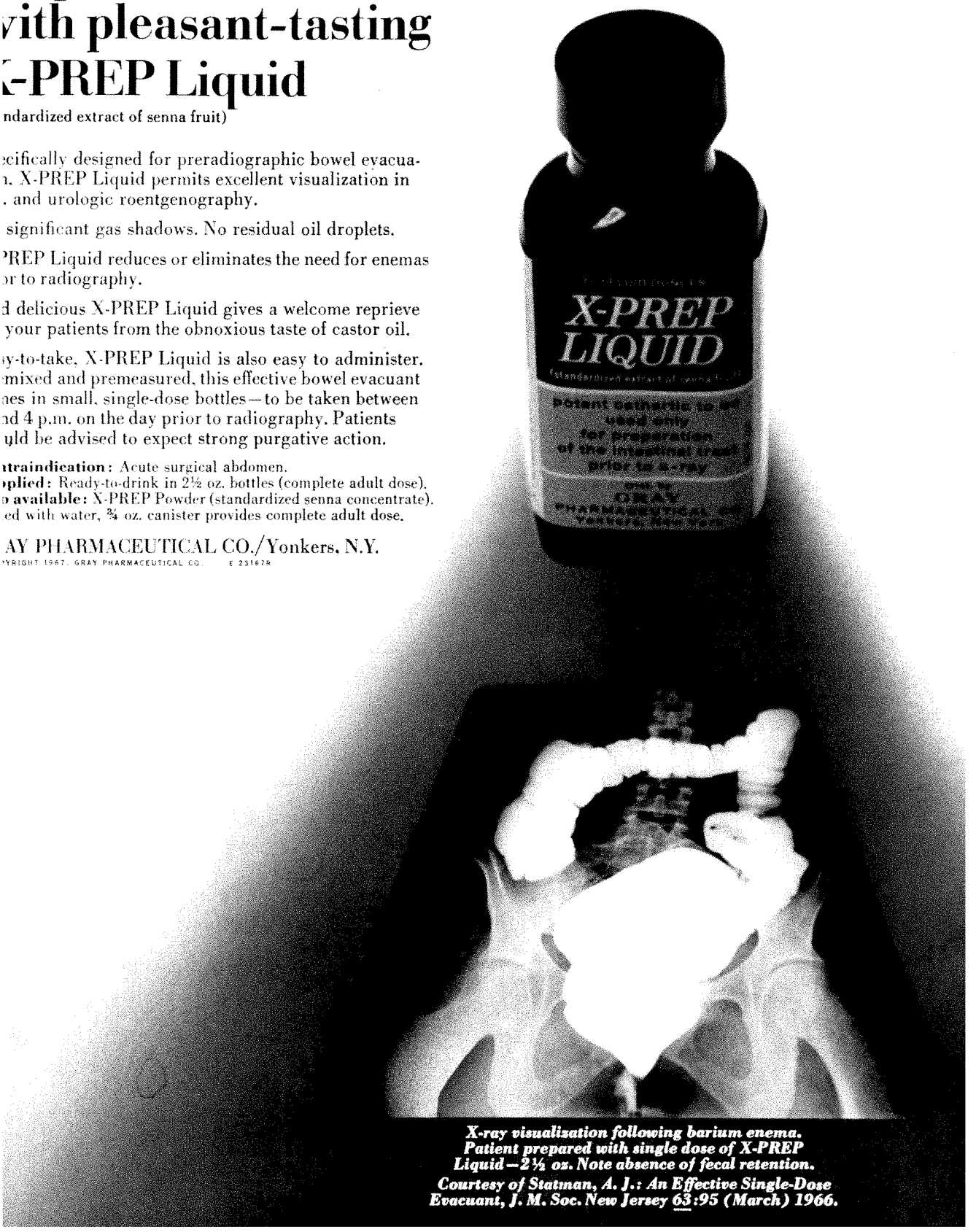
d delicious X-PREP Liquid gives a welcome reprieve
your patients from the obnoxious taste of castor oil.

y-to-take, X-PREP Liquid is also easy to administer.
mixed and premeasured, this effective bowel evacuant
nes in small, single-dose bottles—to be taken between
nd 4 p.m. on the day prior to radiography. Patients
uld be advised to expect strong purgative action.

**ntraindication:** Acute surgical abdomen.
**plied:** Ready-to-drink in 2½ oz. bottles (complete adult dose).
**o available:** X-PREP Powder (standardized senna concentrate).
ed with water, ¾ oz. canister provides complete adult dose.

AY PHARMACEUTICAL CO./Yonkers, N.Y.

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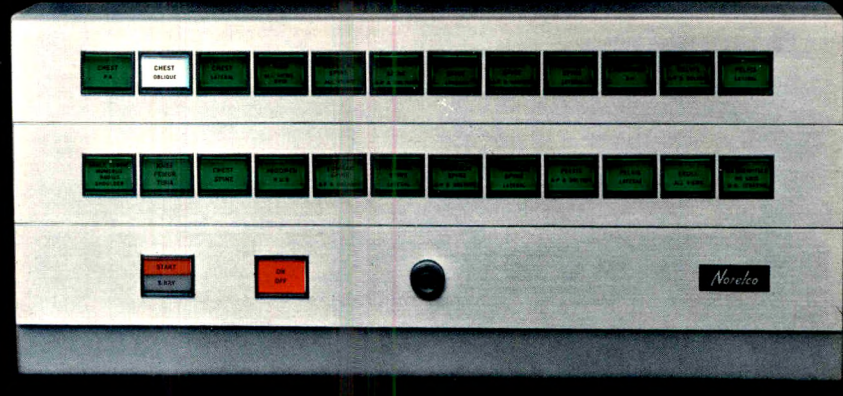



***X-ray visualization following barium enema. Patient prepared with single dose of X-PREP Liquid—2½ oz. Note absence of fecal retention.***

***Courtesy of Statman, A. J.: An Effective Single-Dose Evacuant, J. M. Soc. New Jersey 63:95 (March) 1966.***

## THE NORELCO PROGRAMMED SYSTEM:

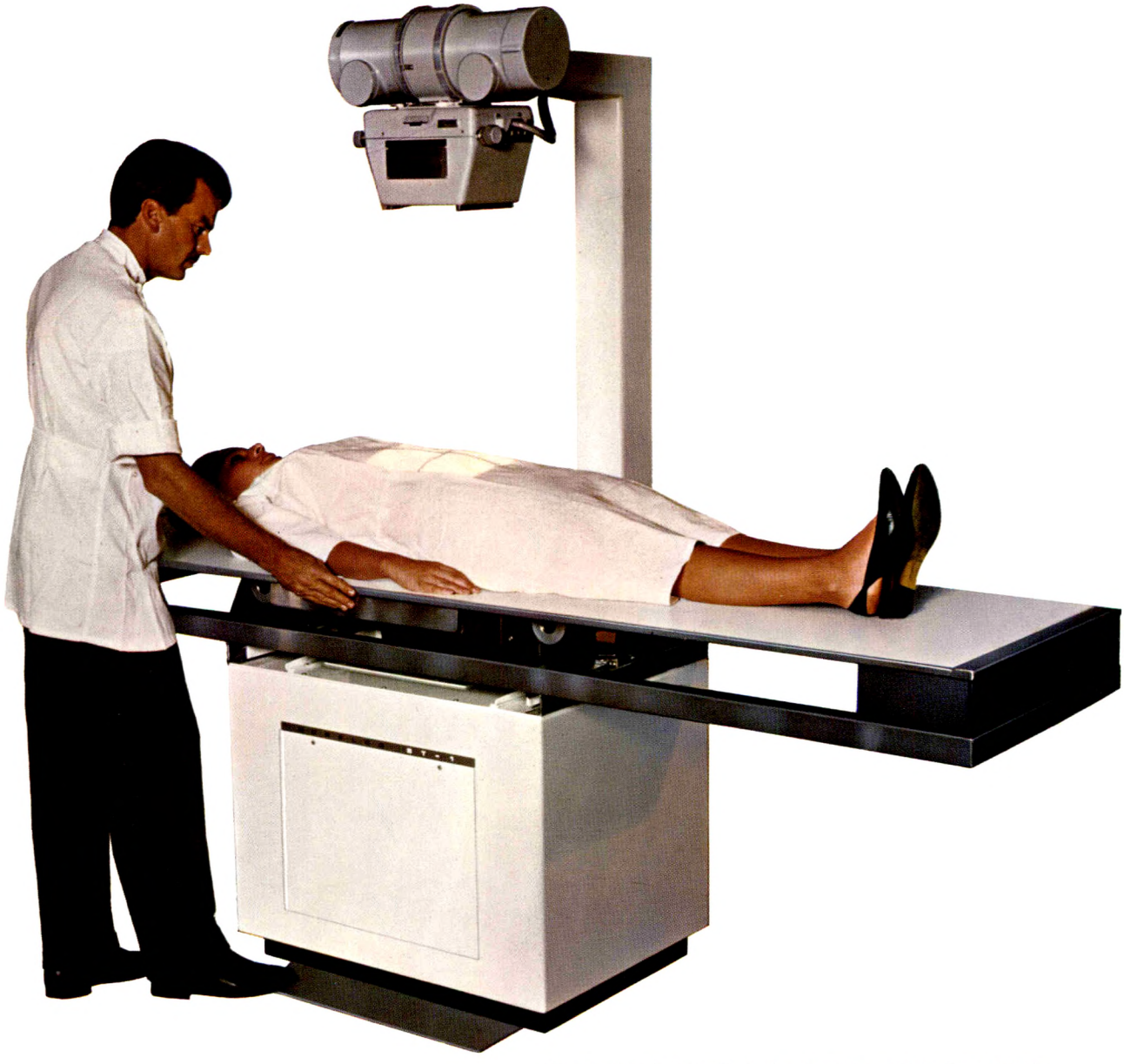
# MORE EFFICIENT USE OF FACILITIES, PERSONNEL and TIME




This automated laboratory for routine radiographic procedures eliminates factor selection, technique charts, tube centering, collimator adjustment and FFD adjustment. It enables technologists to concentrate on patient care and positioning, while producing consistently excellent radiographs – in the shortest possible times.

Under your guidance, exposure data are programmed and set into the Norelco System Control – in place of printed exposure charts. Once the control and installation are set, The System does everything except choose the examination, position the patient, and initiate the exposure.

# ALL YOU DO IS
# CHOOSE THE EXAMINATION, POSITION THE PATIENT, AND INITIATE THE EXPOSURE.



For further information please write or telephone North American Philips Company, Inc., 100 East 42nd Street, New York, N.Y. 10017 (212-697-3600), contact your Norelco Branch Office or your local Norelco Medical Equipment Dealer.

M-47

Norelco® MEDICAL EQUIPMENT

NORTH AMERICAN PHILIPS COMPANY, INC.
Professional Products Division, 100 East 42nd St., New York, N.Y. 10017

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Radiological Society of
North America
Palmer House, Chicago
November 26th to 30th
Booths Nos. 1–5

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**We would like to tell you more — we would like to give you facts, figures and specifications. We have representatives all over the world. Write for full information.**

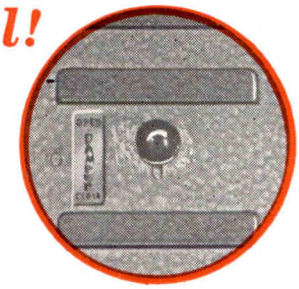
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66:2M

# What's so special about Halsey Film Dryers?... the climate compensator control!





Model No. 600-D



Model No. 601-D

The biggest bugaboo of film dryers is humidity in the darkroom . . . environmental moisture which turns the most efficient unit into a sluggish, slow-acting flop.

Now HALSEY introduces the *climate compensator* — a system of air control that lets you shut out outside humidity when it's too high . . . or bring in outside air when it's cool and dry.

The result . . . faster film drying — regardless of climate conditions!

**No. 600-D.** Fatigue-free loading from the top. Handles 12 films every 20 minutes. Complete with automatic timer — a guard against fire hazard due to failure to turn off the heater. Dimensions: 23″ wide, 33″ high, 17½″ deep.

**No. 601-D.** Economy version of above — loads from the front. Recommended for stacking on top of No. 600-D for stoop-free loading, when extra capacity is desired. Dimensions: 23″ wide, 33″ high, 17½″ deep. Automatic Timer (No. 602-DT) available.

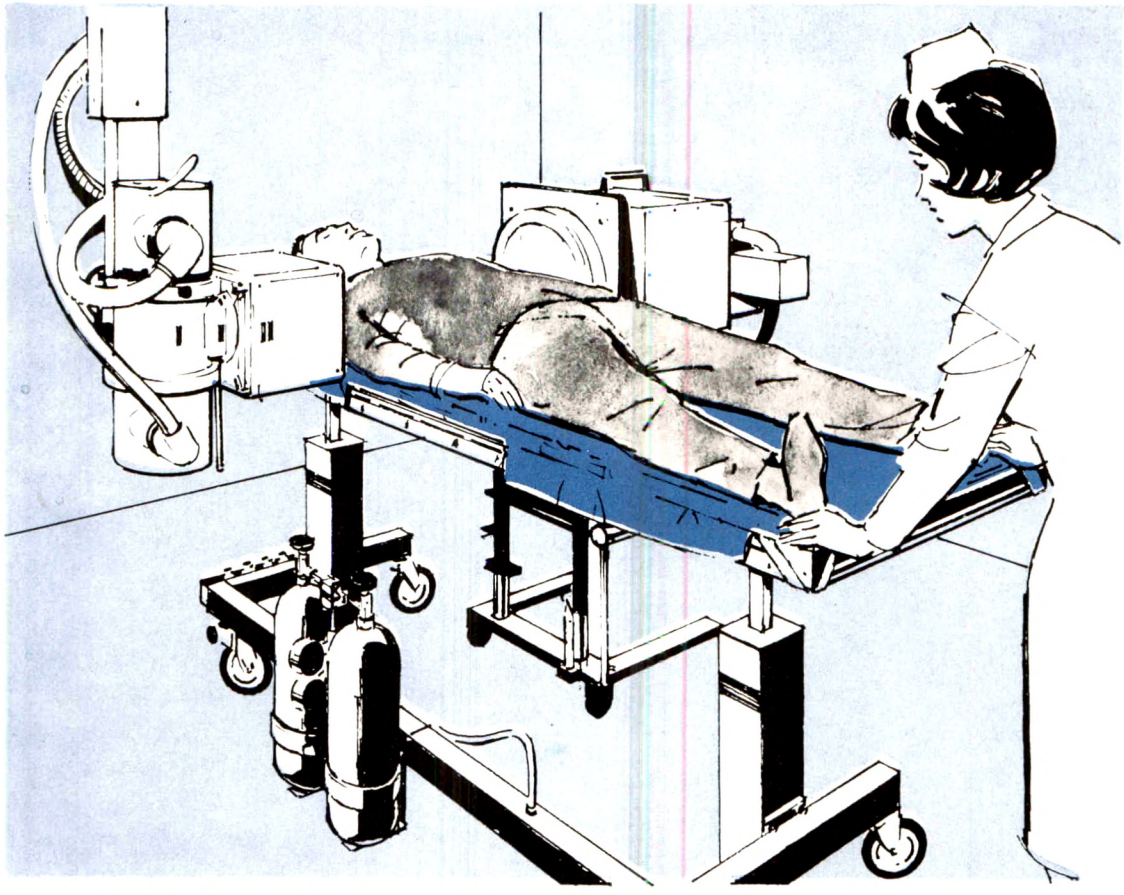
**No. 601-D-H.** For restricted space — similar to No. 601-D, but almost 10″ narrower; accommodates 6 films. Dimensions: 13½″ wide, 33″ high, 17½″ deep.

**Write for detailed literature**

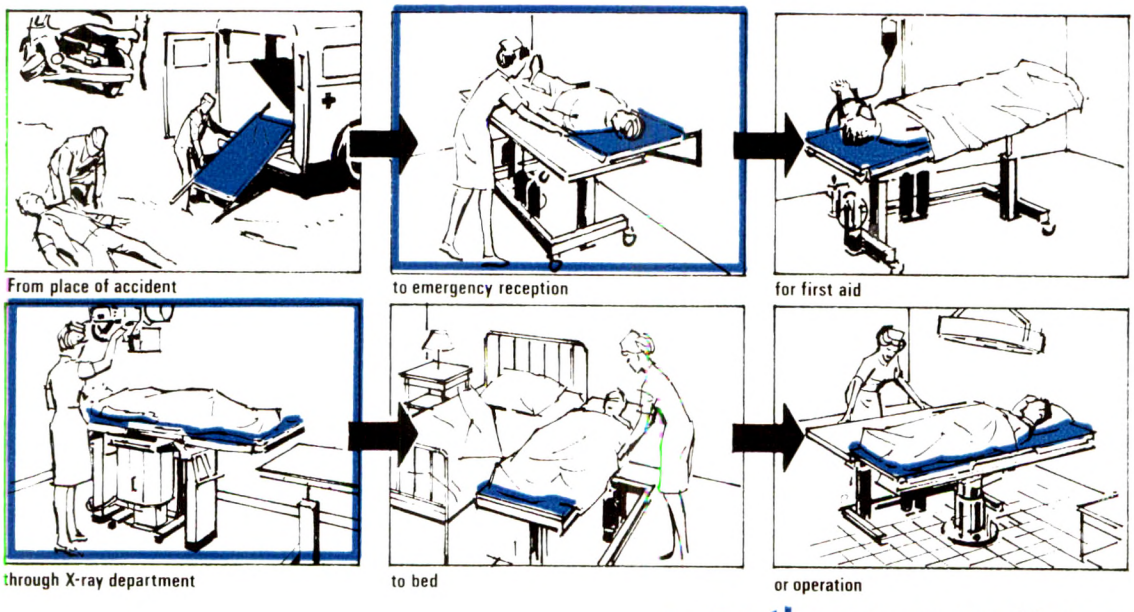
HALSEY

HALSEY X-RAY PRODUCTS, INC.
1425-37th STREET, BROOKLYN, N.Y. 11218

CHICAGO: 7321 Monticello Avenue, Skokie, Illinois 60076
LOS ANGELES: 1816 W. 8th St., Los Angeles, Calif. 90057



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Chicago Shop Facilities are now available to have your used Cut Film Changers completely reconditioned by Factory-Trained Engineers. This well-established service program includes:

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2. Complete refinishing of housing.
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Arrangements *must* be made with your local X-Ray dealer to take advantage of:

- Special shipping containers for your units.
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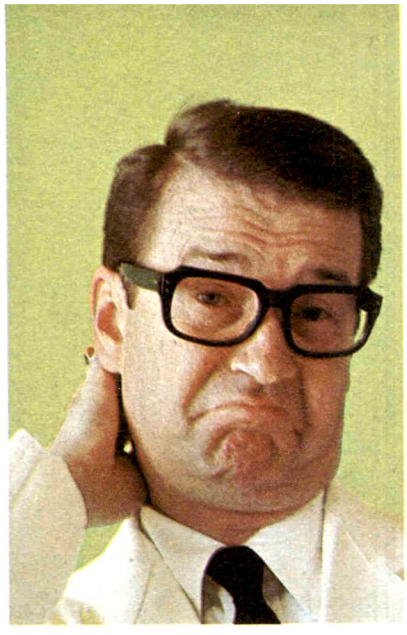
SCHICK X-RAY

***SCHICK X-RAY CO., INC.***

415 Green Bay Road • Wilmette, Ill. 60091

Phone (312) 256-4700

Visit Booths Number 62 and 63 at the Radiology Meeting



**"Oh I suppose Litton's Profexray Division might sell me a new film processo if they came up with one that handles both 90-second and 3½-minute cycles and doesn't need daily cleaning and is twice as rugged and has guaranteed service. And if they gave it to me for half price."**

Okay.

We'll do it: A new automatic film processor that's interchangeable between 90-second and 3½-minute cycles (no extra charge). And that costs about half what you'd expect to pay for a 90-second processor.

Considering the price, you might think we'd have to cut a few corners on a processor like that.

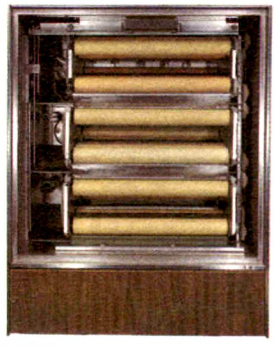
Not at all.



The new Profexray automatic processor is *more* rugged than any of the others. Notice the gear train on the unit above. The gears are almost three times bigger. Construction is simpler, heavier, and far more trouble-free.

We've been field testing this unit in hospitals and laboratories throughout the country for the last two years. Under the toughest possible conditions, here's what users have found:

Service is fast and easy, because everything that might need adjustment is totally accessible from the top (see picture below).



Whether the unit is cleaned daily or not makes no noticeable difference in its operation (chemical replenishment is a positive, cycled stream—there are no drip-drip-drip mechanisms to clog).

The Profexray automatic processor uses about a third as much water as other units, with substantial savings in electric heating costs. It's earned —already—a remarkable record of continuous, trouble-free operation.

Convinced?

We don't expect you to be. Maybe you'd like to see how this revolutionary processor performs in your lab. So try it. On an unconditional money-back guarantee for 60 days. You'll find it's everything we say.

The Profexray Division of Litton Industries guarantees it. Your local Profexray serviceman guarantees it. And you're getting it for half price.

That's worth something, isn't it?

As a start, let us give you more information. Phone your local Profexray representative. Or write for a free brochure on the new Profexray automatic 90-second film processor. You'll find it works better than any processor you can buy.

Even at twice the price.

PROFEXRAY
DIVISION OF LITTON INDUSTRIES

515 East Touhy Avenue • Des Plaines, Illinois 60018

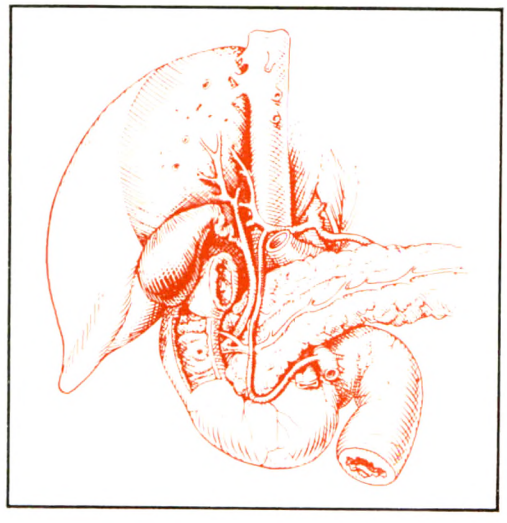


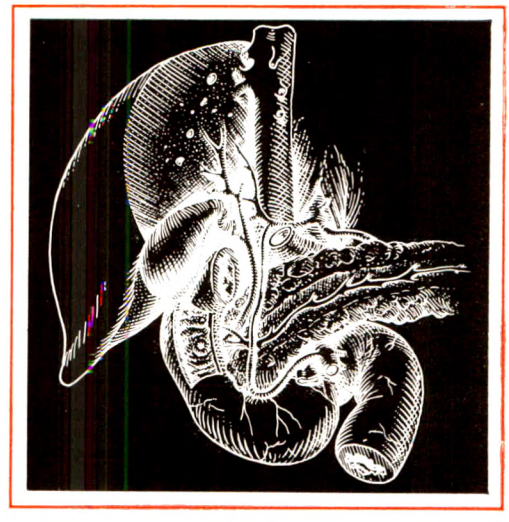
SQUIBB

a research concept in contrast visualization

# for better definition of disease:

## documenting differences between oral contrast agents





no. 2 in series:

# Oragrafin®

Squibb Ipodate

## maximum concentration with better patient toleration in oral cholangiography and cholecystography

For rapid or routine studies of the gallbladder and biliary ducts, Oragrafin provides rapid, more complete absorption, for maximum concentration and excellent contrast.

**definition of maximum concentration**

**high absorption index**—"No one will dispute the statement that the diagnostic reliability of oral cholecystography depends upon the degree of absorption of the contrast medium employed."[1] Radioisotope studies with ipodate sodium show a markedly improved absorption index (approximately 70 per cent) with no increase in the toxic properties.[1]

**less bowel residue**—"Incomplete and variable absorption of a cholecystographic agent is not desirable, for the opacification of the gallbladder is then less dependent on the status of the gallbladder and more a reflection of the percentage absorption."[2] "Opaque material in the bowel was found in 46 per cent of [105 patients in] the... [iopanoic acid] group as compared with 9 per cent of those [99] of the [Oragrafin Sodium Capsules]...group."[2] In another study, 49 of 100 patients receiving iopanoic acid had residue in the gastrointestinal tract, while only 15 of 100 patients receiving Oragrafin had such residue.[3]

**excellent opacification**—"In addition to its ease of administration and safety, its principal advantages are a high yield of diagnostic films...."[4]

**definition of better toleration**

**low incidence of untoward reactions**—In contrast to findings with other cholecystographic agents, Lewitan and Garcia[5] reported in their 246 patient study: "With

ipodate calcium...there was no clinical evidence of immediate or delayed nephrotoxicity in any of the patients given it...."[5] To study possible renal toxicity, "Creatinine clearance tests were also done before and after administration of multiple doses in excess of 6 gm. in 8 cases, and showed no significant alterations."[5] However, "Multiple doses beyond 6 gm. are not recommended."[5] In their study of 120 patients, Glenn and O'Brien reported "...no reactions in this series of patients attributable to the administration of [Oragrafin]...."[4] And, McCrory reported that Oragrafin was "...used routinely in cholecystographic studies in approximately 2000 patients with excellent diagnostic films and only rare and mild reactions."[6]

**side effects compared to iopanoic acid**

side effects encountered in administration of 3 Gm. of Oragrafin Sodium Capsules and 3 Gm. iopanoic acid *

| | Oragrafin Sodium Capsules (Squibb Sodium Ipodate) | iopanoic acid |
|---|---|---|
| no. of cases | 99 | 105 |
| no. of side effects | | |
| nausea | | |
| slight | 10 | 14 |
| severe | 2 | 6 |
| vomiting | 0 | 1 |
| diarrhea | | |
| mild | 7 | 22 |
| severe | 3 | 16 |
| cramps | 5 | 15 |
| dysuria | 9 | 13 |
| total/per cent | 36 (36.3%) | 87 (82.9%) |

*Adapted from White, W. W., and Fischer, H. W.[2]

Juhl,[3] in his study of 200 patients (100 on each agent), found no significant difference between iopanoic acid and sodium ipodate in the incidence of nausea; vomiting occurred in an equal number of cases.

# definition of convenience: Oragrafin® Calcium Granules

## Squibb Calcium Ipodate

**visualization of poorly functioning gallbladder reduces the need for I.V. studies**—In a concentrating gallbladder, Oragrafin Calcium Granules (Squibb Calcium Ipodate) will show storage, concentration, delivery or stones. A patent biliary duct can generally be expected to visualize. Provided the cystic duct is patent, Oragrafin will visualize the normal gallbladder, the abnormal gallbladder containing papillomata, nonopaque stones or radiodense calculi, and gallbladders where concentrating power is diminished.

**patient convenience**—Routine cholecystography night-before procedure is easy for patients to follow; palatable granules further enhance patient acceptability. Rapid clearance of medium permits same-day administration and gallbladder and ductal films, and, if necessary, same-day re-examination; reduces need for I.V. studies.

Optimal concentration in the hepatic and biliary ducts usually occurs within 1 to 3 hours. Although the gallbladder is optimally opacified 10 hours after ingestion of the agent, diagnostically valuable information can often be obtained within 5 hours or less.

**rapid absorption permits same-day re-examination**—To determine the cause of nonopacification after routine cholecystography, most physicians require re-examination by repeating the procedure at a later date (sometimes doubling dose), or





A-2803

by administering more agent the evening of the first unsuccessful examination (again sometimes doubling dose), and repeating the study the next day. "The advantage of the calcium ipodate method is that the examination can be completed in five additional hours with a limited dose of contrast agent."[7]

**a valuable medium for peroral cholegraphy**[5]—Rapidly absorbed from the gastrointestinal tract, calcium ipodate has been reported by some investigators to be diagnostically superior to other oral cholangiographic contrast agents. With careful timing of the examination and the use of tomograms or laminograms, the frequency of good results can approximate that obtained with intravenously administered agents. According to Lewitan and Garcia,[5] the medium's relative safety makes it a valuable medium for peroral cholegraphy. Timesaving and economical Oragrafin Calcium Granules may be particularly useful in certain patients for whom I.V. radiography presents potential hazards, such as elderly patients, those with cardiovascular disease, or patients who may exhibit sensitivity to the test dose of an intravenous agent.

Unique among oral media, Oragrafin Calcium Granules permits same-day films of the gallbladder and ductal system.

dosage schedule for films of gallbladder and ductal system

| | |
|---|---|
| 8 A.M. | 2 packets of granules |
| 9 A.M. | visualization of ducts |
| 10 A.M. | optimal visualization of ducts |
| 1 P.M. | visualization of gallbladder |

**References: 1.** Sanen, F. J.: Amer. J. Roentgen. *88*:797 (Oct.) 1962. **2.** White, W. W., and Fischer, H. W.: Amer. J. Roentgen. *87*:745 (April) 1962. **3.** Juhl, J. H., *et al.*: Radiology *80*:87 (Jan.) 1963. **4.** Glenn, J. C., Jr., and O'Brien, P. S.: Southern Med. J. *56*:167 (Feb.) 1963. **5.** Lewitan, A., and Garcia, J. F.: Amer. J. Dig. Dis. *10*:219 (March) 1965. **6.** McCrory, E.: J. Tenn. Med. Ass. *58*:258 (Aug.) 1965. **7.** Crummy, A. B.: Wisconsin Med. J. *65*:84 (Feb.) 1966.

**Contraindications:** Contraindicated for persons sensitive to oral iodine compounds or for patients with combined renal and hepatic disease or severe kidney impairment. Gastrointestinal disorders, which may interfere with absorption, or liver dysfunction, which may result in inadequate biliary secretion of medium, are likely to result in unsatisfactory visualization.

**Precautions and Side Effects:** Mild and transient nausea, vomiting, or diarrhea sometimes occur; but the incidence can be reduced by using the calcium granules and restricting the dosage to 3 Gm. Transient headache, dysuria, or abdominal pains may occur.

Hypersensitivity reactions may include urticaria, serum sickness-like reactions (fever, rash, arthralgia), other skin reactions, and rarely anaphylactoid shock. They are more likely to occur in the individual with a history of allergy, asthma, hay fever, or urticaria and in the individual who is known to be hypersensitive to iodine compounds. Antihistamines and corticosteroids are used to control hypersensitivity reactions; but the occasional serious anaphylactoid reactions require the immediate use of epinephrine or phenylephrine, oxygen, and intravenous corticosteroids.

For full information see Package Insert.

**Supply:** The *calcium* salt (Oragrafin Calcium Granules) is available in single-dose foil packets providing 3 Gm. of calcium ipodate as Granules dispersed in flavored sucrose. The *sodium* salt is available in capsule form (Oragrafin Sodium Capsules) providing 0.5 Gm. sodium ipodate per capsule.

**in oral cholangiography and cholecystography**

# Oragrafin®

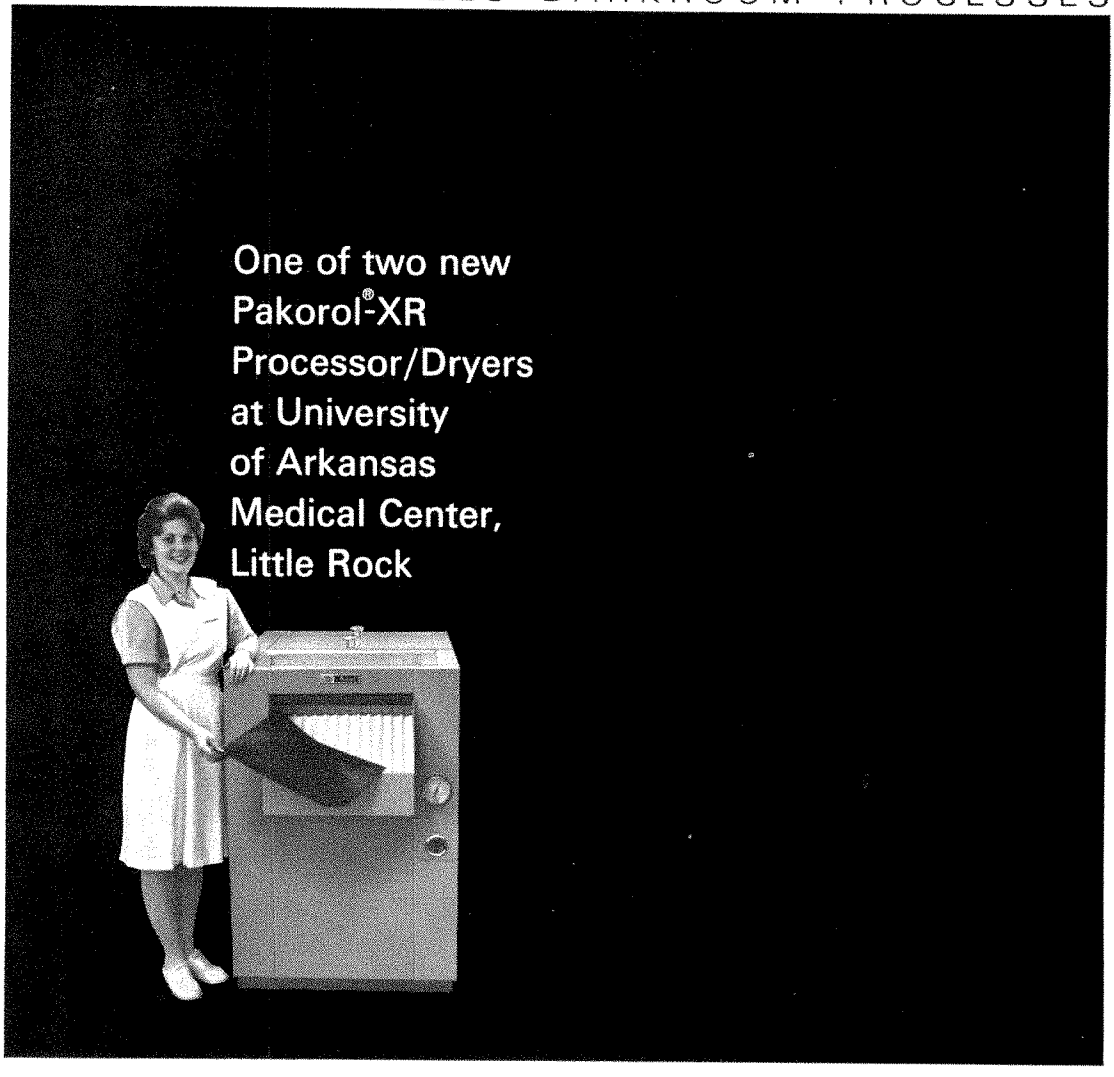
Squibb Ipodate

**for maximum concentration with better patient toleration**

SQUIBB

'The Priceless Ingredient' in every product is the honor and integrity of its maker.

PAKO SYSTEMATIZES DARKROOM PROCESSES




*University Medical Center needed x-ray processors that could change with their needs*

The University of Arkansas Medical Center in Little Rock, recently installed two Pakorol-XR's. These units not only serve their needs now, but can be modified later to faster processing with new films and new chemistry.

Normal Pakorol-XR processing time is 3½-4 minutes, dry-to-dry. They can be easily converted to 7½ minute or high speed (RP) processing. Their proven roller-transport and automatic control of variables result in consistent, high quality processing.

Pakorol-XR's process a variety of films: emergency, surgery, arteriography, orthography, mammography, fluorography and ciné. Contact your Pako X-Ray Distributor for information. Or write Pako Corporation, 6300 Olson Memorial Highway, Minneapolis, Minnesota 55440.

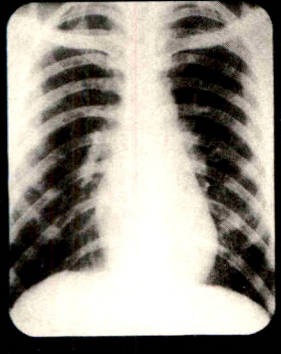
PAKO CORPORATION PAKO®

PS Processing Systems for the Photographic, Graphic Arts, Motion Picture, Medical and Industrial X-Ray Industries

# this is Hunt

# 90/90

DEVELOPER & REPLENISHER






***...a new ultra-concentrate***

***for processing all 90-SECOND***

***x-ray films at 90 to 94° F.***

# Hunt 90/90

DEVELOPER & REPLENISHER

...the first in a new generation of ultra-concentrates created especially for 90-second X-ray film processing.

...a product born from a remarkable discovery of Hunt research, CYCON*, a newly synthesized molecule that breaks the barriers of high temperatures and low solubility.

...a major technological achievement that offers radiologists the perfect 90-second processing chemistry because of these significant advantages:

***Reduced processing temperatures (90 to 94°F)***
● produces excellent sensitometric response formerly available at higher temperatures (98 to 107°F) ● minimizes mottle and haze ● affords greater control of chemical fog ● facilitates easier control of incoming tempered water supply.

***Less density variation / fluctuation*** despite changes in developer temperature (up to 6°F variation possible).

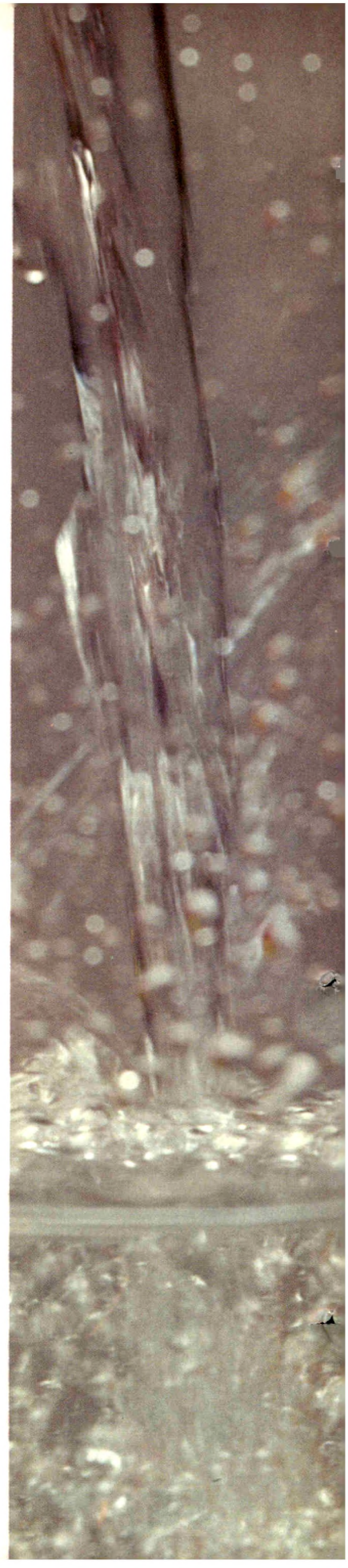
***Improved diagnostic film clarity*** of archival quality offers the radiologist more consistent, increased information than was ever possible with other processing chemistry at higher temperatures on 90-second film.

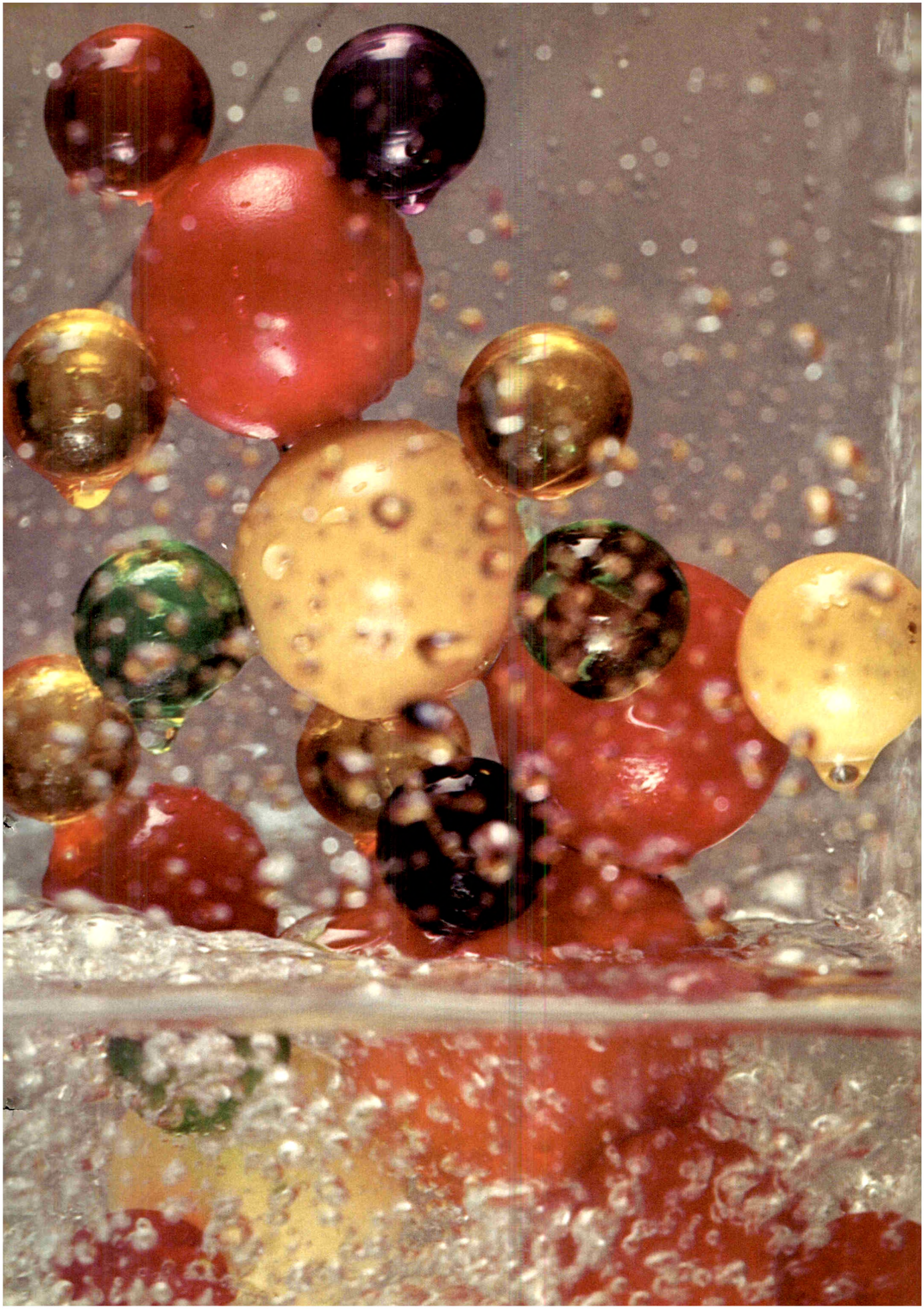
***Increased chemical concentration*** effected by CYCON reduces the standard package needed to make 20 gallons of ready-to-use solution to approximately: half the package size, to half the weight, and to one-sixth the mixing time. The design of the Hunt 90/90 carton makes the dispensing and mixing of this processing chemistry a foolproof procedure.






*Patent Pending



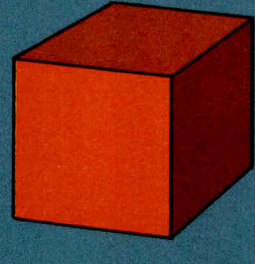


# *a word about CYCON, the new molecule that made Hunt 90/90 possible.*

*Hunt 90/90 Developer & Replenisher* is not just another concentrate. It is based on a totally new family of synthesized molecules called Cycon. For many years, the invention of a true ultra-concentrate photographic developer and replenisher which would combine the ease of use of a liquid concentrate with the size and weight of a dry powder has eluded the chemists attempting to develop highly concentrated liquid photographic processing chemicals. This Hunt molecule is a new organic chemical of high solubility which allows us to formulate highly concentrated liquid developers. The use of this new molecule eliminates the previously used inorganic salts which have only moderate solubility. It is this kind of product research and application that has made Hunt the leader in the evolution of automatic processing chemistry. The dedication of the Hunt research program is your assurance of continued advances in the technology of x-ray chemistry.

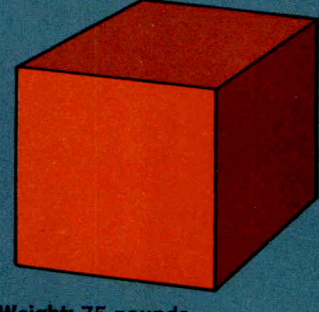
To obtain the ultimate diagnostic film quality in 90-second processing chemistry call Hunt today. Our sales force will guide you with up-to-the-minute advice on every aspect of the 90-second system and our trained staff of service engineers is ready to aid you in converting (if required) your existing processors to the 90-second cycle.

**90/90**
**Made with Cycon**



**Weight:** 46 pounds
**Size:** 1691 cubic inches
**Mixing time for 20 gallons:** 2 min.

**Former Developer**
**For 90-Second Films**

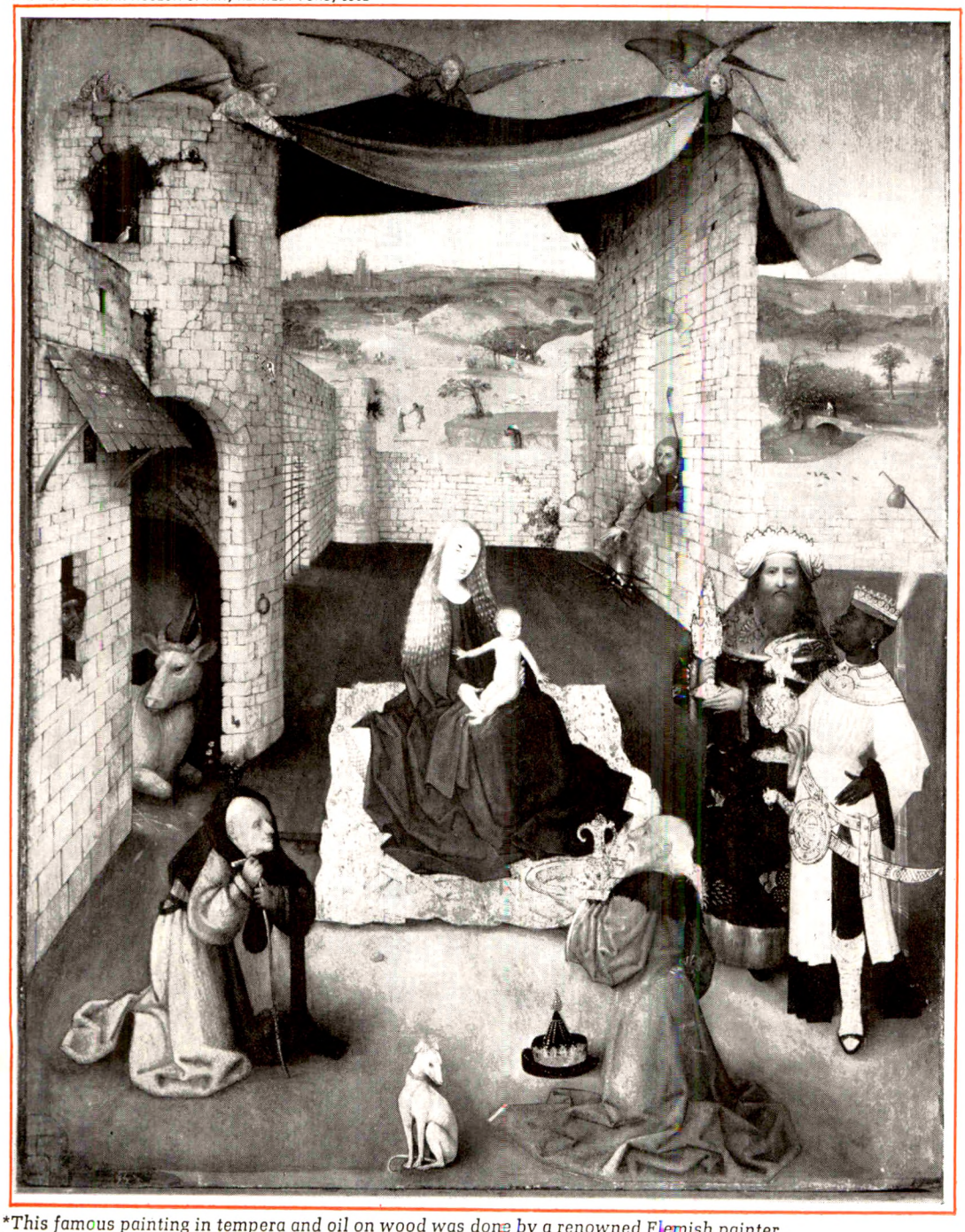


**Weight:** 75 pounds
**Size:** 2617 cubic inches
**Mixing time for 20 gallons:** 12 min.

MANUFACTURING CHEMISTS
HUNT
ESTABLISHED 1909

PHILIP A. HUNT CHEMICAL CORPORATION

THE METROPOLITAN MUSEUM OF ART, KENNEDY FUND, 1912



*This famous painting in tempera and oil on wood was done by a renowned Flemish painter. Do you know who it was?

Heir to a proud legacy... Agfa-Gevaert follows a brilliant heritage of craftsmanship with the production of medical X-Ray film known for its superb quality the world over.

*The Adoration of the Magi
Hieronymus Bosch

LXC | GEVAERT
X-RAY FILM

LOW X-RAY CORPORATION
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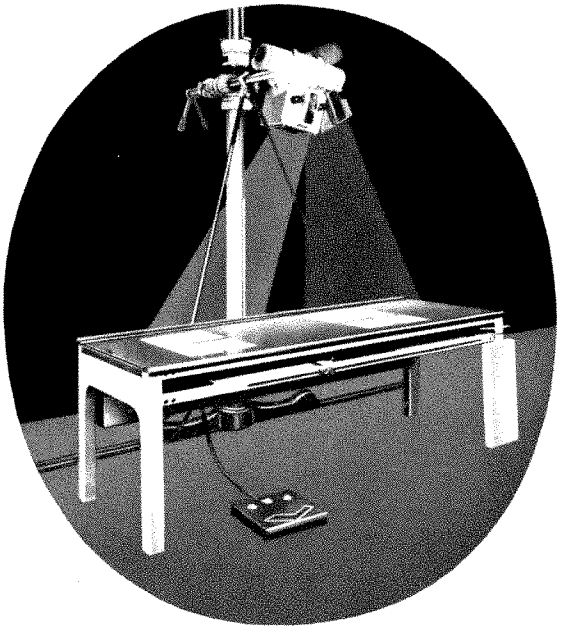
Continuing the Tradition of Flemish Excellence



# ARTERIOGRAPHY

## a simple unit permits exposures 14″ by 56″

Same table can also be used for routine radiography with or without bucky.



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**SQUIBB**

a research concept in radiopharmaceuticals

# The first sterile generator for Technetium-99m

The new Technetope Sterile Generator is a unique development in the field of radiodiagnostics and reflects the broad Squibb experience and technical know-how in this area. It offers a simple means of obtaining a sterile, non-pyrogenic supply of the radiopharmaceutical-Technetium-99m ($Tc^{99m}$) – a versatile scanning agent used for visualizing the brain that can be used both orally and intravenously. $Tc^{99m}$ is the decay product of Molybdenum-99 ($Mo^{99}$) and is produced by separation from its parent isotope. The relatively simple operation involves allowing the $Mo^{99}$ parent to decay and generate $Tc^{99m}$, then removing the $Tc^{99m}$ from the generating column by selective elution.

**non-pyrogenic and sterile, with important safeguards**

For the first time the physician can be assured of a supply of $Tc^{99m}$ that is both sterile and non-pyrogenic, since the Technetope generator is prepared with non-pyrogenic materials and sterilized by autoclaving. It consists of a specially designed lead shield containing an alumina-packed glass column that releases $Tc^{99m}$ upon elution. The lead shield has two access ports to the rubber closures at the top and bottom of the glass column, allowing aseptic elution and storage under conditions of constant shielding. (See Figure 1.) Additional shielding during shipment and during the elution procedure is provided by a removable lead sleeve that surrounds the entire assembly.

Eluting the generator every 24 hours will provide optimal amounts of $Tc^{99m}$. Most laboratories, therefore, will find it convenient to elute the generator at a specific time each day. However, the generator may be eluted whenever sufficient amounts of $Tc^{99m}$ have accumulated within the column.

**available with four different quantities of $Mo^{99}$**

The quantity of $Tc^{99m}$ eluted from the generator is, of course, dependent upon the quantity of $Mo^{99}$ present in the column. Technetope sterile generators contain either 50, 100, 200, or 300 millicuries of $Mo^{99}$ at the time of assay, depending on which activity has been requested. The activity obtained from subsequent elutions will depend on the time interval between elutions. (See Fig. 2.)

**Warning:** Proper radiation safety precautions should be maintained at all times. The glass column containing $Mo^{99}$ need not be removed from the lead shield at any time. The radiation field surrounding an unshielded column is quite high. Solutions of $Tc^{99m}$ withdrawn from the generator should always be adequately shielded. The early elutions from the generator are highly radioactive. For radiation protection, a lead shield for the collecting vial is included with Technetope.

**Precautions:** Radiopharmaceuticals should not be administered to pregnant women or patients under 18 unless the indications are very exceptional.

## This sterile and non-pyrogenic generator is a Squibb first. Of course, others may emulate it, but none can offer the body of experience that made it possible.

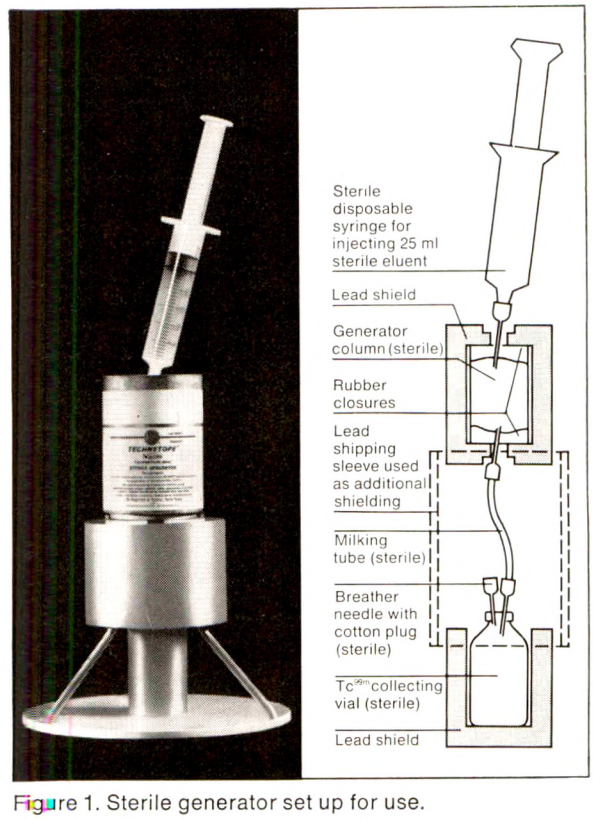



Figure 1. Sterile generator set up for use.

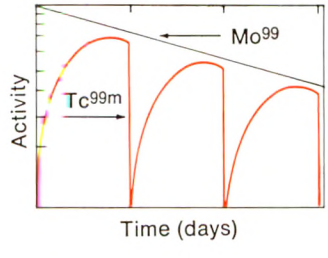



Figure 2. $Mo^{99}$ decay and $Tc^{99m}$ growth after daily elutions.

# Technetope®

## Squibb Technetium-99m Sterile Generator

SQUIBB 'The Priceless Ingredient' of every product is the honor and integrity of its maker.

# Diagnostic Problem:

**When**
- **Mechanical difficulties** (deformity, anomaly, etc.)
- **Debilitated states**
- **Infancy**
- **Advanced age**
- **Urinary tract infection**
- **Pregnancy**
- **Extensive pelvic surgery**

**contraindicate the use of cystoscope or catheter**



# Diagnostic Remedy:

## Excretory urography with Hypaque® 50%

# 4 Diagnostic Advantages:

1. **Precisely defined urograms of kidneys, ureters, and bladder, with valuable information about their functional status.**
2. **No interference with functional activity (sometimes a problem with instrumentation).**
3. **No age limit to the procedure.**
4. **No anesthesia required.**

**Warning:** Do not use Hypaque sodium for myelography. Injection of even a small amount into the subarachnoid space may produce convulsions and result in fatality.

**Contraindications:** Advanced renal destruction associated with severe uremia, and multiple myeloma.

**Precautions:** Caution is advised in patients with hyperthyroidism, hypertension, severe cardiovascular disease, active tuberculosis, and a history of asthma or other allergy, especially to iodine. Because of the possibility of temporary suppression of urine, it is wise to allow an interval of at least 48 hours before repeating retrograde or excretory urography in patients with unilateral or bilateral reduction of normal renal function.

**Preventive Measures:** The following precautions have been recommended to help prevent reactions in intravenous urography: (1) obtain a history of personal and familial allergies (for example, bronchial asthma, hay fever, and eczema), of previous iodine studies and of sensitivity to iodine and other drugs, (2) make a preliminary sensitivity test, (3) give preliminary antihistaminic medication, and (4) have medications on hand for emergency use.

**Side Effects:** Excretory urographic agents may produce symptoms of an anaphylactic* nature in sensitive persons. Patients should be watched carefully during injection as serious reactions, including fatalities, have occurred with all commonly used mediums. Although allergic reactions generally occur quickly, occasionally they may not be manifested for 10 or even 15 minutes. Warning signs and symptoms of possible intolerance or allergy include respiratory difficulty (wheezing, dyspnea or sensation of suffocation, and tightness in the throat or chest), sneezing, itching or urticaria, nausea or vomiting, and fainting. Minor reactions, such as nausea, vomiting, excessive salivation, flushing, dizziness, urticaria, and muscular twitching may occur. Special care is advisable in persons with a history of bronchial asthma or other allergic manifestations of sensitivity, especially to iodine. Infrequently "iodism" (Salivary gland swelling) appears two days after exposure and subsides by the sixth day. In euthyroid patients following excretory urography with Hypaque, protein-bound iodine is elevated and the thyroid $I^{131}$ uptake is lowered, returning to normal after the 3rd and 4th day respectively.

**Supplied:** In sterile aqueous solution, Ampuls of 30 cc. (with 1 cc. test ampuls), boxes of 1, 10 and 25. Also, rubber stoppered vials of 20 cc. and 30 cc., boxes of 1, 10 and 25.

*Physicians should study the package insert for information on preventive measures and management of untoward reactions *before* administering Hypaque (diatrizoate sodium).

## Urograms with excellent anatomical detail

# Hypaque® 50% (sodium)

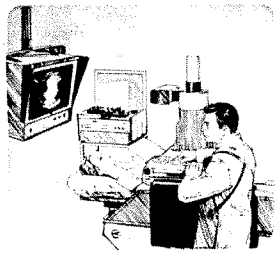
*Write for illustrated booklet containing complete information*

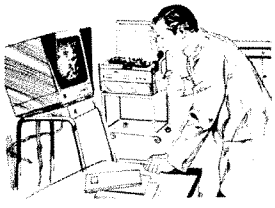
brand of **sodium diatrizoate**

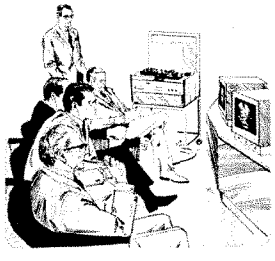
Winthrop PIONEERS IN PRODUCTS FOR RADIOLOGIC DIAGNOSIS

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# BROADENING THE SCOPE OF TV FLUOROSCOPY









## MEDICAL VIDEO RECORDER

**stores TV X-ray images and additional sound signals**

The Medical Video Recorder - designed exclusively for diagnostic radiology - completes any TV fluoroscopy installation.
It stores on magnetic tape the X-ray images transmitted via the TV chain from the image intensifier. Audio signals can be recorded simultaneously.
A perfect synchronization system, ample bandwidth and excellent screening against interference signals result in recorded images of outstanding quality, comparable to that of the original "live" TV images. Facilities for rapid starting and remote operation, presentation of still images and interchangeability of tapes.

**Applications**

- radiological training
- re-examination without patient
- post-examination diagnosis
- pre-operative study of recorded examinations
- motion and function study
- repeated viewing and more concentrated study of TV fluoroscopic images
- study of essential phases **during** as well as **immediately after** e.g. heart | examination
- quick quality check of cinefluorographic shots

Further information supplied by your national Philips organization or agent, as well as from: Philips X-ray & Medical Equipment Division Eindhoven - the Netherlands.

PHILIPS

# Swing to a better look with Cenco X-ray film

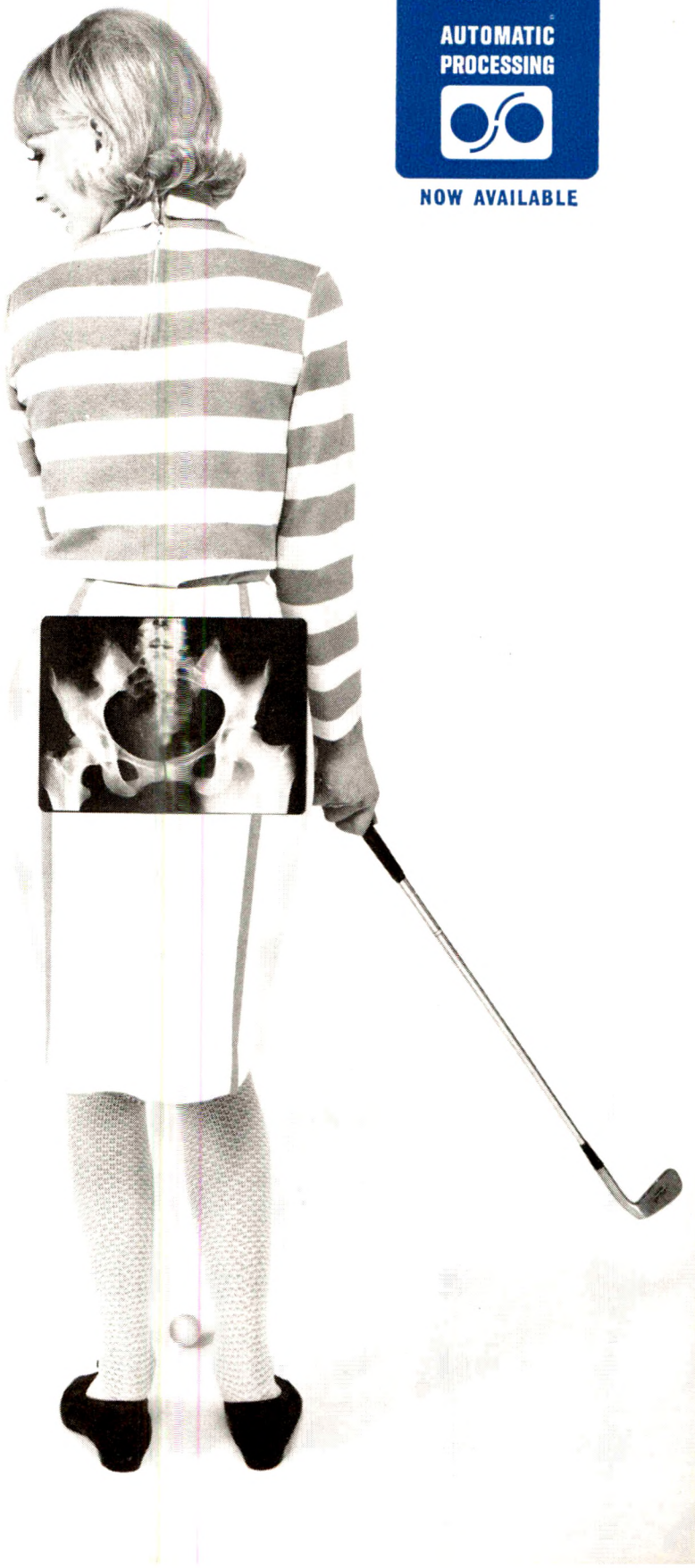
NEW!
**Ferrania N**
X-RAY FILM FOR
**AUTOMATIC PROCESSING**
**NOW AVAILABLE**

Hips look better on Cenco-Ferrania Type N X-ray film. It's a premium-quality, yet moderately-priced, film that gives you superior contrast, detail, and definition sheet after sheet. Not to be overlooked is the fact that Type N is also faster than the medical X-ray film most widely used in the United States. Type N can be processed either manually or automatically and is available in thrifty bulk packs or standard boxes.

Your Cenco X-ray dealer can immediately deliver Type N as well as other Cenco-Ferrania films for non-screen or fine-tissue work, photofluorography, or film to give you direct copies in as little as two minutes. He can also deliver Cenco processing chemicals that enable you to produce superior radiographs with all brands of film.

You will find that your Cenco X-ray dealer breaks par with service and value. He is uniquely attuned to your particular needs because serving radiologists is his *only* business. Why not call him today? If you need his number or a copy of our film catalog, write us.

CXR-8-212



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# Shedding a conventional 12½ lb. leaded x-ray apron and donning our new light one is just like emptying your pockets of 1 silver dollar, 2 half dollars, 87 quarters, 38 dimes, 99 nickels, 181 pennies, a ball point pen, 1 pocket knife and 4 keys.



But despite this lower weight, you lose no protection with this apron. Our new Lite-Gard apron fully provides the required 0.5 mm lead equivalent protection at 80 KV. Yet it actually weighs up to 4 lbs. less than conventional x-ray aprons.

If you now challenge your Picker salesman to prove that your current apron is unnecessarily overweight, he's likely to pull out a scale (unrigged) right on the spot. Try it.

But you need not wait for said demonstration: details will follow your request for file MA-32.

**PICKER X-RAY** Corporation, 1275 Mamaroneck Ave., White Plains, N.Y. 10605




## Red Seal X-ray film on Melinex® polyester base

The premium Red Seal X-ray film now being supplied is made with an entirely new and highly efficient emulsion coated on Melinex polyester base and is designed to produce maximum radiographic information.

Red Seal on Melinex base is a high speed film with excellent resolving power, resulting in minimum exposures and sharp, clean images. Fog is at an absolute minimum for a film of this speed. This new Red Seal emulsion will give equal satisfaction throughout the entire medical KV range.




## Standard X-ray film on Melinex® polyester base

Standard X-ray film on Melinex base, like Red Seal, is designed to produce maximum radiographic information.

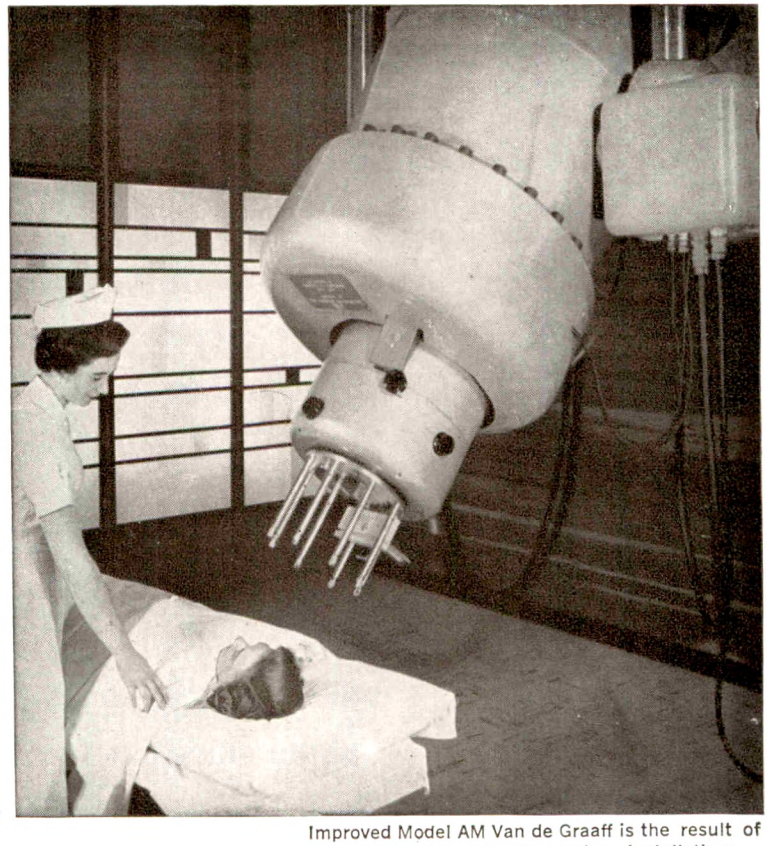
This fine film offers all the qualities sought by radiologists: high contrast, medium speed, fine grain (particularly suitable for use with high KVP techniques); exceptionally low fog level, and the famous Ilford batch-to-batch and box-to-box consistency.

Standard X-ray emulsion on Melinex base is particularly recommended for all investigations where the highest contrast and definition are required.

Both Ilford Red Seal and Standard X-ray films on Melinex assure superior performance in all "full cycle" and "half cycle" processing systems. In addition, these new films permit substantial reduction in drying temperatures. Packings include 300 and 500 sheet economy bulk packs, and 75 and 25 sheet cartons in all popular sizes. We invite a trial test of their excellence.

# ILFORD INC.

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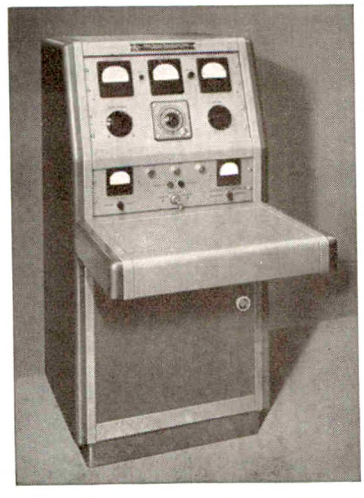


Improved Model AM Van de Graaff is the result of experience gained in 40 previous installations.

# IMPROVED *Van de Graaff*® ACCELERATOR MEETS THE MODERN NEED FOR LOW-COST PRECISION THERAPY

Today's Model AM 2 MeV Van de Graaff Therapy Unit continues to keep pace with the trend to low-cost precision therapy in leading hospitals and cancer clinics throughout the Free World. And for five good reasons:

- Its less than 3 mm "point source" of x-rays allows optimum technics for both large and small treatment fields.
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# THE ROENTGENOLOGIC AND RADIOISOTOPIC ASSESSMENT OF HYDATID DISEASE OF THE LIVER*

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HYDATID disease is comparatively uncommon in Australia[9,10] but its frequency is not decreasing.[15] The lungs are involved in 4–14 per cent of patients[9] and this can be readily detected. The liver is affected in 75 per cent of cases[2] but in the absence of characteristic calcification the roentgenologic diagnosis of hydatid disease of the liver is difficult.[19,25,27] Blanchon *et al.*[3] in 1960, and Czerniak *et al.*[7] in 1961 reported the usefulness of liver scanning in the detection of hydatid disease and since then other workers have confirmed its value.[1,11,28] In this report 16 cases with a provisional diagnosis of initial or recurrent hydatid disease of the liver, leading to laparotomy, are described. The roentgenologic and radioisotopic features are discussed.

## MATERIALS AND METHODS

The provisional diagnosis of hydatid disease of the liver was based upon the history and physical examination, and on laboratory and roentgenologic investigations (Table 1). Liver scans were performed using a commercial scanner* after the intravenous injection of 100 μc of colloidal† $Au^{198}$. Liver-lung scans were obtained using radioactive colloidal gold $Au^{198}$ and $I^{131}$ macroaggregated human serum albumin with a combined $Au^{198}$ and $I^{131}$ window. All patients were scanned in at least two planes; an anterior scanning was first performed then a right lateral scanning. Posterior scannings were performed only if a defect was posteriorly placed. The costal margin, liver edge and other palpable masses were outlined on the patient's skin and transcribed to the scan record (Fig. 1). Physical examination of the liver was repeated on completion of the scanning with particular attention to regions correspond-

* Picker Magnascanner 3×2 inch crystal, 19 hole collimator, 2107.
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TABLE I

SUMMARY OF CASE MATERIAL

| Case No. | Illustrations | Age (yr.) | Sex | Country of Birth | Place of Childhood Residence | Mode of Presentation | Physical Findings | History of Previous Operations | Casoni Intradermal Test | Hydatid Complement Fixation |
|---|---|---|---|---|---|---|---|---|---|---|
| I | Fig. 2, *A–D*; and 3, *A* and *B* | 21 | M | Australia | Rural Australia | Epigastric mass | Hepatomegaly | 1963: thoracotomy for right lung hydatid | Not done | Negative |
| II | Fig. 4, *A–D*; and 5, *A–D* | 52 | F | Australia | Rural Australia | Initially pleural effusion; subsequently right hypochondrial pain | Initially right pleural effusion | 1959: right thoracotomy | Positive | Positive |
| III | Fig. 6, *A–D* | 70 | F | Australia | Rural Australia | Abdominal swelling and pain; dyspnea | Gross abdominal distention | Nil | Negative | Not done |
| IV | Fig. 7, *A–D* | 69 | M | Australia | Rural Australia | Pyrexia; anemia; right hypochondrial pain; hepatomegaly | Hepatomegaly with resonance to percussion in the right hypochondrium | Nil | Negative | Positive |
| V | Fig. 8, *A–D*; and 9, *A–D* | 57 | M | Australia | Rural Australia | Pruritus and jaundice | Jaundice; hepatomegaly | 1932: large hydatid cyst removed from right lobe of liver. 1941: cholecystectomy | Not done | Positive |
| VI | Fig. 10, *A–D* | 62 | M | Greece | Greece | Pruritus and jaundice | Jaundice | 1952: laparotomy and liver biopsy. 1957: splenorenal anastomosis | Positive | Positive |
| VII | Fig. 11, *A* and *B* | 41 | M | Australia | Rural Australia | Upper abdominal pains for 18 months | Marked hepatomegaly | Nil | Positive | Positive |
| VIII | Fig. 12, *A* and *B* | 25 | F | Greece | Rural Greece | 1962: unresolved pneumonia. 1965: hemoptysis | 1962: unresolved pneumonia. 1965: hepatomegaly; right basal consolidation | 1949: thoracotomy for lung hydatid. 1963: laparotomy for liver hydatid | Negative | Negative |
| IX | Fig. 13, *A–F* | 42 | F | Australia | Rural Australia | Calcified cyst noted on barium meal examination | Deep right hypochondrial tenderness | Nil | Positive | Negative |
| X | Fig. 14, *A–F* | 53 | F | Australia | Rural Australia | Round lesion seen on routine chest roentgenogram; scanning done as routine follow-up | Normal | 1957: thoracotomy for lung hydatid | Positive | Positive |
| XI | Fig. 15, *A–D* | 59 | M | Australia | Rural Australia | Right basal pleuritic pain | Right basal chest signs (hydatid elements detected in pleural aspirate) | Nil | Negative | Positive |
| XII | Fig. 16 | 47 | M | Cyprus | Cyprus | 1953: jaundice. 1961: recurrence of hydatid cyst and jaundice. 1965: 24 hour history of enlarging epigastric mass | Tender epigastric mass | 1953, 1961: removal of hydatid cysts from liver | Negative | Negative |

TABLE I (*Continued*)

| Eosinophil Cells/mm.³ | Serum Alkaline Phosphatase (King-Armstrong units) | Roentgenologic Assessment | Radioisotopic Assessment | Operative Diagnosis |
|---|---|---|---|---|
| 95 | 15 | Possible hepatomegaly; right hemidiaphragm considered normal; however, on review after the scanning there was a localized elevation of the right hemidiaphragm corresponding to the upper edge of the defect seen on the scan; no calcification | A hemispherical defect anterosuperiorly in the right lobe | Single hydatid cyst anterosuperiorly in the right lobe<br>Operation 1964 |
| 3,000 | 12 | In serial roentgenograms after initial operation localized right hemidiaphragmatic elevation was regarded as constant; on review after the scanning an increase in elevation was thought to have occurred; no calcification | Hemispherical defect posterosuperiorly in the right lobe; costal margin evident in both anterior and lateral scans | Single hydatid cyst posterosuperiorly in the right lobe<br>Operation 1964 |
| Normal | 22 | Large marginally calcified gas-fluid filled multiseptate cyst in liver with marked elevation of the right hemidiaphragm | Large single defect superolaterally in the liver with gross displacement of the remainder of the liver | Large single hydatid cyst containing liters of fluid<br>Operation 1963 |
| 900 | 61 | Large gas-fluid filled cyst below right diaphragm; no right hemidiaphragmatic elevation; no calcification | Marked displacement of liver to the left | Large single hydatid cyst<br>Operation 1965 |
| 163 | 36 | Localized elevation of right hemidiaphragm; calcification present | Inferolateral portion of right lobe absent, with defect between remainder of right lobe and hypertrophied left lobe; biliary excretion of $I^{131}$ rose bengal delayed; interlobar defect less obvious on postoperative scan | Large masses of small hydatid cyst filling the porta hepatis separating the right and left lobes causing bile duct obstruction and extending vertically through to the upper surface of the liver causing elevation of the right hemidiaphragm<br>Operation 1964 |
| 1,000 | 66 | Splenoportogram (1957) showed a space occupying lesion in the right lobe of the liver; no calcification | Multiple areas of reduced and absent uptake throughout the liver; renal scan demonstrated a defect at the lateral edge of the right upper pole | Multiple small hydatid cysts throughout the liver macroscopically resembling metastases; portal hypertension present but its cause was uncertain; histopathologic examination of liver biopsy specimen revealed hydatid disease<br>Operation 1957 |
| 400 | 12 | Roentgenograms of chest and abdomen normal; screening of diaphragms normal; Graham's test normal; no calcification | Large hemispherical defect involving the inferior edge of the right lobe | Hydatid cyst of the right lobe of the liver<br>Operation 1965 |
| 800 | 11 | Abdominal roentgenogram normal; no calcification | Hemispherical defect inferolaterally in the right lobe | Single large hydatid cyst of the right lobe of the liver<br>Operation 1965 |
| 1,000 | 9 | Large calcified hydatid cyst in liver; initially right hemidiaphragm considered normal; on review after scanning a localized elevation of the right hemidiaphragm evident in the right lateral roentgenogram corresponded to the upper edge of the defect seen on the scan | Large defects in upper portion of the right lobe; the scan defects were more extensive than the area of calcification | Two large hydatid cysts superolaterally in the right lobe<br>Operation 1965 |
| 1960: 2,200<br>1965: 48 | 12 | Follow-up chest roentgenogram clear; roentgenogram of liver after scanning demonstrated a large calcified hydatid cyst in the right lobe; on review of the right lateral roentgenogram, there was a localized elevation corresponding to the upper edge of the defect seen on the scan | Large posteriorly placed hemispherical defect superolaterally in the right lobe | Large hydatid cyst superolaterally in the right lobe<br>Operation 1965 |
| 500 | 17 | Localized elevation of right hemidiaphragm, with an area of pulmonary consolidation above it; no calcification | Liver-lung scan showed a defect between the right upper lateral surface of the liver, and the lower lobe of the right lung | Hydatid cyst in the right subphrenic space, originating from the liver, and extending upwards through the diaphragm<br>Operation 1965 |
| Not done | 24 | Hydatid cyst in right second rib, and in lung; right hypochondrial mass continuous with liver; no calcification | Left lobe not seen; right lobe enlarged; no scan defect corresponding to the palpable mass | Pedunculated hydatid cyst originating in the liver and extending into the anterior abdominal wall<br>Operation 1965 |

TABLE I—(*Continued*)

| Case No. | Illustrations | Age (yr.) | Sex | Country of Birth | Place of Childhood Residence | Mode of Presentation | Physical Findings | History of Previous Operations | Casoni Intradermal Test | Hydatid Complement Fixation |
|---|---|---|---|---|---|---|---|---|---|---|
| XIII | Fig. 17 | 20 | M | Greece | Rural Greece | Right basal pneumonia | Liver normal; right basal chest consolidation | Nil | Negative | Positive |
| XIV | Fig. 18 | 7 | F | Australia | Urban Australia | Pruritus 6 mo.; right hypochondrial mass detected by mother | Hepatomegaly with localized mass at lower right edge | Nil | Negative | Positive |
| XV | Fig. 19, *A* and *B* | 37 | F | Australia | Urban Australia | Initially right hypochondrial swelling, vomiting and pruritus; subsequently numerous admissions with abdominal pain | No abdominal masses palpable; left lobe not palpable | 1962: multiple hydatid cysts in region of celiac axis; cyst of right lobe of liver and gallbladder bed | Positive | Negative |
| XVI | Fig. 20, *A–C* | 67 | M | Australia | Rural Australia | 1965: round lesion discovered on routine chest roentgenogram | Normal | Nil | Not done | Not done |

ing to scan defects. All the scans were interpreted strictly in conjunction with the clinical, roentgenologic and laboratory findings. The final diagnosis was established in each case by surgical exploration. Operation was performed in the same year as the scan in all patients except Case VI.

## REPORT OF CASES

CASE I. (Fig. 2, *A–D*; and 3, *A* and *B*). A 21 year old male had a left thoracotomy for lung hydatid 1 year previously. Hepatomegaly was present on follow-up examination. The hydatid complement fixation test was negative and the eosinophil count and serum alkaline phosphatase were normal. Roentgenologic examination suggested hepatomegaly but the right hemidiaphragm was considered normal. Liver scan revealed downward displacement of the liver and there was a large hemispherical defect anterosuperiorly in the right lobe. When the chest roentgenograms were re-examined localized elevation of the right hemidiaphragm corresponded to the upper edge of the defect evident on the scan.

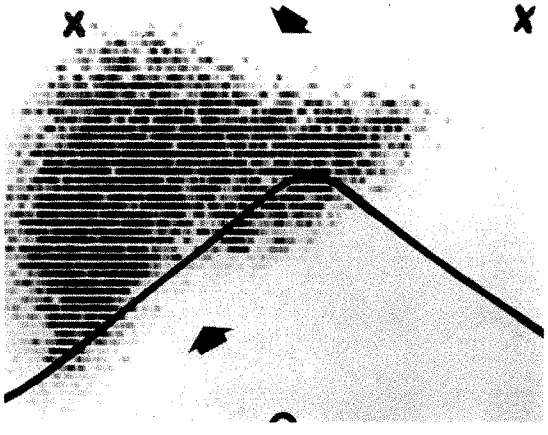


FIG. 1. Normal liver scan. The upper arrow indicates the cardiac impression, the lower arrow the hepatic notch. This scan was obtained in a male patient. X refers to the position of the nipples. The costal margin is drawn.

CASE II. (Fig. 4, *A–D*; and 5, *A–D*). A 52 year old female presented with pleural effusion. The Casoni and hydatid complement fixation tests were positive, there was marked eosinophilia, but the serum alkaline phosphatase was normal. A thoracotomy revealed a hydatid cyst protruding through the right hemidiaphragm. Follow-up investigation included serial roentgenograms. Six years after the initial operation, liver scan revealed a large hemispherical defect posterosuperiorly in the right lobe of the liver. Review of the roentgenograms indicated that a localized elevation of the right hemidiaphragm was present above the scan defect. At exploration a hydatid cyst was found. The defect was less apparent in a postoperative scan.

CASE III. (Fig. 6, *A–D*). A 70 year old female first presented 12 years prior to scanning when she was diagnosed as having disseminated hydatid disease of the liver and peritoneal cavity. Exploration was not undertaken at

Table I—(*Continued*)

| Eosinophil Cells/mm.³ | Serum Alkaline Phosphatase (King-Armstrong units) | Roentgenologic Assessment | Radioisotopic Assessment | Operative Diagnosis |
|---|---|---|---|---|
| 510 | 15 | Rounded opacity in posterior segment of lower lobe of right lung evident after resolution of initial pneumonia; no calcification | Liver scan normal | One hydatid in the lung and one, 1.5 cm. in diameter, on the upper surface of the right lobe of the liver<br>Operation 1965 |
| 190 | 58 | Hepatomegaly; no calcification | Hepatomegaly; isotope uptake was uniform throughout the liver including the palpable mass | Operative biopsy revealed hepatitis with possible biliary cirrhosis<br>Operation 1964 |
| 250 | 11 | Calcified hydatid cyst in left lobe of liver | A poorly defined left lobe and a defect at the medial edge of the right lobe; no mass palpable in the region of the scan defect | Calcified plaque on the inferior surface of the left lobe of the liver; no hydatid cyst<br>Operation 1964 |
| 150 | Not done | Hydatid cyst in lower lobe of right lung; calcification in region of the left lobe of the liver | Liver scan normal; no scan defect corresponding to calcification evident on roentgenogram | Single cyst in the right lower lobe of the lung; no cyst evident in the liver<br>Operation 1965 |

that time. She was readmitted complaining of right sided abdominal pain, gross abdominal distention and dyspnea. The Casoni test was negative, hydatid complement fixation was not done and there was no eosinophilia. The serum alkaline phosphatase was 22 King-Armstrong units. Roentgenologic examination disclosed marked elevation of the right hemidiaphragm and a large marginally calcified gas-fluid filled multiseptate cyst. The liver scan indicated a large single defect with gross displacement of the remaining liver. At laparotomy a single cyst containing 16 liters of fluid was present.

Case iv. (Fig. 7, *A–D*). A 69 year old male was admitted with pyrexia, anemia, right hypochondrial pain and hepatomegaly. The right hypochondrium was resonant to percussion. The Casoni test was negative, the hydatid complement fixation test was positive and there was an eosinophilia of 900 cells/mm.³. The serum alkaline phosphatase was 61 King-Armstrong units. Roentgenologic examination showed a large multiseptate gas-filled cyst below the right hemidiaphragm. No calcification was present. Liver scan demonstrated gross displacement of the liver. At laparotomy a large single hydatid cyst was found containing fluid and purulent gelatinous material.

Case v. (Fig. 8, *A–D*; and 9, *A–D*). A 57 year old male had a large hydatid cyst removed from his liver 32 years before he presented with jaundice, pruritus and hepatomegaly. The hydatid complement fixation test was positive, there was no eosinophilia and the serum alkaline phosphatase was 36 King-Amstrong units. Roentgenologic examination showed localized elevation of the right hemidiaphragm and calcification was present. Liver scan showed the inferolateral part of the right lobe to be absent. As no mass was palpable in this region this was attributed to the previous operation. There was a scan defect extending through to the diaphragm between the remainder of the right lobe and the hypertrophied left lobe. Biliary excretion of $I^{131}$ labelled rose bengal was delayed. At laparotomy large masses of small hydatid cysts were seen filling the porta hepatis, obstructing the bile ducts and extending through to the upper surface of the liver. Postoperative scans showed the defects to be smaller and roentgenograms indicated a normal right hemidiaphragm.

Case vi. (Fig. 10, *A–D*). A 62 year old male had a laparotomy in the course of investigation of jaundice and the liver was thought to be diffusely involved with secondary neoplasm; biopsy, however, revealed multiple hydatid cysts. At this time the Casoni test and the hydatid complement fixation tests were positive and eosinophilia was present. The serum alkaline phosphatase was 66 King-Armstrong units. He subsequently developed portal hypertension and splenoportography demonstrated varices and a space occupying lesion of the right lobe of the liver. Liver scan 12 years later demonstrated multiple areas of reduced and absent isotope uptake throughout the liver.

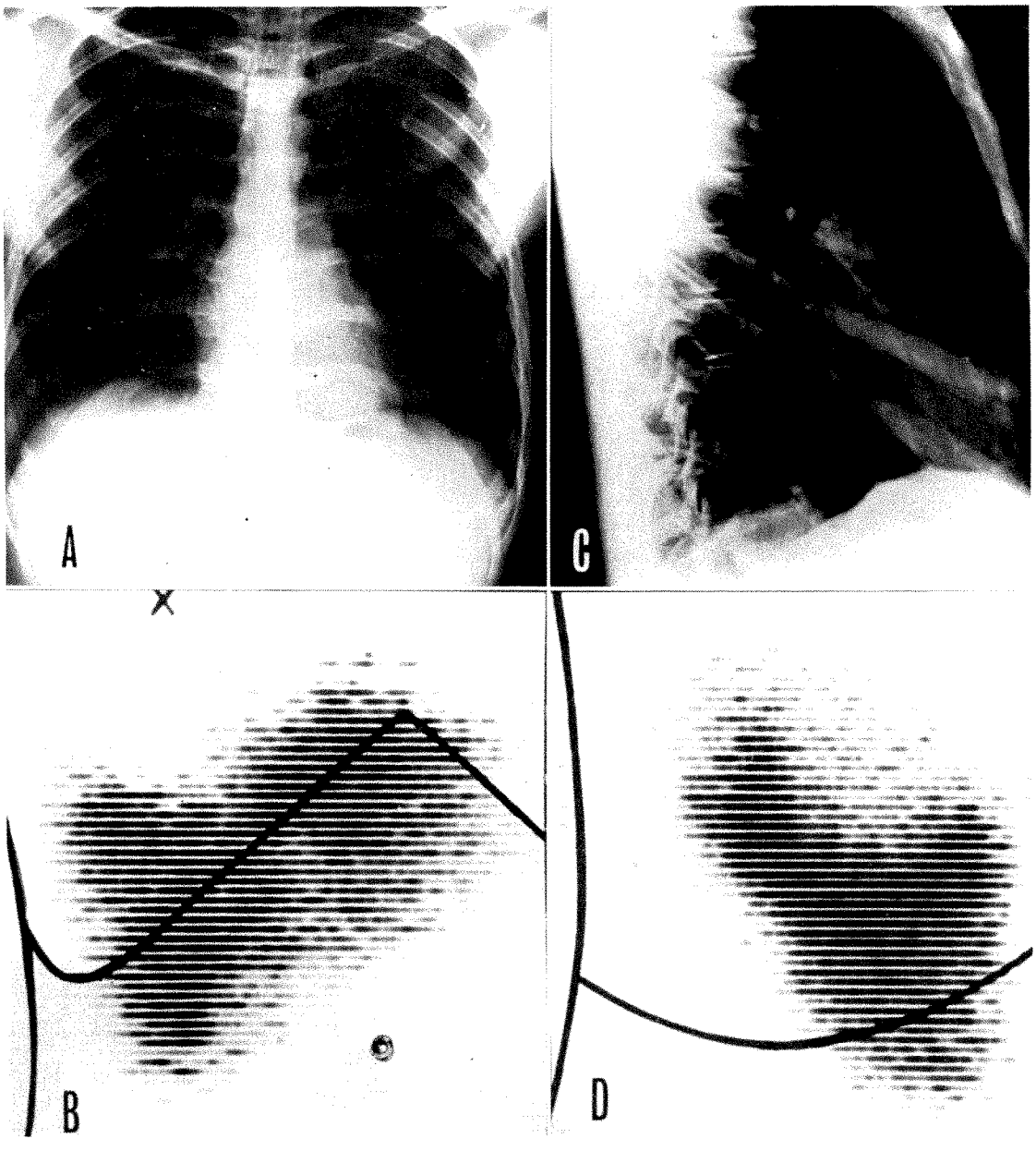



FIG. 2. Case 1. (*A*) A hydatid cyst is seen in the upper zone of the left lung. (*A* and *C*) The right hemidiaphragm, although initially considered normal, shows a localized elevation completing the spherical outline of the liver cyst. (*B*) Anterior and (*D*) right lateral scans demonstrate a hemispherical defect anterosuperiorly in the right lobe.

A renal scan was performed using $Hg^{203}$ chlormerodrin and a defect at the lateral edge of the upper pole of the right kidney was demonstrated.

Case VII. (Fig. 11, *A* and *B*). A 41 year old male had complained of intermittent upper abdominal pain for 18 months. Examination revealed marked hepatomegaly. The Casoni and hydatid complement fixation tests were positive but the eosinophil count and serum alkaline phosphatase were normal. Roentgenologic examination demonstrated some limitation of movement of the right hemidiaphragm but plain abdominal roentgenogram, Graham's test and an intravenous cholangiogram were normal. The liver scan indicated a large hemispherical defect in the lower part of the right lobe; this defect was immediately above the large palpable mass. At laparotomy a hydatid cyst of the lower part of the right lobe of the liver was present.

Case VIII. (Fig. 12, *A* and *B*). A 25 year old female had a thoracotomy for lung hydatid 16 years previously and a laparotomy for removal of a liver hydatid 12 years before she presented with hemoptysis. On examination there was a large palpable mass in the right hypochondrium that was clinically considered to be of hepatic origin. The Casoni and hydatid complement fixation tests were negative and the serum alkaline phosphatase was normal. Part of the right lobe had been removed at the previous operation but there was a large hemispherical scan defect inferolaterally in the right lobe with a palpable mass corresponding to the scan defect. At laparotomy a large multilocular cyst was present protruding from the undersurface of the right lobe of the liver.

Case IX. (Fig. 13, *A–F*). A 42 year old female presented with a 3 year history of vague upper abdominal pain. Barium meal examination was performed and a calcified area was noted in the region of the liver. The Casoni test was positive, the hydatid complement fixation test was negative, the eosinophil count was elevated and the serum alkaline phosphatase was normal. A large defect corresponding to the area of calcification was evident on the liver scan. There was an additional scan defect medial to this. In the lateral scan the upper third of the right lobe was absent. At exploration two hydatid cysts in the right lobe were found to be re-

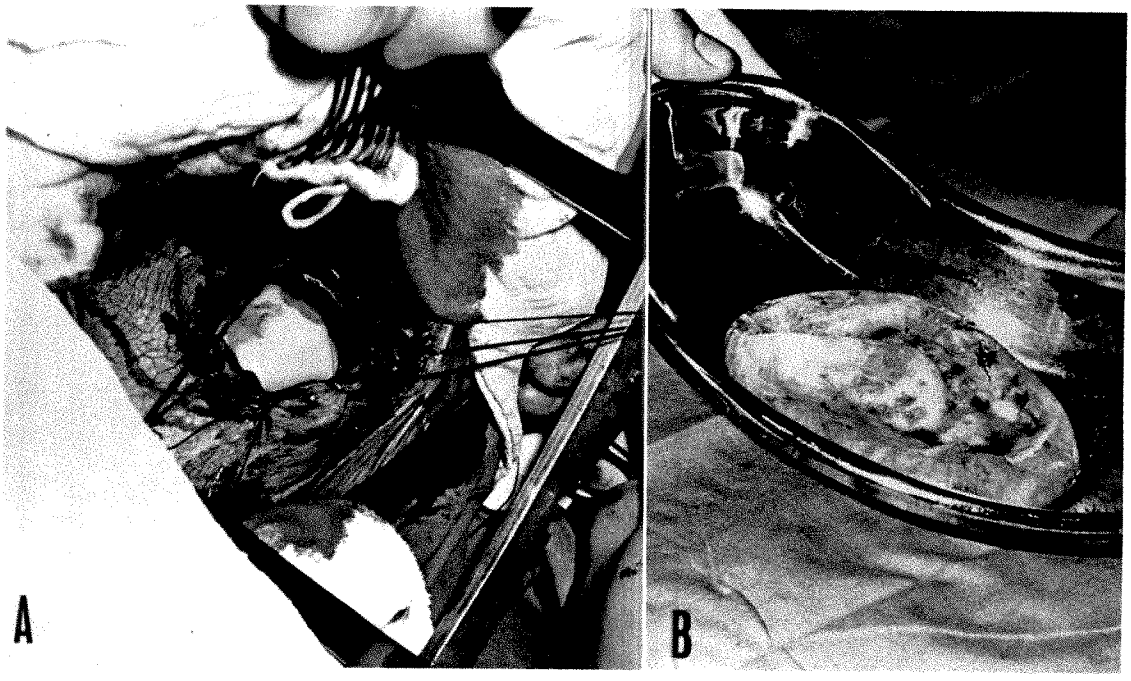



Fig. 3. Case I. (*A*) At operation the white upper surface of the cyst is seen through the diaphragmatic incision. (*B*) Collapsed hydatid cyst after removal.

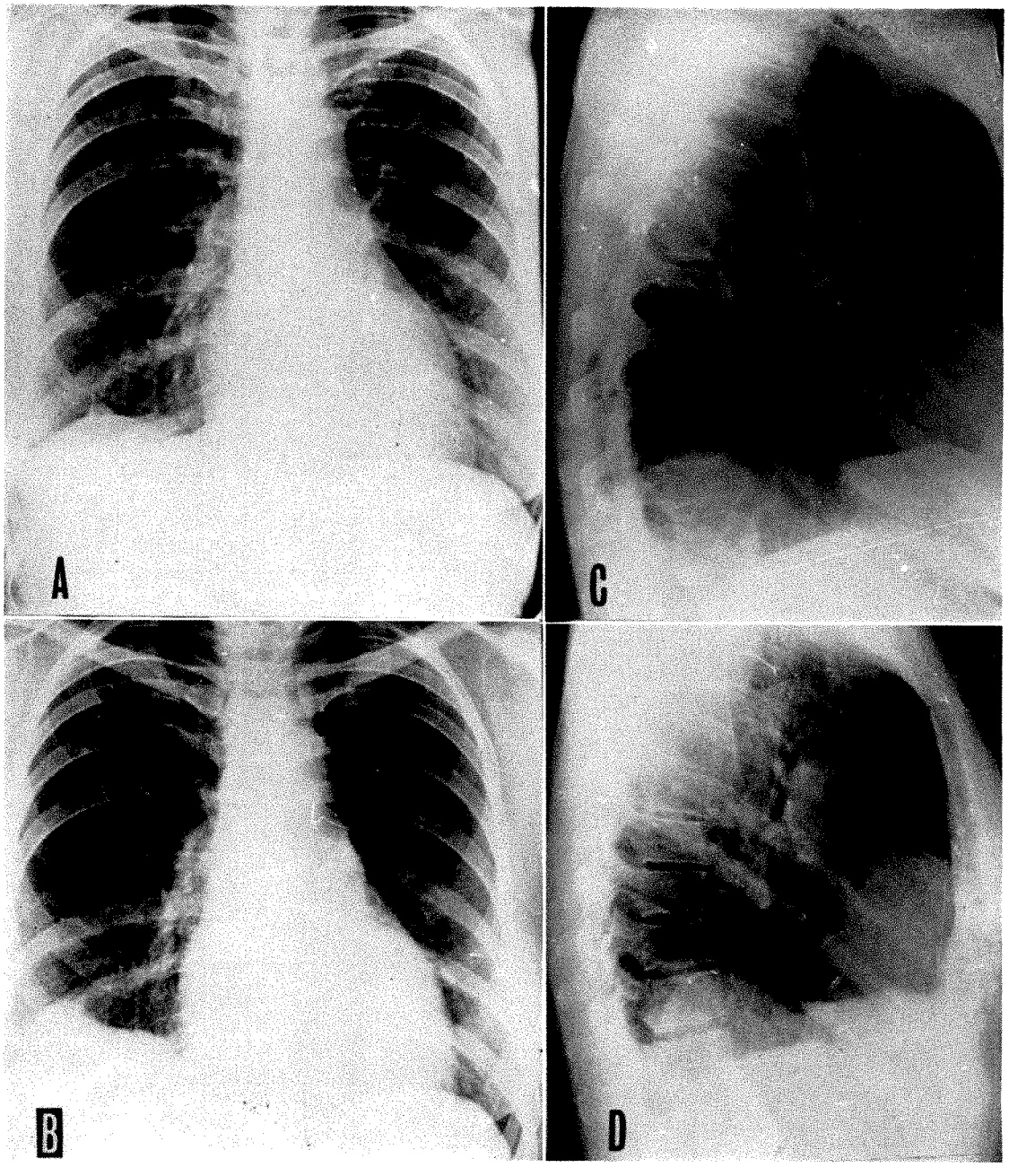



FIG. 4. Case II. (*A* and *C*) In 1960, anteroposterior and right lateral roentgenograms demonstrated small localized elevation of the right hemidiaphragm. (*B* and *D*) In 1963 this elevation had increased.

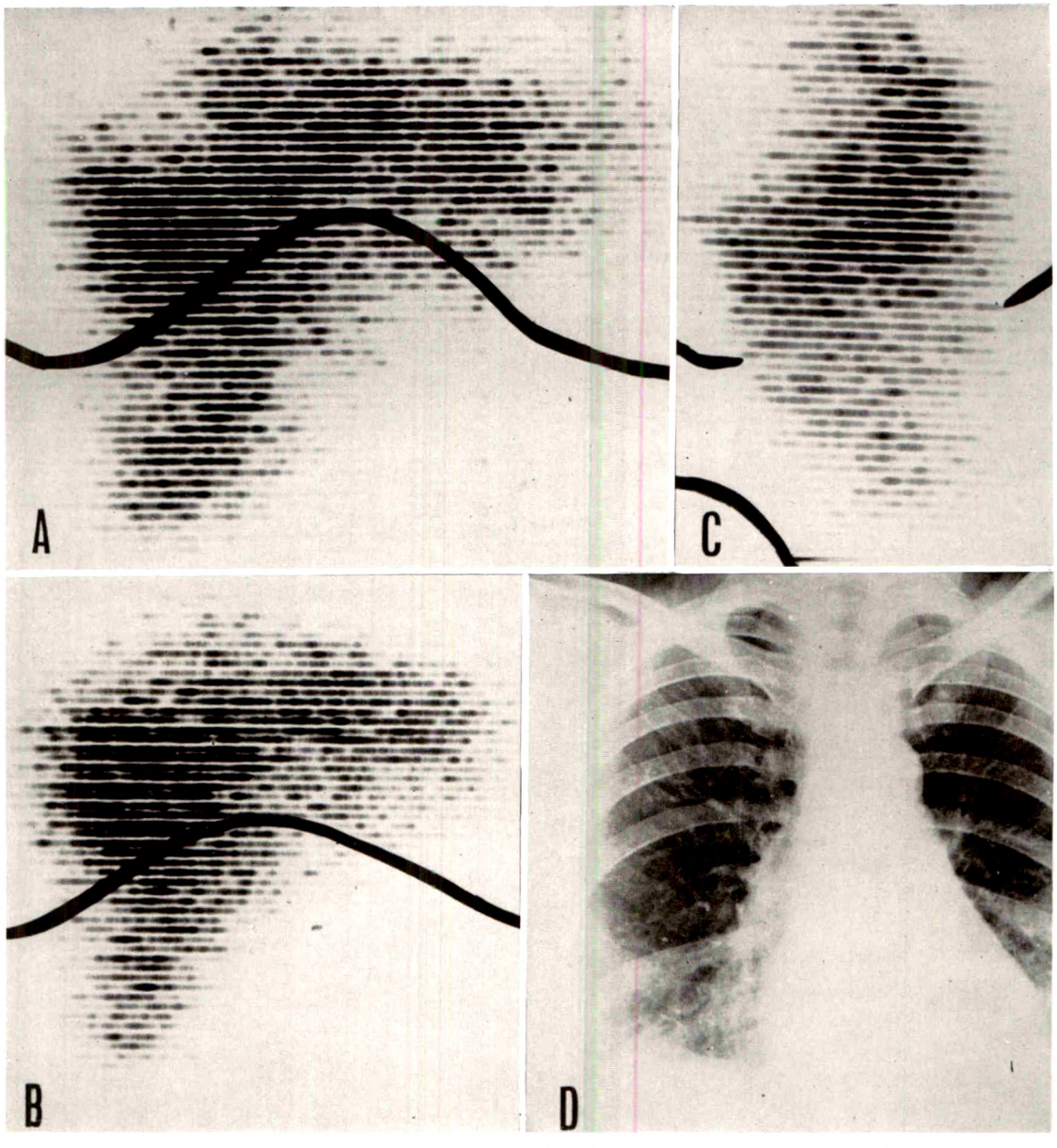



Fig. 5. Case 11. (*A* and *C*) Preoperative anterior and right lateral liver scans demonstrate a hemispherical defect posterosuperiorly in the right upper lobe. (*B*) This defect is less obvious in the postoperative scan. (*D*) In the postoperative roentgenogram the diaphragmatic elevation has decreased. The diminished radioactivity beneath the right costal margin is the costal impression.

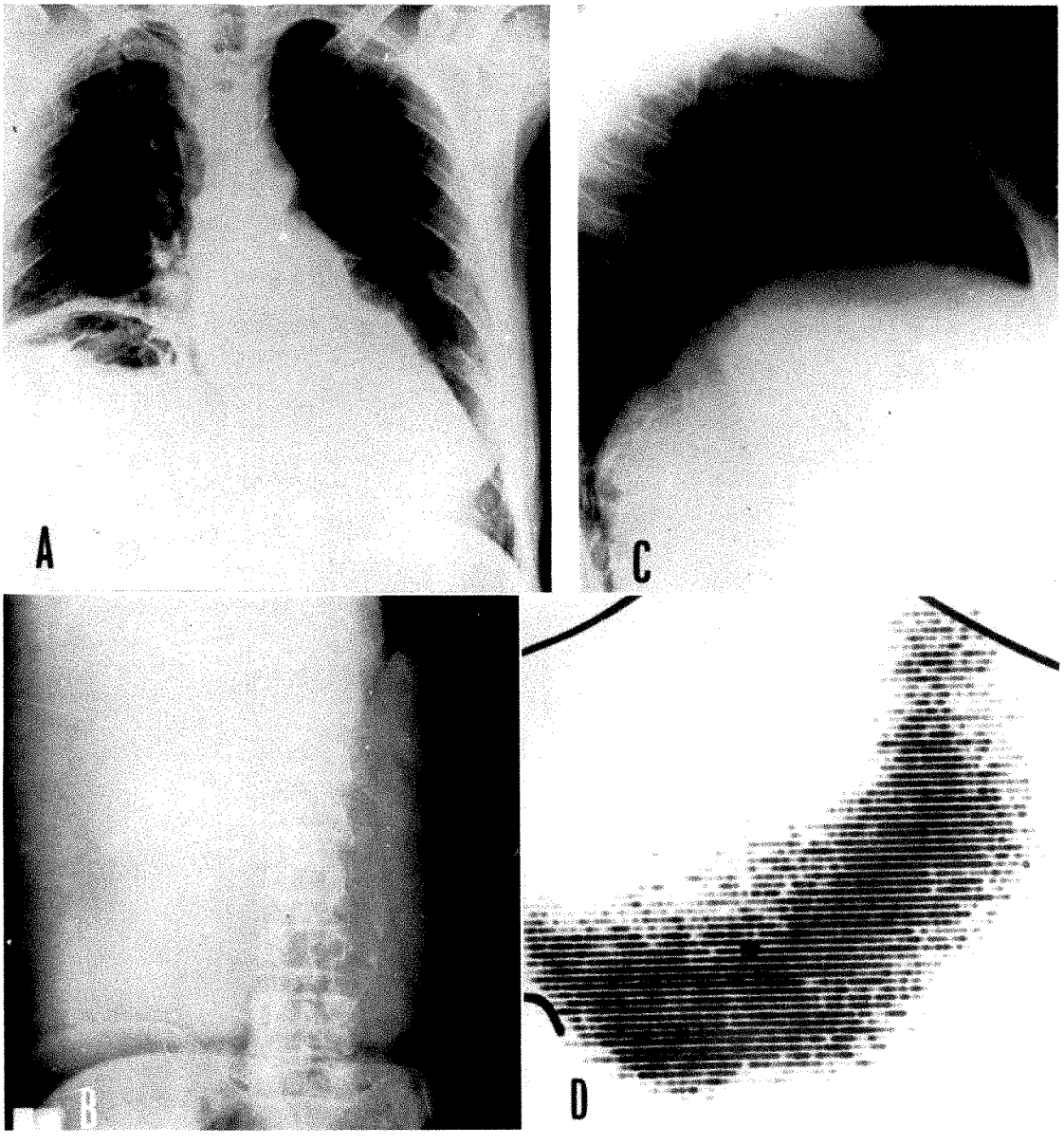



FIG. 6. Case III. (*A*, *B* and *C*) A large cyst of the liver with marked elevation of the right hemidiaphragm and marginal calcification of the cyst wall is seen. The cyst contains gas and multiple septa are apparent. (*D*) Anterior liver scan. There is gross displacement of the liver and a large hemispherical defect.

sponsible for the roentgenologic and scan abnormalities.

Case x. (Fig. 14, *A–F*). A 53 year old female had a thoracotomy for removal of a pulmonary hydatid 8 years before. Annual roentgenologic follow-up examination of the chest was regarded as normal and a liver scan was done as part of a routine follow-up study. The scan demonstrated a large posteriorly placed defect superolaterally in the right lobe. Review of the right lateral roentgenogram indicated a localized elevation of the diaphragm corresponding to the upper surface of the cyst, and liver roentgenogram demonstrated a large calcified cyst in the region of the scan defect.

Case xi. (Fig. 15, *A–D*). A 59 year old male presented with right basal pleural effusion. Hydatid elements were detected on microscopic examination of the pleural aspirate. The Casoni test was negative. The hydatid complement fixation test was positive, the eosinophil count elevated and the serum alkaline phosphatase was 17 King-Armstrong units. Roentgenography indicated elevation of the right hemidiaphragm with an area of pulmonary consolidation above it. Liver and liver-lung scans demonstrated defects at the upper lateral edge of the right lobe of the liver and between the base of the right lung and upper surface of the liver. At exploration a hydatid cyst of the right subphrenic space was present. This involved the

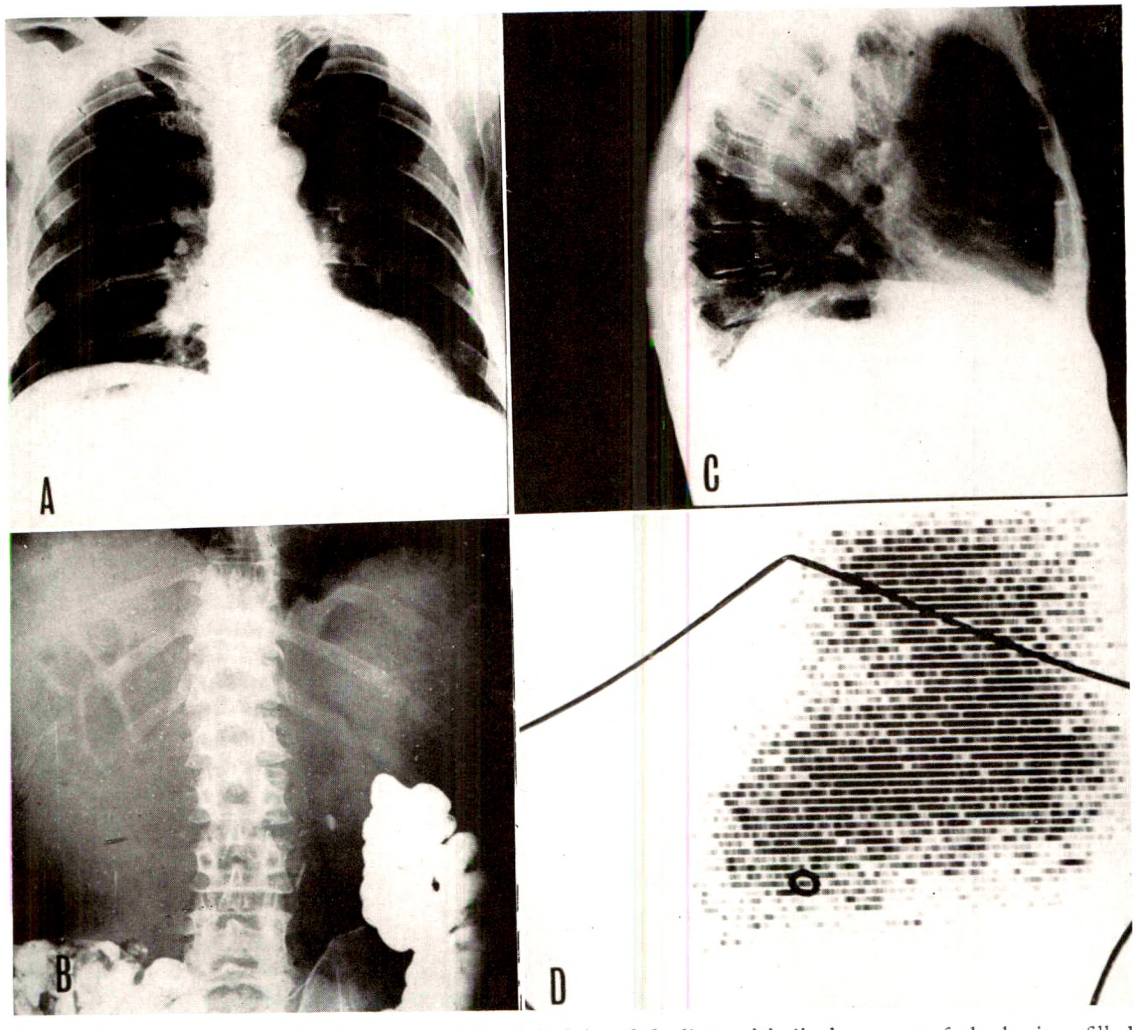



Fig. 7. Case iv. (*A*, *B* and *C*) A large cyst of the right lobe of the liver with displacement of the barium filled intestine is seen. The cyst contains some gas and numerous septa are apparent. (*D*) Anterior liver scan demonstrates marked displacement of the liver to the left.

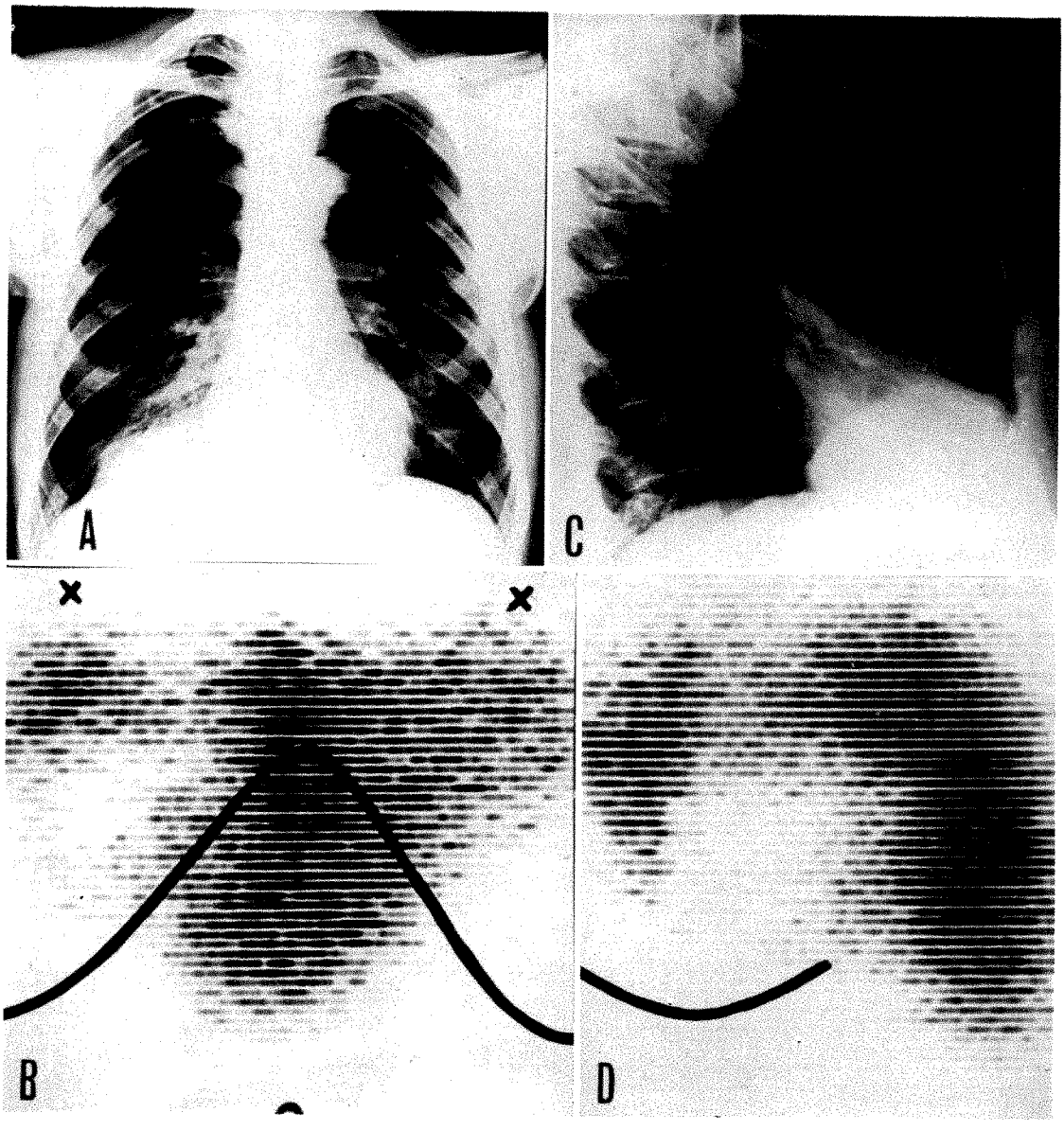



FIG. 8. Case v. (*A* and *C*) Preoperative roentgenograms showing localized elevation of the right hemidiaphragm with calcification at the periphery. (*B* and *D*) Preoperative scans. The inferolateral portion of the right lobe had been removed at a previous operation. There is a scan defect extending through to the diaphragm between the remainder of the right lobe and the hypertrophied left lobe. In the right lateral scan wide separation of the right and left lobes is evident.

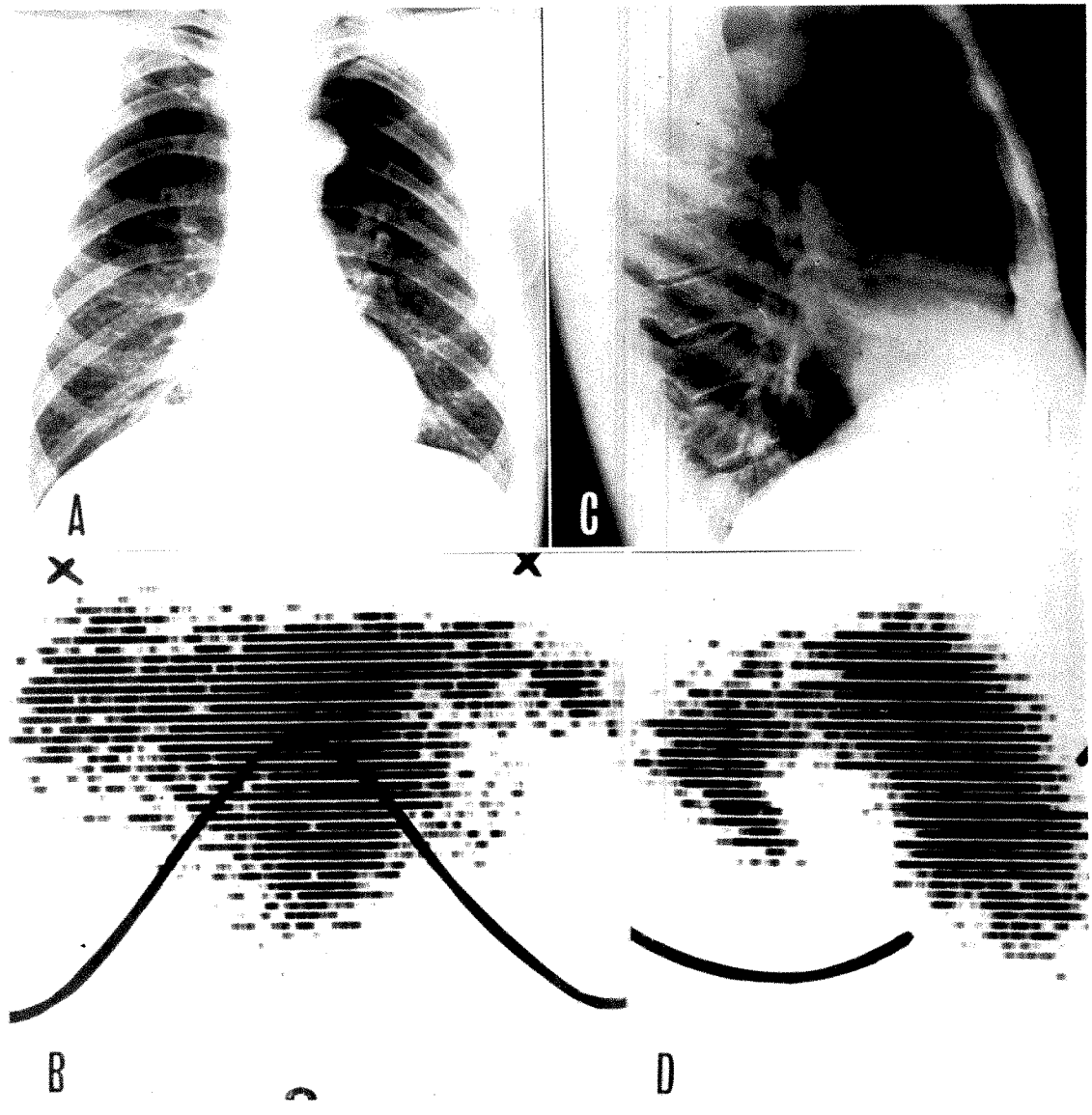



FIG. 9. Case v. (*A* and *C*) Postoperative roentgenograms. The diaphragmatic contour is now normal. Calcification is still present. (*B* and *D*) Postoperative scans. The interlobal defect is less obvious and in the lateral scan there is less separation of the right and left lobes.

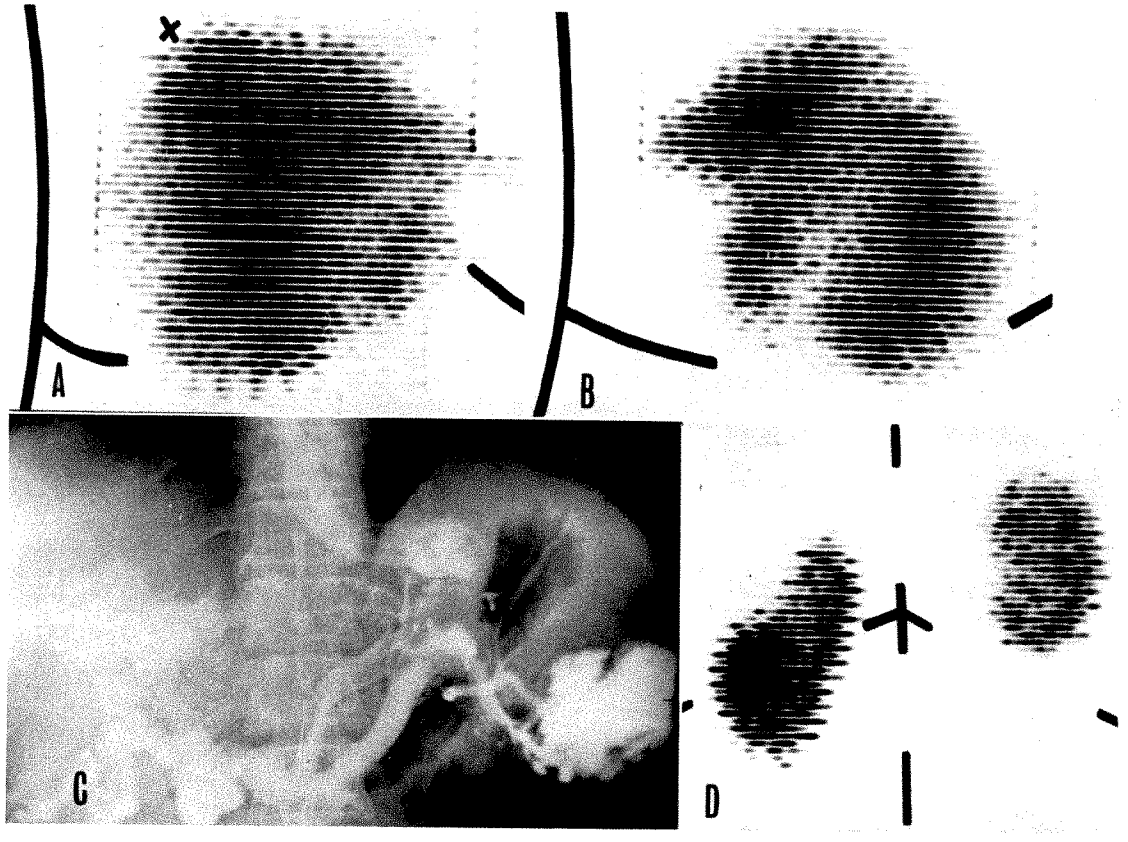



FIG. 10. Case VI. (*A* and *B*) Anterior and right lateral liver scans demonstrating multiple defects. (*C*) Splenoportogram showing displacement of intrahepatic vessels and evidence of varices. (*D*) Prone renal scan revealing a defect at the lateral edge of the right upper pole.

upper surface of the liver and extended upwards through the diaphragm.

CASE XII. (Fig. 16). A 47 year old male presented with a 24 hour history of an enlarging painful epigastric swelling. He had been operated on 12 and 4 years previously for hydatid liver disease. The Casoni and hydatid complement fixation tests were negative. Eosinophil count was not performed and the serum alkaline phosphatase was 24 King-Armstrong units. Roentgenologic examination suggested hydatid cysts in the right second rib and in the right lung. There was no scan defect corresponding to the palpable mass. At laparotomy a pedunculated hydatid cyst originating in the liver and extending into the anterior abdominal wall was present.

CASE XIII. (Fig. 17). A 20 year old male was admitted with right basal pneumonia; roentgenologic examination demonstrated a rounded opacity in the posterior segment of his right lower lobe. The Casoni test was negative but the hydatid complement fixation test was positive and eosinophilia was present. The serum alkaline phosphatase was normal. No defect was evident on the liver scan. At thoracotomy a hydatid cyst was removed from the lung and a small cyst 1.5 cm. in diameter was removed from the upper surface of the right lobe of the liver.

CASE XIV. (Fig. 18). A 7 year old female who had complained of pruritus for 6 months was found to have a palpable mass in the right hypochondrium. A vague history of contact with dogs suggested that hydatid disease of the liver be excluded. The Casoni test was negative but the hydatid complement fixation test was positive. The serum alkaline phosphatase was 58 King-Armstrong units. A roentgenogram indi-

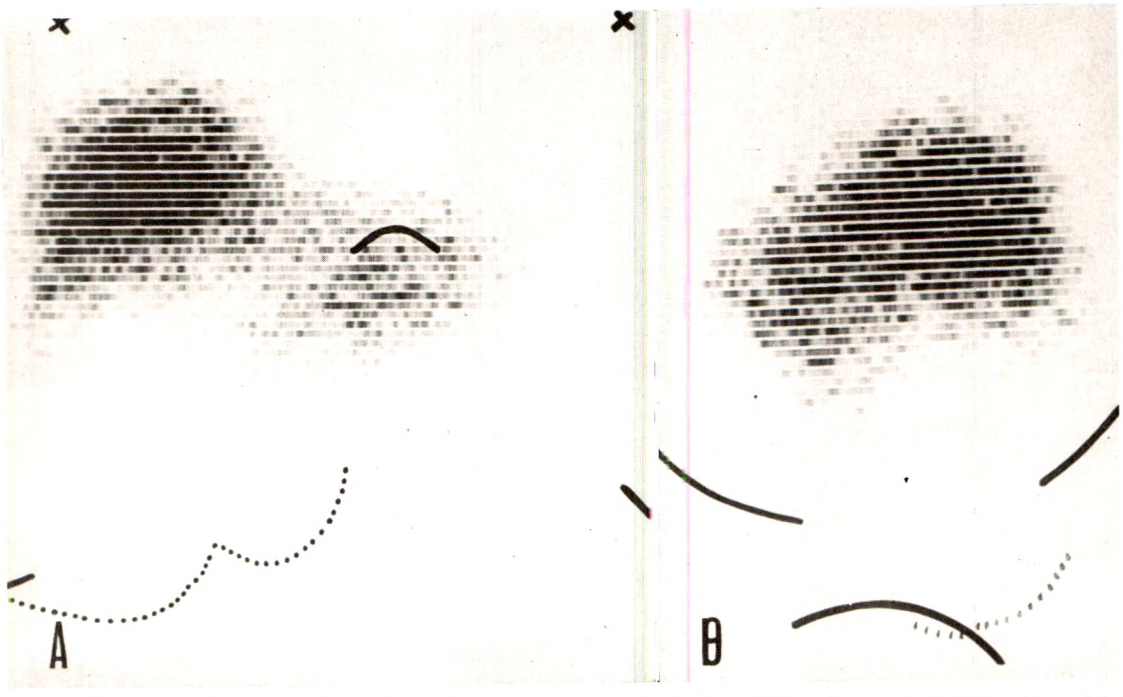



FIG. 11. Case VII. (*A*) Anterior liver scan demonstrating a large hemispherical defect at the inferior edge of the right lobe. The palpable inferior edge of the cyst is indicated by the broken line. (*B*) Right lateral liver scan indicating that the apex of the cyst is located in the mid portion of the right lobe.

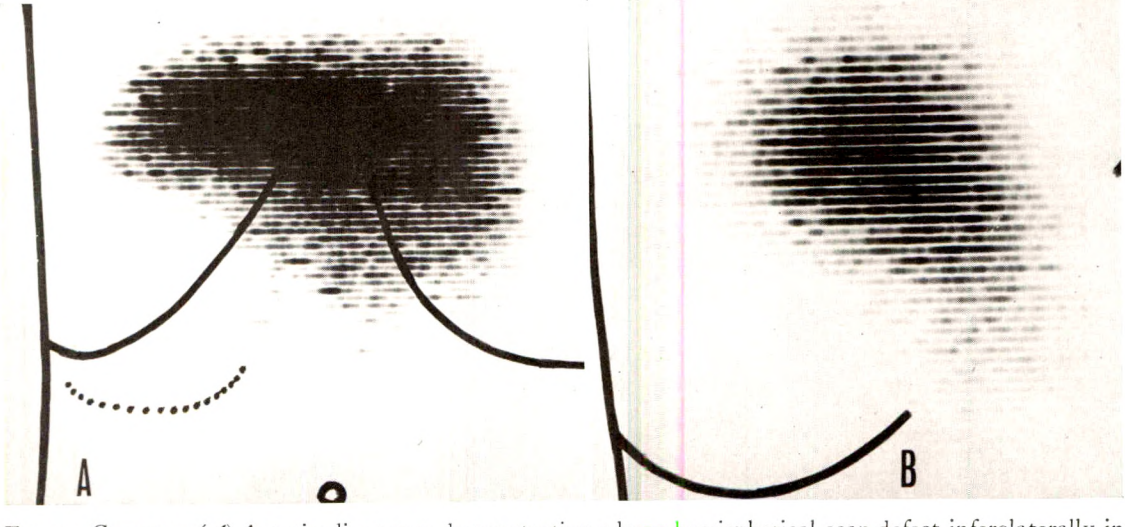



FIG. 12. Case VIII. (*A*) Anterior liver scan demonstrating a large hemispherical scan defect inferolaterally in the right lobe. The lower palpable border of the cyst is indicated. (*B*) Right lateral liver scan indicating that the cyst is located posteriorly.

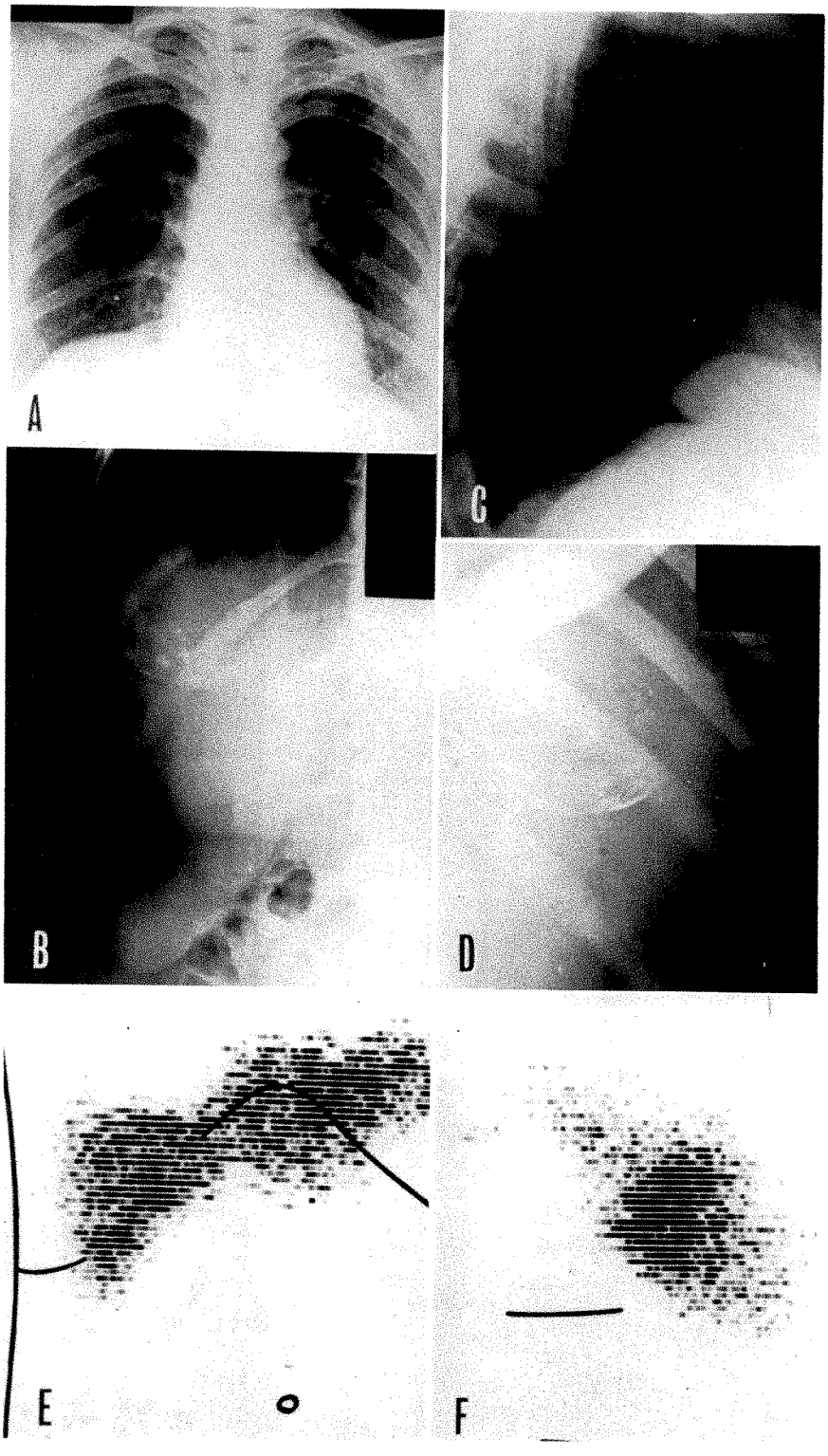



FIG. 13. Case IX. (*A* and *C*) Chest roentgenograms which were regarded as normal prior to the scan. (*B* and *D*) Liver roentgenograms showing characteristic reticular calcification. (*E* and *F*) Anterior and right lateral liver scans demonstrating extensive defects in the upper portion of the right lobe.

cated hepatomegaly without calcification. A liver scan confirmed the presence of hepatomegaly but indicated uniform isotope uptake by the palpable hepatic mass, which negated the suggested diagnosis of hydatid disease of the liver. However, laparotomy was performed and liver biopsy revealed hepatitis with possible biliary cirrhosis.

Case xv. (Fig. 19, *A* and *B*). A 37 year old female had a hydatid cyst removed from her liver 12 years previously. On three subsequent occasions daughter cysts had been resected and during the 3 years prior to the scan she had been admitted on numerous occasions complaining of persistent abdominal pain. No abnormal mass was palpable in the region of the liver. Casoni test was positive, hydatid complement fixation test was negative, eosinophil count and serum alkaline phosphatase were normal. Abdominal roentgenogram indicated calcification in the region of the left lobe of the liver. Liver scan demonstrated a deficient left lobe, but careful palpation indicated that there was no mass corresponding to this defect. This was therefore attributed to postoperative deformity. Laparotomy revealed a calcified plaque on the undersurface of the left lobe. There was no evidence of hydatid cyst.

Case xvi. (Fig. 20, *A–C*). A 65 year old male was found on routine mass-miniature roentgenography to have a round lesion in the right mid zone. No hepatic abnormality was evident on physical examination. The Casoni and hydatid complement fixation tests were not performed. The eosinophil count was normal. The serum alkaline phosphatase was not estimated. In a barium meal study an area of calcification was seen in the region of the left lobe of the liver. Liver scan was normal, no defect was evident in the region of calcification and no

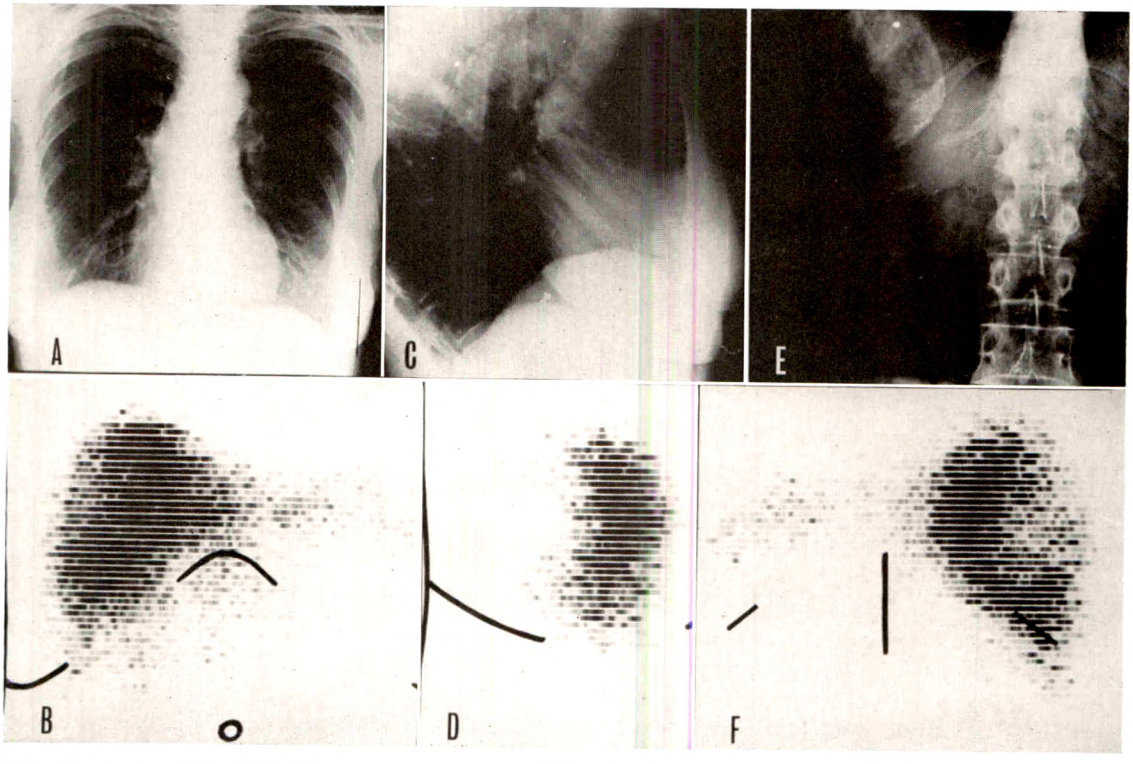



Fig. 14. Case x. (*A* and *C*) Chest roentgenograms. These were regarded as normal and no calcification was seen. After review with the liver scan, localized elevation of the right hemidiaphragm was apparent in the right lateral roentgenogram. (*E*) The calcified cyst in the right lobe of the liver is clearly seen in this roentgenogram. (*B*, *D* and *F*) Anterior, right lateral and posterior liver scans demonstrating a posteriorly placed hemispherical defect superolaterally in the right lobe. This defect is not clearly apparent in the anterior scan.

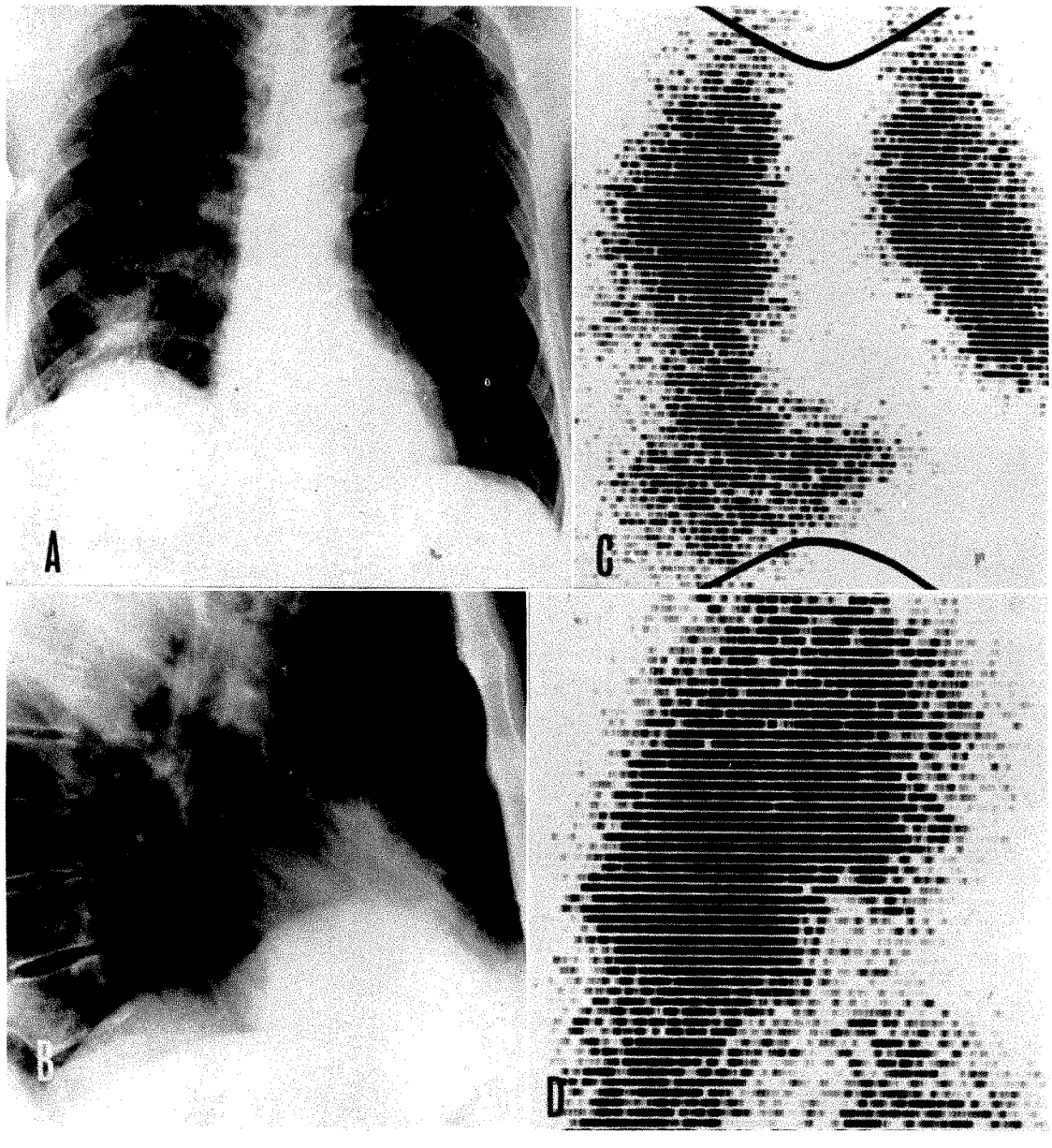



FIG. 15. Case XI. (*A* and *B*) Localized elevation of the right hemidiaphragm with an area of pulmonary consolidation above it. (*C*) Anterior liver-lung scan demonstrating a defect between the right superolateral surface of the liver and the lower border of the lung. (*D*) Right lateral liver-lung scan indicating a lung scan defect corresponding to the elevation of the right hemidiaphragm with a liver scan defect directly below it.

mass was palpable. At exploration a single hydatid cyst was found in the right lower lobe of the lung. No liver hydatid was detected.

## RESULTS

Hydatid disease of the liver was proven at laparotomy in 13 of the 16 patients; of the remaining 3, one had hepatitis, one a calcified hepatic plaque and there was no evidence of hepatic hydatid cyst in the third despite the roentgenologic demonstration of an area of calcification in the region of the left lobe of the liver.

The diagnostic accuracy of the individual investigations is summarized in Table II. In no patient with proven hydatid disease were all tests negative. The Casoni test was correct in 5 of 11 instances and in 1 case it was positive in a patient with a proven past history but no current evidence of hydatid disease. The hydatid complement fixation test was correct in 8 of 12 cases and there was 1 false positive in a patient with hepatitis (Case XIV). Eosinophilia was present in 6 of the 12 cases of hydatid disease. It was not present in any of the 3 cases without hydatid disease. The serum alkaline phosphatase was elevated in 5 of the 13 proven cases and in Case XIV.

Roentgenologic examination correctly predicted the diagnosis in 7 patients initially and in a further 2 when the roentgenologic data were reviewed after the scanning (Cases I and II). The roentgenologic abnormalities included: calcification (6), diaphragmatic elevation (5), pneumohydrocyst (2) and hepatomegaly (2). Splenoportography was performed in 1 patient (Case VI) and the diagnosis based on this was correct. Oral and intravenous cholangiographies were performed in 1 patient (Case VII); these were not helpful in a patient with a large cyst involving the inferior edge of the right lobe. Roentgenologic opinion incorrectly favored a diagnosis of hydatid cyst in 3 patients (Cases XIV, XV, XVI); in 2 of these this was based on the presence of calcification and in 1 on hepatomegaly.



FIG. 16. Case XII. The broken line indicates the edge of a palpable pedunculated hydatid cyst which originated in the liver and extended into the anterior abdominal wall.

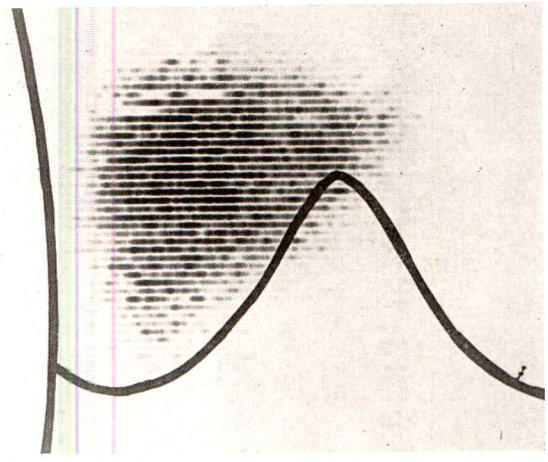


FIG. 17. Case XIII. Normal anterior liver scan. The defects superolaterally in the right lobe are due to instrumental faults.

Radioisotopic assessment was correct in 11 of the 13 proven cases. The scan abnormalities included: hemispherical defects (7), hepatic displacement (6), multiple defects (1), lobar separation and delayed excretion of $I^{131}$ rose bengal (1). In the 2 cases in which the scan assessment was incorrect one was a pedunculated cyst (Case XII) situated principally in the anterior abdominal wall, the second (Case XIII) was a small cyst 1.5 cm. in diameter located on the surface of the liver. In the 3 patients with no evidence of hydatid disease at

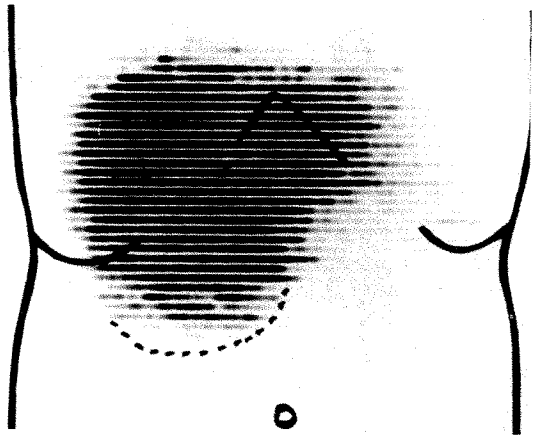


FIG. 18. Case XIV. Anterior liver scan. Isotope uptake was uniform throughout the liver including the palpable mass which is indicated by the broken line.

operation (Cases XIV, XV, XVI) the scan diagnosis was correct.

## DISCUSSION

In the past, roentgenologic examination of the liver has been the principal method for diagnosis of hydatid disease of this organ.[4,5,9,10,12,14,17,24,25,26,27] Moderate hepatomegaly is difficult to diagnose on a plain roentgenogram but the demonstration of marked hepatomegaly may be facilitated by contrast studies indicating displacement of adjacent viscera (Case IV, Fig. 7, *A–D*). The roentgenogram may not distinguish between generalized hepatomegaly, hepatomegaly due to local replacement, or displacement by lesions of similar density (Case I, Fig. 2, *A–D*). Hypertrophy of uninvolved liver is common in liver hydatid disease[8,9] and this makes correct roentgenologic assessment of hepatomegaly more difficult.

The incidence of calcification of liver hydatids is poorly documented; in Czerniak *et al.'s* series[7,8] of 65 cases, calcification in the hepatic region was present in 35 per cent. In our series it was evident in 6 instances.

Hepatic calcification may uncommonly occur with hepatoma and in hepatic metastases from carcinoma of the colon.[14]

Calcification of liver hydatids may appear as a thin rim, over portion or all of the surface of a cyst (Case III, Fig. 6, *A–D*) or the cyst may be extensively calcified in a reticular pattern (Case IX, Fig. 13, *A–F*; Case X, Fig. 14, *A–F*). In Australia the presence of this reticular calcification is the most specific roentgenologic sign and is almost diagnostic of hydatid disease. If calcified cysts are demonstrated, additional noncalcified cysts may be missed (Case IX, Fig. 13, *B–F*) and the extent of calcification is not always a reliable guide to size or presence of a cyst (Case XVI, Fig. 20, *B–C*). The decision to operate on asymptomatic calcified cysts is difficult to make. This may be clarified by the addition of scanning.

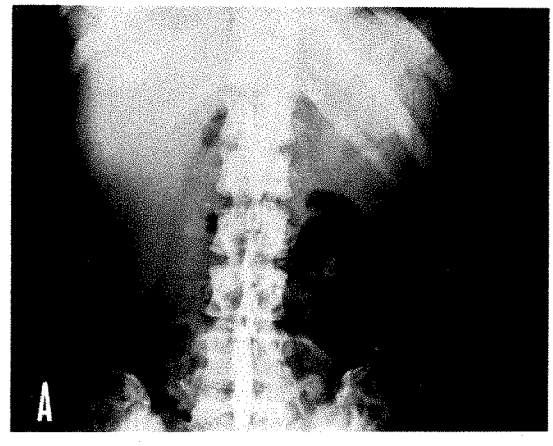






FIG. 19. Case XV. (*A*) Calcification in the left lobe of the liver. This calcified plaque remained unchanged following operation in 1962. (*B*) Anterior scan indicating a deficient left lobe and a defect at the medial edge of the right lobe. There was no palpable mass corresponding to the scan defects.

In Case x (Fig. 14, *A–F*) the cyst was more extensive than suggested by roentgenologic examination and in Case ix (Fig. 13, *A–F*) an additional noncalcified cyst was present adjacent to the calcified one evident on the roentgenogram. Conversely, the presence of calcification without a corresponding scan abnormality may provide reasonable evidence against operation (Case xv, Fig. 19, *A* and *B*; Case xvi, Fig. 20, *A–C*). Incomplete removal of a calcified cyst may cause difficulties in assessment at subsequent roentgenologic examination (Case v, Fig. 8, *A–D*, and 9, *A–D*; Case xv, Fig. 19, *A* and *B*).

Roentgenologic examination of the liver must include accurately exposed views of this area. One patient (Case x, Fig. 14, *A–F*) had posteroanterior and right lateral chest roentgenograms annually for 7 years after removal of a lung hydatid. These were regarded as normal; roentgenograms of the liver made after liver scanning had indicated a large defect, demonstrated a large cyst with characteristic reticular calcification.

Generalized or localized diaphragmatic elevation may occur with liver hydatids (Case iii, Fig. 6, *A–D*; Case v, Fig. 8, *A–D*). Localized elevation is an important sign but this may be easily overlooked (Case i, Fig. 2, *A* and *C*; Case ii, Fig. 4, *A–D*; Case ix, Fig. 13, *A–F*; Case x, Fig. 14, *A–F*). Generalized and localized right hemidiaphragmatic elevations are difficult to evaluate on roentgenograms alone. A pneumohydrocyst of the liver (Case iii, Fig. 6, *A–D*; Case iv, Fig. 7, *A–D*) may occur in hydatid disease and the membrane debris or daughter cysts may be visible. The clinical and roentgenologic features of rupture of liver hydatids have been well described[17,29] and the roentgenologic appearances seen in intrapulmonary rupture of liver hydatids have been adequately recorded.[7,17,27,29]

Roentgenologic assessment of the right hemidiaphragm may be considerably enhanced by liver scanning[6,11,22,28] and minor variations may assume a different significance when reviewed in conjunction with the scan (Cases i, ii, ix, x). In selected cases liver-lung scanning[20] may be useful (Case xi).

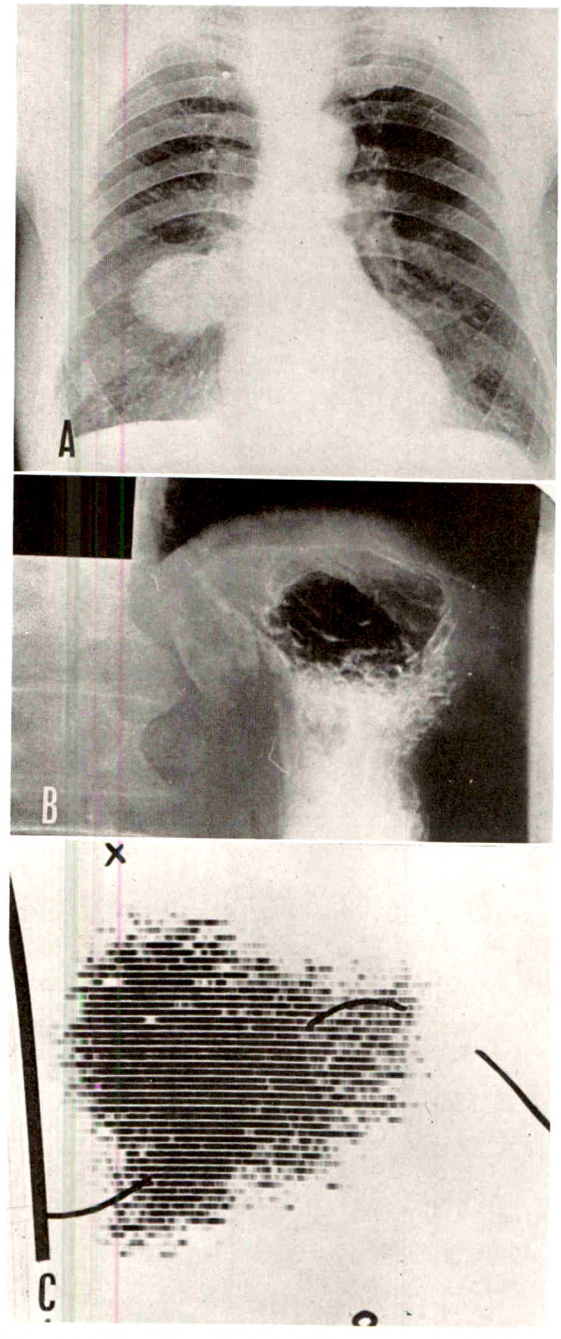



Fig. 20. Case xvi. (*A*) Hydatid cyst in the lower lobe of the right lung. (*B*) Calcification in the region of the left lobe of the liver. (*C*) Normal anterior liver scan.

Surgical removal of liver hydatid cysts

TABLE II

ANALYSIS OF INVESTIGATIONS OF PATIENTS WITH OPERATIVE EVIDENCE OF HYDATID DISEASE OF LIVER

| Case No. | Casoni Test | Hydatid Complement Fixation | Eosinophilia | Serum Alkaline Phosphatase | Roentgenologic Assessment | Radioisotopic Assessment |
|---|---|---|---|---|---|---|
| I | N.P. | − | − | − | − (+) | + |
| II | + | + | − | − | − (+) | + |
| III | − | N.P. | − | + | + | + |
| IV | − | + | + | + | + | + |
| V | N.P. | + | − | + | + | + |
| VI | + | + | + | + | + | + |
| VII | + | + | − | − | − | + |
| VIII | − | − | + | − | − | + |
| IX | + | − | + | − | (+) | + |
| X | + | + | − | − | (+) | + |
| XI | − | + | + | − | + | + |
| XII | − | − | N.P. | + | − | − |
| XIII | − | + | + | − | − | − |
| *Accuracy* | 5/11 | 8/12 | 6/12 | 5/13 | 7/13, (9)/13 | 11/13 |
| | ANALYSIS OF INVESTIGATIONS OF PATIENTS WITH NO EVIDENCE OF HYDATID DISEASE OF LIVER AT OPERATION | | | | | |
| XIV | − | + | − | + | + | − |
| XV | + | − | − | − | + | − |
| XVI | N.P. | N.P. | − | N.P. | + | − |
| *Accuracy* | 1/2 | 1/2 | 3/3 | 1/2 | 0/3 | 3/3 |

− Investigation negative or normal.
+ Investigation abnormal and used as evidence in favor of a diagnosis of hydatid disease of the liver.
N.P. Investigation not performed.
( ) Indicates that the roentgenologic evidence was reviewed after the scanning.

may leave defects indistinguishable from a recurrent cyst[1,3,7] (Case XV, Fig. 19, *A* and *B*; Case VIII, Fig. 12, *A* and *B*) and careful surgical records are vital to allow correct interpretation of postoperative scans. Generally, scan defects are nonspecific and the possible causes of a scan defect can only be ascertained by examination of the clinical, laboratory and roentgenologic data and more particularly by correlation of palpatory findings (Case VIII, Fig. 12, *A* and *B*; Case XV, Fig. 19, *A* and *B*). A hemispherical defect is more likely to be caused by a cyst as opposed to neoplastic defects which are more commonly spherical. Occasionally liver hydatid cysts may be pedunculated and cause little distortion of the scan (Case XII, Fig. 16). Liver scanning is important in the exclusion of hydatid disease; uniform isotope uptake by a hepatic mass is strong evidence against a diagnosis of hydatid cyst (Case XIV, Fig. 18).

Cysts smaller than 4 cm. in diameter may be missed by scanning (Case XIII, Fig. 17) but usually single lesions smaller than this are of no immediate clinical concern. This degree of resolution compares favorably with that obtained with selective arteriography[23,24] and is superior to that achieved with splenoportography.[8]

Correct interpretation of liver scans demands a knowledge of the normal variations of the scan.[20,22] Care must be taken in assessing the significance of peripheral defects. There may be a diminution in radioactivity in the region of the porta hepatis[20] (Case II, Fig. 5, *A–D*), and in livers that have been enlarged or ptosed for a sufficient period of time a diminution in radioactivity may be observed beneath the

right costal margin[22] (Case II, Fig. 5, *A–D*). This is known as the costal impression.[20] If hydatid disease of the liver is causing biliary tract obstruction (Case V, Fig. 8, *A–D*, and 9, *A–D*) studies of the rate of biliary excretion of $I^{131}$ rose bengal may be useful.[20]

Diagnostic tests for hydatid disease based on immunologic phenomena have varying degrees of accuracy.[18] Eosinophilia frequently occurs but is not specific. The accuracy of the Casoni intradermal test varies from 60 to 90 per cent in reported series.[18] This variation appears to be related to degrees of purification of the antigen. False positives may occur with infection with Taenia and several other worms, and positivity may persist after removal of the cyst. Suppurating cysts may cause a negative response and the Casoni test is of no value in assessing viability of a cyst. The hydatid complement fixation is an accurate test that rapidly becomes negative after complete removal but it may be negative in sterile or completely calcified cysts. False positives are rare but one did occur in this series (Case XIV). Although the serum alkaline phosphatase is not specific, in this series its accuracy parallelled the Casoni test and in one patient (Case XII) it was the only abnormal test.

The most accurate diagnosis of liver hydatid disease will be achieved with the combined use of immunologic, biochemical, roentgenologic and radioisotopic investigations. Ideally, roentgenologic and radioisotopic evidence should be examined together and jointly interpreted.

## SUMMARY

Liver scanning is a simple, safe and accurate method for improving the diagnostic accuracy and preoperative assessment of hydatid disease of the liver. Scan defects are nonspecific and for accurate interpretation the scan must be carefully correlated with the clinical, laboratory and roentgenologic data. The liver scan is particularly useful in elucidating right hemidiaphragmatic abnormalities. The simplicity and lack of toxicity of this investigation make it an ideal test for assessment of recurrence, but operative details must be carefully recorded to allow correct interpretation of postoperative scans. A normal anterior and right lateral liver scan is usually convincing evidence against a diagnosis of hydatid disease of the liver.

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VOL. 101, NO. 3

# THE VALUE OF LIVER SCANNING IN THE DIAGNOSIS OF POLYCYSTIC DISEASE OF THE LIVER*

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POLYCYSTIC liver is a relatively uncommon congenital disease in which the liver is filled with thin-walled cysts of different size containing clear fluid. Often the whole liver is affected; sometimes only one lobe, mostly the right one.[18] The size of the cysts varies from microscopic to large ones containing several liters of fluid.[4] The cysts grow slowly and the largest are seen in elderly patients.[13]

In 1955, Comfort *et al.*[4] reported a series of 24 cases of polycystic liver disease, and in 1957 Henson *et al.*[12] reported 29 cases. In all, about 300 cases have been published,[16] mostly reports of single cases.[8,10] The frequency of this disease in an autopsy material was only 0.15 per cent.[13]

Despite its size, the polycystic liver causes rather few symptoms. The diagnosis is usually made during operation or at autopsy;[11] very seldom otherwise.[10]

The purpose of this investigation is to clarify the value of radioisotope scanning for diagnosing polycystic disease of the liver.

## METHOD OF LIVER SCANNING

Radioactive colloidal gold ($Au^{198}$) is used by intravenous injection. Over 90 per cent is taken up by the reticuloendothelial cells and Kuppfer's cells of the liver in about 10 to 20 minutes, and the gold which has a physical half life of 2.7 days decays there. The dose of $Au^{198}$ is usually 100 $\mu$c, which yields about 2,000 to 3,000 counts per minute and permits a good resolution for scanning. Recording is performed with a Nuclear Chicago Scanner (Type 1700 +1700P-double-scanner) with photoimpulses on film. We employ 2 collimators, one with 19 holes and the other with 37 holes, and recording speeds of 60 cm./min. and 75 cm./min., respectively. The pulse rate and the contrast are chosen empirically depending on the maximal number of impulses. The scannings are made only in the anterior position.

## CLINICAL MATERIAL

Our series consisted of 26 cases of polycystic disease of the liver—20 women and 6 men. The age ranged from 28 to 74 years, with a mean of 49 years. In addition to a detailed clinical examination, including laboratory tests of liver function and roentgenograms of the thorax and abdomen, radioisotope scanning of the liver was performed on each patient.

The diagnosis of polycystic liver was arrived at as follows:

| | |
|---|---|
| At cholecystectomy | 13 |
| At explorative laparotomy | 5 |
| From liver puncture | 3 |
| At operation for abdominal hernia | 1 |
| At appendectomy | 1 |
| By liver scanning | 3 |
| Total | 26 cases |

The diagnosis was established earlier and the patients were followed before the scanning for:

| | |
|---|---|
| Over 10 years in | 4 |
| 5 to 10 years in | 8 |
| 1 to 5 years in | 10 |
| Under 1 year in | 1 |
| Diagnosis first made by the scanning in | 3 |
| Total | 26 cases |

Although polycystic liver is congenital, it is usually first diagnosed after the age of 40 years and then mostly by chance. The patients often feel discomfort in the upper abdomen or under the right costal margin.

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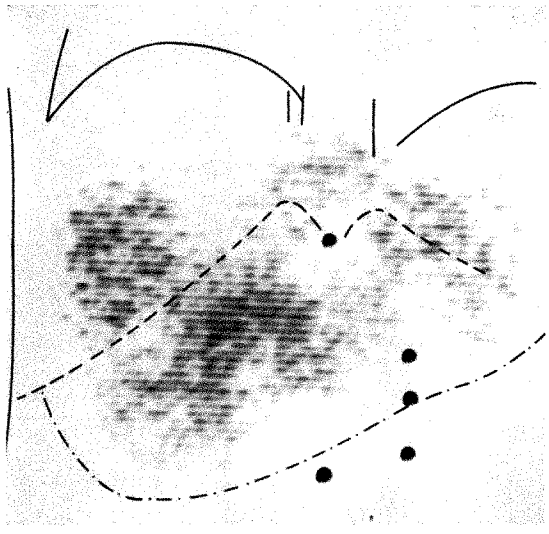


FIG. 1. In a man aged 40 years, the liver which extended to the umbilicus contained numerous cysts of varying sizes. The largest was the size of a fist. (Liver scanning with colloidal $Au^{198}$.)

Twenty-one of our 26 patients (80 per cent) had this discomfort for years, some even for decades, but were otherwise in good condition. Nine had colic-like pains in the upper abdomen. The pain can be caused by rupture of the cyst, or hemorrhage or infection in the cyst. Seven patients had noticed a palpable mass in the abdomen, and 2 observed disappearing nodes. Two had no discomfort at all, and 4 suffered from impaired working capacity because of a large liver.

The laboratory findings were normal.

The roentgen examinations gave little direct information, but 6 patients had impressions on the neighboring organs, 3 had round calcified shadows in the abdomen, 6 had an eventrated right hemidiaphragm with a lobulated contour, and 3 had only an eventrated diaphragm. In 1 case the roentgen diagnosis was made by splenoportography, and in 1 case the cyst was punctured and filled with contrast material and was thus visualized roentgenographically.

### RESULTS OF LIVER SCANNING

The scanning was quite useful for diagnosing polycystic liver.

The results of the scanning were as follows:

| | |
|---|---|
| Nearly normal | 5 |
| Slight changes | 3 |
| Distinct changes | 9 |
| Marked changes | 9 |
| Total | 26 cases |

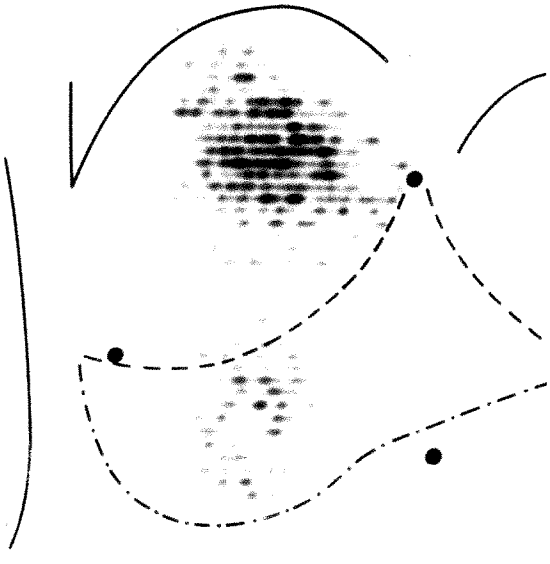


FIG. 2. The liver of a married woman aged 80 years was found at laparotomy 14 years earlier to contain many almost egg-sized cysts. (Liver scanning with colloidal $Au^{198}$.)

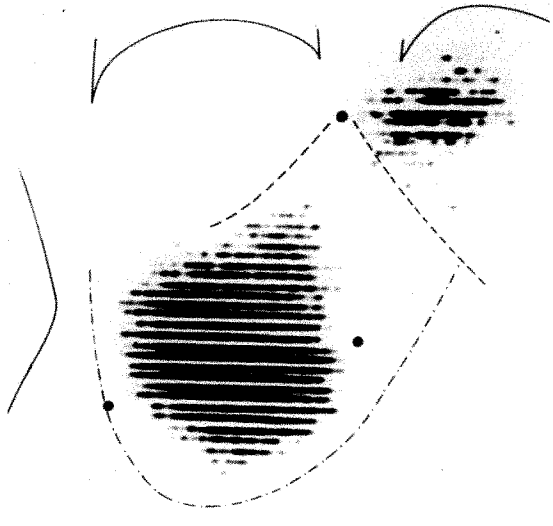


FIG. 3. A woman aged 68 years had been found 13 years earlier to have a large liver, which now extends considerably below the umbilicus. The diagnosis of polycystic liver was confirmed by liver biopsy and splenoportography. (Liver scanning with colloidal $Au^{198}$.)

The size of the largest cyst in each patient of the series was:

| | |
|---|---|
| Under 5 cm. | 10 |
| 5 to 9 cm. | 8 |
| Over 10 cm. | 5 |
| Not known | 3 |
| Total | 26 cases |

The largest cyst contained over 4 liters of clear fluid.

The changes in 7 cases are illustrated in Figures 1–7.

The 5 cases which gave a negative liver scan were:

Case 1. Small cysts, diameter 1/2–2 cm.
Case 2. Several cysts, diameter up to 2 cm.
Case 3. Several cysts, diameter up to 2 cm.
Case 4. Some small cysts, diameter up to 3 cm.
Case 5. One cyst of 3 cm., many with a diameter of 1/2 cm.

We noted that in all the negative cases the cysts were less than 3 cm. in diameter.

## DISCUSSION

As space-occupying lesions, the cysts are well suited for scanning examination. This permits identification of: the size, shape, contour, and position of the liver; the presence of functioning liver tissue and nonfunctioning cysts; and the size, location and number of the cysts.

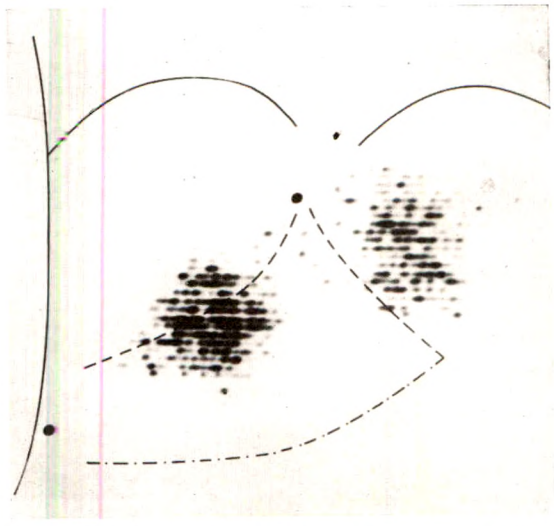


Fig. 5. Polycystic liver was found 2 years earlier in a woman aged 47 years, during an operation for hernia. The largest cysts were egg-sized. (Liver scanning with colloidal $Au^{198}$.)

The radiation dose to the liver when using 100 $\mu$c of colloidal $Au^{198}$ intravenously is about 4 rads.[19] The dose is moderate, not higher than the patient receives in major roentgen examinations.[3] Other colloids

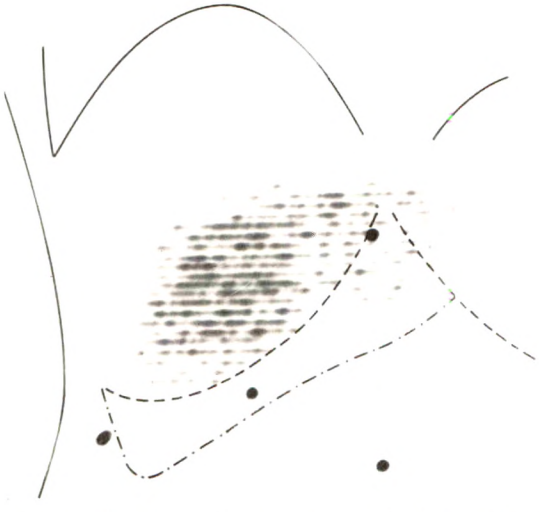


Fig. 4. The liver of a patient aged 58 years contained many cysts, the largest of which were 1–1.5 cm. in diameter. (Liver scanning with colloidal $Au^{198}$.)

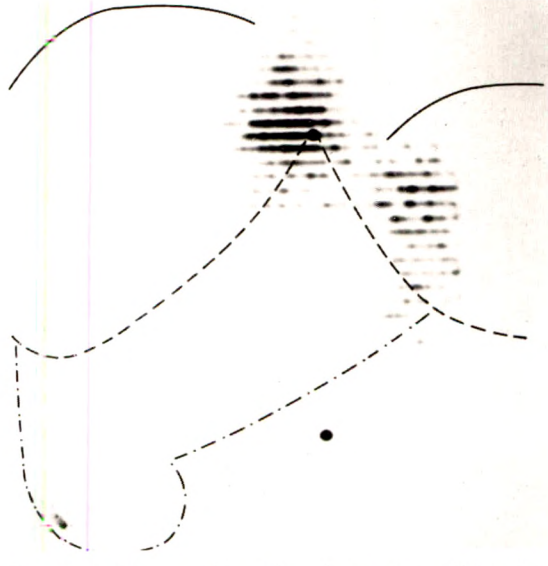


Fig. 6. A 66 year old patient in good condition had had for 7 years a large tuberous liver. Diagnosis of polycystic liver had been confirmed by liver puncture. (Liver scanning with colloidal $Au^{198}$.)

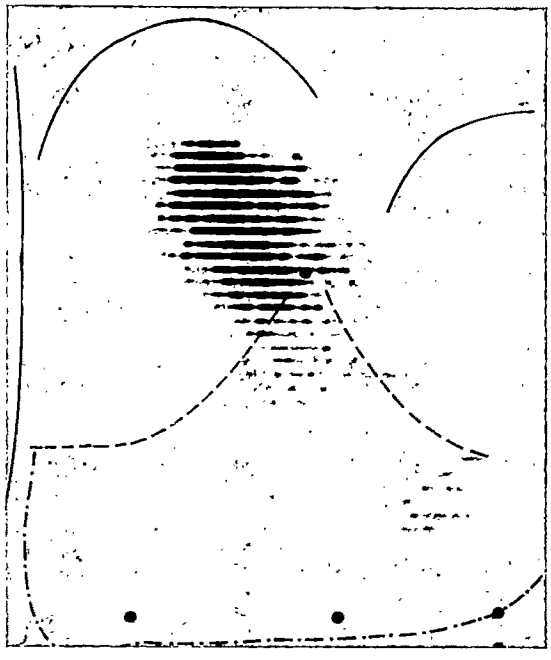


FIG. 7. At laparotomy the liver of a female patient aged 51 years was found to contain many cysts, the largest of which were egg-sized. (Liver scanning with colloidal $Au^{198}$.)

used, such as $Ag^{111}$,[14] $Tc^{99m}$,[1,20] and $I^{131}$ macroalbumin aggregates[1] have not proved superior to $Au^{198}$. Another tracer used for liver scanning is $I^{131}$ rose bengal, taken up by the parenchymal or polygonal cells of the liver. The disadvantage or difficulty of this tracer is its excretion into the bile and via this route into the duodenum.[21] It is, therefore, more suitable for studying liver function and in the differential diagnosis of jaundice.[6,9,19]

Diagnosis of polycystic liver disease is not easy. Despite the size of the liver, the patient has rather few symptoms. Often, discomfort in the upper abdomen is the only symptom—in 80 per cent of our material. The patients are mostly in surprisingly good condition in spite of the large palpable liver. It is very seldom that liver insufficiency develops.[6,8] Deutsch, in 1953, reported a patient who first had acute cholangitis with abscess formation and at last liver coma.[7] Bleeding esophageal varices which have originated as a result of elevated portal blood pressure have also been described in connection with polycystic liver.[2]

The laboratory findings are of no value for diagnosis. They are mostly normal, including liver function tests.

Roentgen examinations may give considerable indirect information about polycystic liver, but they are of no aid to final diagnosis. The dome of the diaphragm may be raised and plain roentgenograms of the abdomen may occasionally, though rarely, reveal cystic calcification shadows.[17] The possibility of polycystic liver can be suspected also from impressions found in examining other upper abdominal organs.

Liver puncture is a good procedure for the diagnosis of polycystic liver. Contrast medium can be injected into the cyst so that it can be visualized roentgenologically.[17] Laparoscopy is an excellent aid in establishing the diagnosis.[11,15] Splenoportography also helped the present authors to verify polycystic liver in 1 case. The last mentioned examinations are rarely necessary and place a fairly great strain on the patient.

Polycystic liver proved to be a gratifying target for radioactive scanning. There were only 5 cases in which no changes were noted. These patients had small cysts dispersed throughout the liver. Even cysts of 3 cm. in diameter were not seen on the scan. It does seem, in fact, that it is possible to diagnose only cysts of 3 cm. or more in diameter by ordinary single plane scanning. A positive result was obtained in 21 out of 26 patients (80 per cent). Hence, it may be said that, combined with the clinical picture, laboratory and roentgenologic findings, liver scanning is an excellent aid in the diagnosis of polycystic liver. Only rarely must we resort to measures that involve a greater strain on the patient, such as laparoscopy, splenoportography and laparotomy.

If we have under examination a patient in good condition who has had an enlarged tuberous liver for years, in whom the liver function tests are normal, the roentgen examinations yield no definite findings,

and the liver scanning reveals manifest defects in the liver parenchyma, we may consider the diagnosis of polycystic liver.

SUMMARY

Liver scanning with 100 μc of colloidal Au $^{198}$ was carried out in 26 patients with polycystic liver. The examination gave very useful information about the size, location and number of the cysts. A definite pathologic condition was noted in 80 per cent of the patients. Only cysts which were small (under 3 cm.) and scattered throughout the liver escaped detection.

A patient with clinically established polycystic liver is in very good condition. The large liver may be tuberous under the costal margin on palpation. It has grown gradually during a period of years and causes no symptoms other than a feeling of weight and fullness in the epigastrium. Despite the very great size of the liver, the laboratory tests are usually normal. Roentgen examination also gives only indirect support to the diagnosis of the disease. The dome of the diaphragm may be lobulated and elevated; calcification shadows caused by the cysts may sometimes be seen; and displacement of the adjacent organs may be demonstrated.

By combining liver scanning with these examinations we seldom need to resort to investigations which expose the patient to a greater risk and a heavier strain, such as laparoscopy or laparotomy, when looking for a polycystic liver.

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# ZINC 69m CHLORIDE, A NEW LIVER SCANNING AGENT

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RADIOACTIVE zinc has been used extensively to study zinc metabolism in the mammal.[1,4,5,6,7,8,10] Much of the experimental investigation has been done using zinc 65 with a physical half life of 245 days and a gamma with a 1.119 mev. energy. This material has been found to concentrate almost preferentially in the livers of the animals studied. When zinc 69m with its 13.8 hour physical half life and 0.438 mev. gamma became available, it was used to extend these studies to humans.[3] The high concentration of the zinc 69m in the liver prompted evaluation of this radioisotope as a scanning agent.

## MATERIALS AND METHODS

Six men ranging in age from 54 to 83 years were each injected with 200 $\mu$c of $Zn^{69m}$ chloride. Each was diagnosed as having prostatic disease. The results of the prostatic scans will be presented at another time. The concentration of radioactivity in the liver was recorded and compared to that of a "neutral" abdominal probe with the aid of a dual probe uptake system. The liver of each patient was scanned, in addition to the prostate, one hour after injection. All scans were made with a commercial 5×2 inch crystal scanner.*

The $Zn^{69m}$ was produced by neutron irradiation of 99.3 per cent isotopically enriched $Zn^{68}O$ targets in the Oak Ridge Research Reactor. The average specific activity produced was 2.1 mc/mg. zinc and contained approximately 0.02 per cent $Zn^{65}$ impurity produced by the $Zn^{64}$ (n, $\gamma$) $Zn^{65}$ reaction.

* Picker Magnascanner V.

## RESULTS

When radioactive zinc chloride was injected intravenously in man, it had about a 5 minute half life in the circulation (Fig. 1). A detector probe over the liver revealed a prompt rise in radioactivity in that organ (Fig. 2). Scintiscanning at 1 hour post-injection produced technically good scans of the liver as is seen in Figure 3.

## DISCUSSION

Radioactive zinc has been used extensively as zinc 65 chloride in animal studies.[1,4,5,6,10] In addition, a few human studies have been performed with this isotope.[7,8] Animal studies have shown it to have an unsuitable photon emission for scintiscanning with currently available scanning devices.[4,9]

Zinc 69m chloride became available for investigation this year. As would be expected, zinc 69m and zinc 65 chloride had similar distribution in mammalian tissue. Following intravenous injection, zinc 69m concentrated promptly in the liver and hepatic scintiscan was obtained with ease.

In dosage of 200 $\mu$c, zinc 69m provides 0.13 rad to the liver. A similar amount of gold 198 gives 10 rad to the liver.[2]

0.583 da. = $T_{eff}$
Dose = 200 $\mu$c
Tissue = liver—1,700 gm. with a 10 cm. radius—$\overline{gm.}$ = 69
0.0346 = gamma constant
2.3 = r/mc/hr./im
f 1/2 = fraction of dose in whole blood distributed to organ of interest = 0.35
Calculation = 200/1,700 × 0.583 (0.0346 × 69 × 2.3) 0.35 = 0.131 rads

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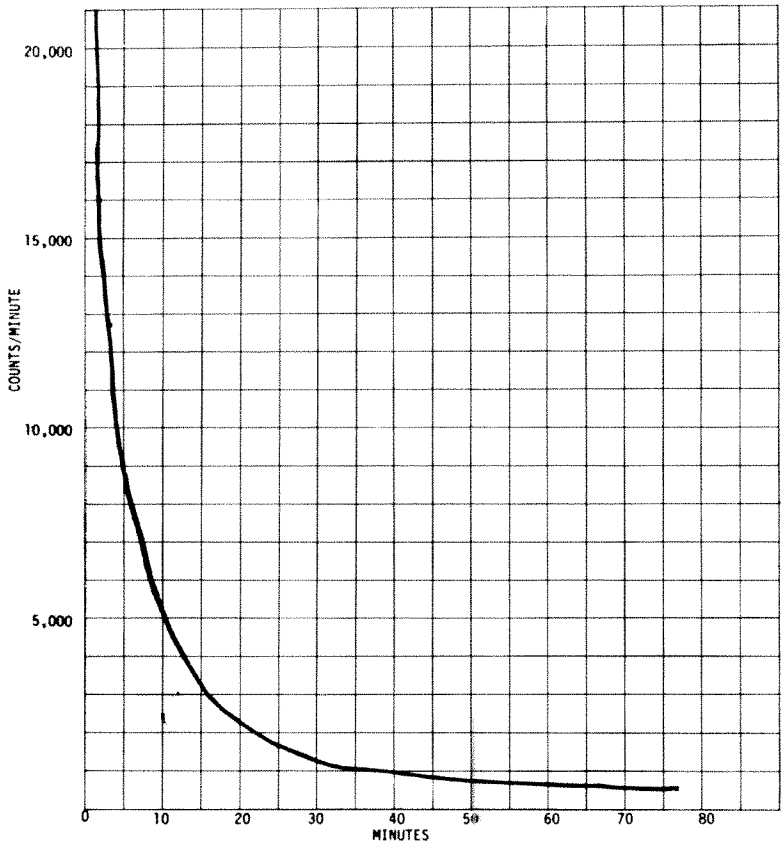



FIG. 1. The blood clearance curve of zinc 69m chloride, average of 5 patients, plotted in net counts per minute against time in minutes.

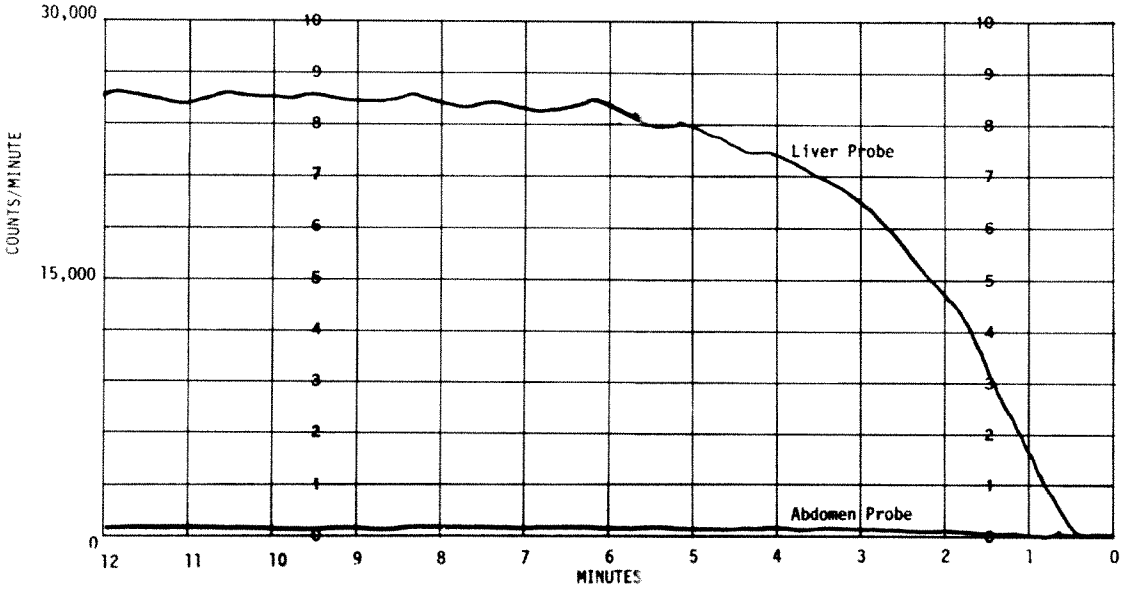



FIG. 2. With one probe of a dual scintillation probe apparatus placed over the liver, the other probe over the abdomen, zinc 69m chloride was injected intravascularly and hepatic and abdominal uptakes were recorded from time of injection for 12 minutes.

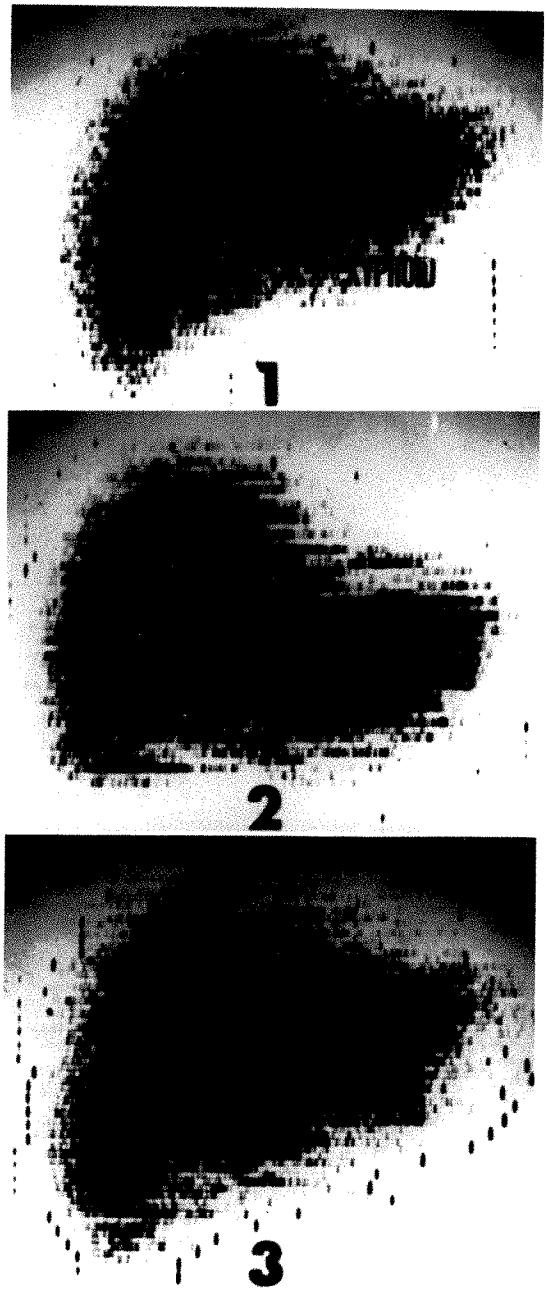



FIG. 3. Liver scans of 3 patients performed 1 hour after intravenous administration of 200 μc of zinc 69m chloride.

SUMMARY

Following intravenous injection of zinc 69m chloride, much of the zinc 69m was promptly removed by and concentrated in the liver. Technically good liver scans were obtained using this radioactive agent. The half life and photon energy of zinc 69m make it a desirable isotope for clinical work.

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# VISUALIZATION OF FILLING DEFECTS IN A LIVER PHANTOM CONTAINING $Tc^{99m}$, $Hg^{197}$, $I^{131}$ OR $Au^{198}$ USING A RECTILINEAR SCANNER OR SCINTILLATION CAMERA*

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IN CLINICAL scanning studies of the human liver using radioactive materials, focal disease is detected by its lower concentration of radioactivity than that found in normal surrounding liver tissue. As a result, defects in the liver are somewhat more difficult to visualize than focal disease in other organs, such as the brain, in which there is a higher concentration of radioactivity in the defective area than in normal brain tissue. Thus all aspects of liver scintiphotography must be studied carefully if results of clinical value are to be obtained.

Instruments employed for liver scintiphotography include the rectilinear scanner or the scintillation (Anger) camera. Techniques employed in operating the detector (including collimators), analyzer and display portions of these devices may be varied in a number of ways. All such variables must be understood and controlled for successful visualization of focal liver disease. Additional factors affecting the visualization of a space-occupying defect in the liver are defect size, the thickness of the liver in which the defect lies, the depth of the defect within the liver[6] and the properties of the radioisotope used.

The purpose of the authors is to relate the results of a series of experiments designed to evaluate the scintillation camera (Nuclear Chicago) and a rectilinear scanner (Picker Magnascanner) as devices for detecting filling defects in a liver phantom containing a solution of 1 of 4 radioactive isotopes—$Tc^{99m}$, $Hg^{197}$, $I^{131}$, and $Au^{198}$. The scintillation camera and scanner were operated in accordance with techniques established at this institution for routine clinical studies with these units.

## EXPERIMENTAL METHOD

The phantom used in this study was constructed by embedding an adult liver from the autopsy table in a pan containing liquid paraffin. After the paraffin solidified, the liver was removed and the space it had occupied was filled with water into which the radioactive material was added. Total volume of the liver phantom was 1,600 ml. with a variable depth from a minimum of 2 cm. in the left lobe to a maximum of 9 cm. in the deepest portion of the right lobe.

Spherical agates served as filling defects for our study. Determinations were made of the smallest detectable filling defect at selected positions within the phantom filled successively with each of the radioisotopic solutions. For each study with the scintillation camera, a total of 500,000 counts were accumulated using a 20 per cent window (10 per cent above and below the gamma energy being detected). For $Tc^{99m}$, $I^{131}$, and $Au^{198}$ studies, a 3 inch thick lead collimator was used with the face of the collimator placed at 3 cm. from the phantom surface. A $1\frac{1}{2}$ inch thick lead collimator was used in studies with $Hg^{197}$ because of its lower gamma energy (77 kev.). Interpretations were made from the Polaroid prints of the activity as displayed on the cathode ray tube of the camera.

The rectilinear scanner used for these studies had a 3 inch diameter NaI (Tl)

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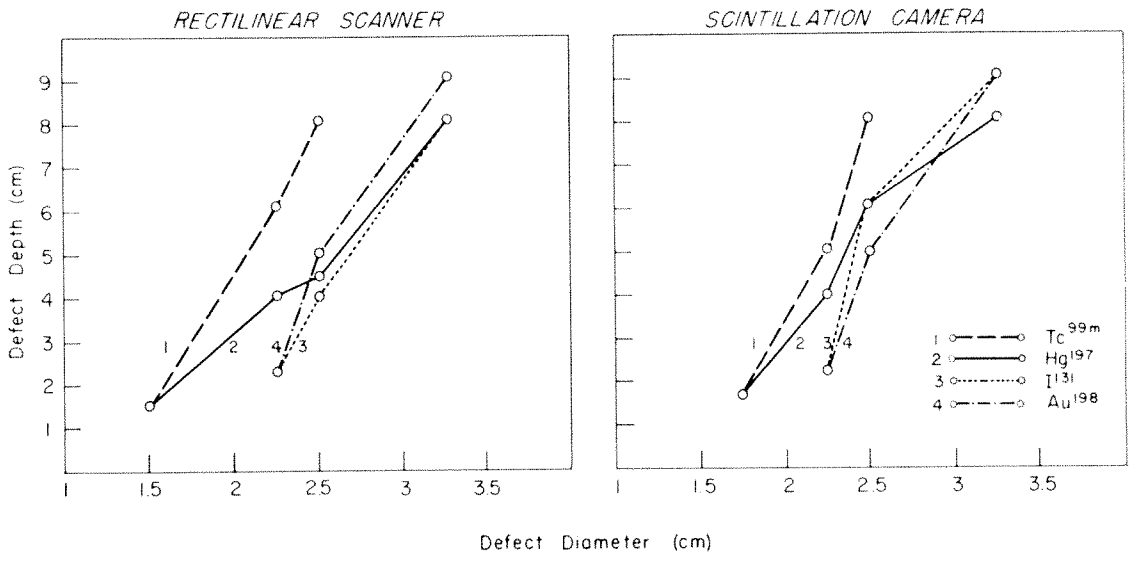



FIG. 1. Visualization of spherical defects at various depths in a liver phantom (phantom depth = 9 cm.). Scintiphotograms were obtained of the phantom filled with 1 of 4 radioisotopic solutions using either a rectilinear scanner or scintillation camera. Graphs 1, 2, 3 and 4 were obtained using $Tc^{99m}$, $Hg^{197}$, $I^{131}$ and $Au^{198}$ respectively.

crystal detector which travelled at a scan speed of 60 cm. per minute. A 3 inch thick focused lead collimator with 19 hexagonal holes and a $3\frac{1}{2}$ inch focal length was employed. The collimator face was placed 5 cm. from the phantom surface in keeping with our usual clinical practice. Spectrometer settings were comparable to those used for the scintillation camera. Interpretations were made exclusively from the photoscans.

The amount of radioisotope loaded into the phantom varied from a minimum of 100 microcuries when using $Au^{198}$ to a maximum of 1 millicurie when using $Tc^{99m}$. The concentration of radioisotope in the phantom was chosen so as to approximate the amount of radioactivity expected in the liver of a patient scheduled for a liver scan.

## EXPERIMENTAL RESULTS

The resolution obtainable with the Anger camera increases as the total number of counts accepted per picture is increased. The resolution also increases as the window or spectrometer width is reduced to accept a narrower band of photon energies. However, the counting rate is reduced by a decrease in window width, thus increasing the time required to obtain the same total number of counts. As a result, a compromise must be made between the total counts accepted and the window setting in order to obtain the best possible resolution in a reasonable amount of time. Thus, a series of preliminary experiments were conducted to obtain information on optimal conditions for using the scintillation camera.

These preliminary experiments were conducted with the liver phantom loaded with $Tc^{99m}$ in order to obtain information on optimal window setting for the camera. The phantom was loaded with 750 microcuries of $Tc^{99m}$ and the window setting varied from 1.0 to 60 per cent. A fixed time of 2 minutes was used in taking the pictures and they were examined to determine the optimal resolution of 2.25 cm. defect located at a depth of 5 cm. This study indicated that a 20 per cent window appeared to give the best resolution of the defect in the 2 minutes allotted and served as the window setting for the remainder of the studies.

Figure 1 consists of sets of 2 graphs relat-

ing to the visualization of defects at various depths in the phantom. The first set is a plot of defect diameter *vs.* the maximum depth at which the defect was barely visible on the photoscan obtained using the rectilinear scanner. The second set is a similar plot based on scintiphotograms from the scintillation camera. For this part of the study, phantom thickness was maintained at a constant value of 9 centimeters.

Both sets in Figure 1 indicate that defects can be visualized at greater depths with $Tc^{99m}$ than with the other 3 isotopes. For surface defects the same resolution was found with $Hg^{197}$ as with $Tc^{99m}$, but at increasing depths the resolution with $Hg^{197}$ dropped off rapidly. Similar resolution was achieved with both camera and scanner when using either $Au^{198}$ or $I^{131}$ in the phantom. Neither of these isotopes was capable of revealing surface defects as small as those seen with $Tc^{99m}$ and $Hg^{197}$, and both exhibited poorer resolution of defects deep in the phantom.

Figure 2, *A–C* illustrates the method used to determine a single point on the plot of data in Figure 1. A 1.5 cm. defect was suspended in the phantom at depths of 5, 6, and 7 cm. The phantom thickness in this region was 9 cm. The defect is visible with the scintillation camera when placed at depths of 5 and 6 cm. (Fig. 2, *A* and *B*), but not at a depth of 7 cm. (Fig. 2*C*).

Figure 3 consists of 2 sets of graphs comparing the bare visualization of defects just below the phantom surface when using the camera and scanner. As the thickness of the underlying phantom was increased, it was necessary to increase defect size proportionately in order to detect its presence. Similar results were obtained when using either $Tc^{99m}$ or $Hg^{197}$ in the phantom. The minimum defect size that

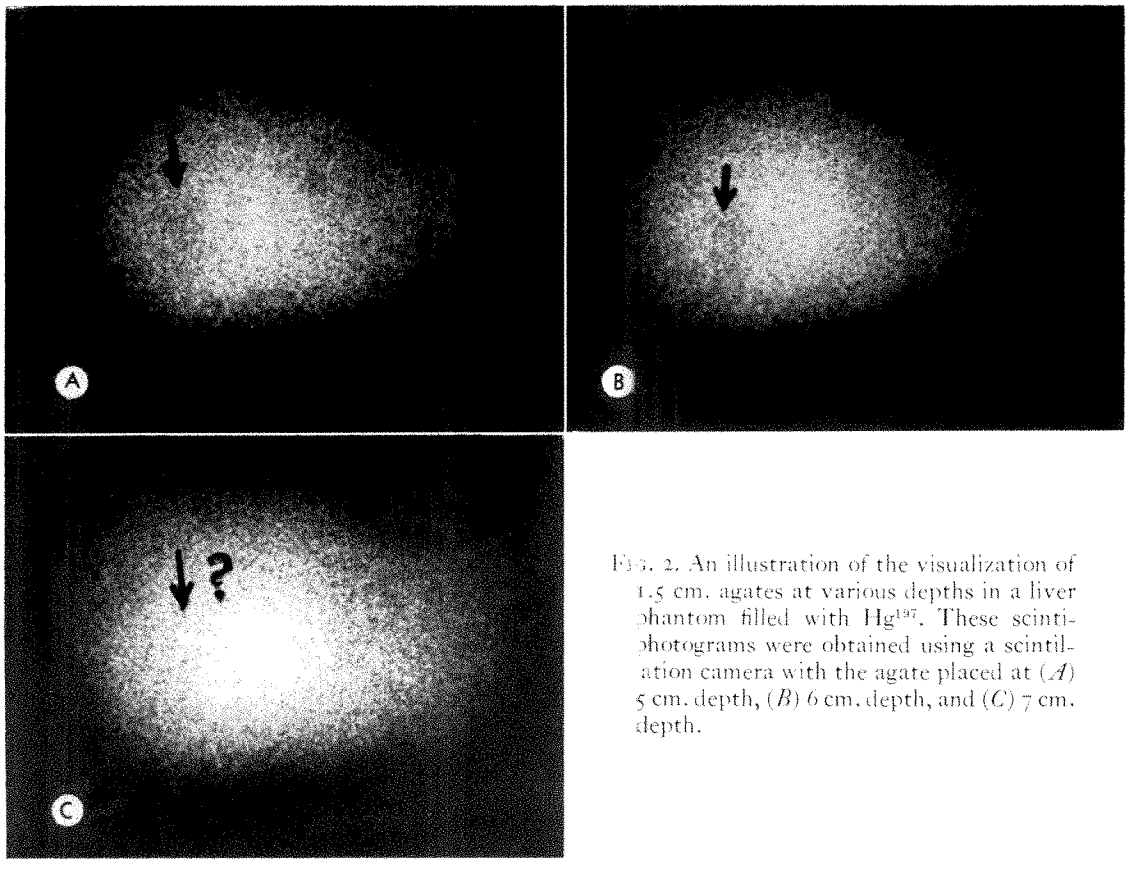



FIG. 2. An illustration of the visualization of 1.5 cm. agates at various depths in a liver phantom filled with $Hg^{197}$. These scintiphotograms were obtained using a scintillation camera with the agate placed at (*A*) 5 cm. depth, (*B*) 6 cm. depth, and (*C*) 7 cm. depth.

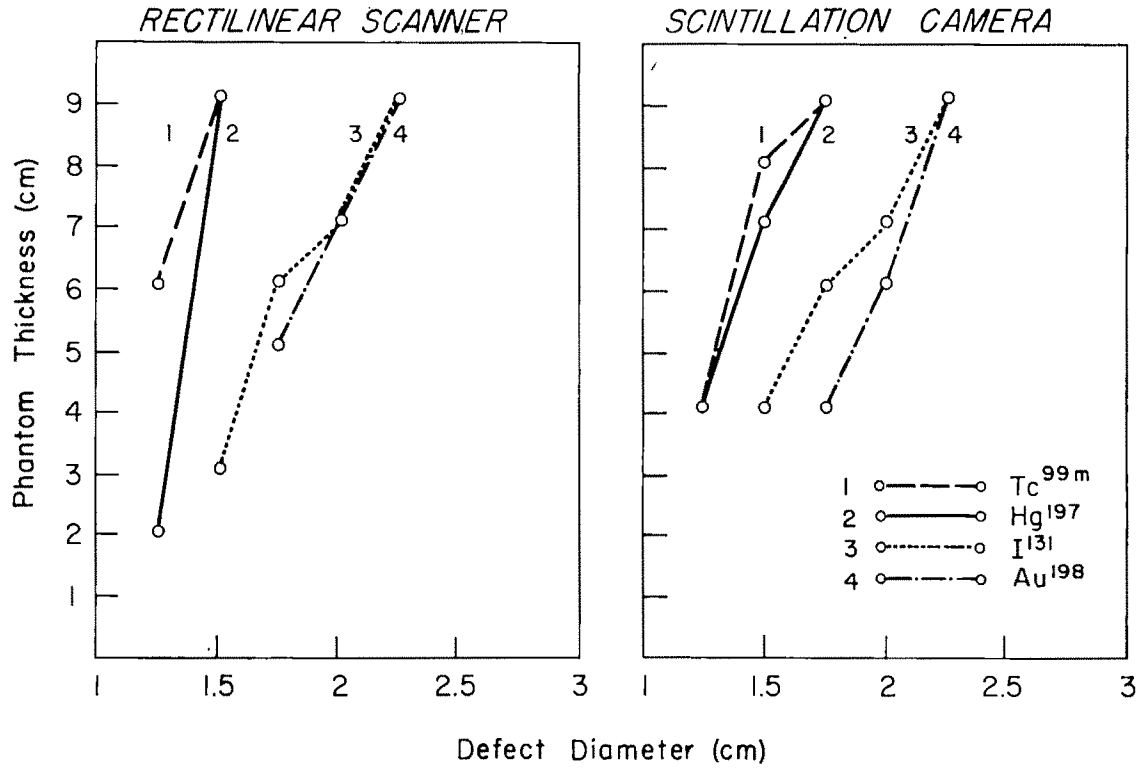



FIG. 3. Visualization of spherical defects at the surface of a liver phantom (variable phantom depths) using either a rectilinear scanner or scintillation camera. Scintiphotograms were obtained of the phantom filled with 1 of 4 isotopes. Graphs 1, 2, 3 and 4 were obtained using $Tc^{99m}$, $Hg^{197}$, $I^{131}$ and $Au^{198}$ respectively.

could be resolved with either camera or scanner when using $Tc^{99m}$ and $Hg^{197}$ was 1.25 cm. in diameter. As found in the study shown in Figure 1, agates were less well detected when $I^{131}$ and $Au^{198}$ were used in the phantom than when either $Tc^{99m}$ or $Hg^{197}$ was used. Slightly better results were obtained with $I^{131}$ than with $Au^{198}$ for both the camera and the scanner. The minimum defect size that could be resolved using $I^{131}$ was 1.5 cm. in diameter for both the camera and scanner, whereas 1.75 cm. was the smallest agate visible in the phantom containing $Au^{198}$.

Figure 4, *A* and *B* illustrates a slight difference in resolution of agates by the camera and scanner when the phantom was loaded with $Hg^{197}$. Four defects of 1.5 cm. diameter were submerged just below the phantom surface. They were located over phantom thicknesses of 6, 7, 8, and 9 cm. On the scan all 4 defects are clearly visible, whereas only the 2 defects located over 6 and 7 cm. thickness of phantom are visible on the scintiphotogram from the scintillation camera.

## DISCUSSION

According to previous reports the minimum defect size that can be visualized in the liver has been somewhat variable. Studies using a liver phantom and the isotopes $Au^{198}$ and $I^{131}$ have indicated in one case that "spherical defects 2.0 to 2.5 cm. in diameter can usually be demonstrated,"[2] while in another study it was determined that a 2.5 cm. spherical defect at the surface of the liver could be seen regardless of the thickness of the liver, whereas defects 1.7 cm. in diameter could not be seen.[1] Clinical evidence from patient liver scans places the lower limit for defect discrimination at 2 cm. diameter.[4,5] Another clinical report places the limitation at 1.5 cm. for nodules

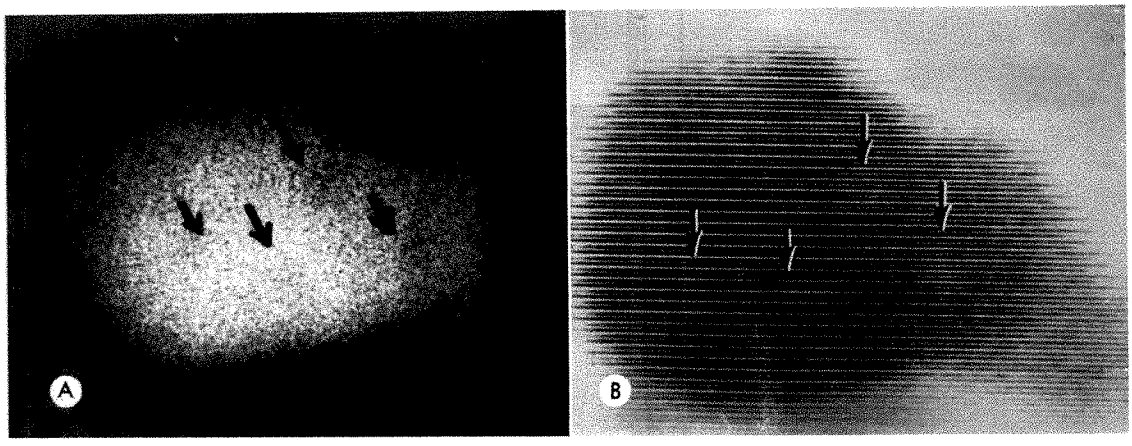



FIG. 4. An illustration of scintiphotograms obtained of 1.5 cm. agates at the surface of a liver phantom. (*A*) Scintiphotogram obtained using a scintillation camera. (*B*) Scintiphotogram obtained using the rectilinear scanner. The depth of the liver phantom increased gradually moving from right to left so that agates near the left hand side of the phantom have more underlying radioactivity and are more difficult to visualize.

in the left lobe or dome, but indicates that lesions of 2.0 to 3.0 cm. may escape detection in the deep right lobe.[1]

Our study has revealed that experimental glass agate defects as small as 1.25 cm. diameter can be visualized at the surface of a liver phantom when the isotopes $Tc^{99m}$ or $Hg^{197}$ are used. $Tc^{99m}$ and $Hg^{197}$ exhibit similar resolving ability at shallow defect depths with both scintillation camera and rectilinear scanner. At increasing depths, however, the resolution with $Hg^{197}$ decreases rapidly, apparently due to absorption of its relatively low (77 kev.) photon by the greater phantom thickness between detector and defect.

Scans made with both $I^{131}$ and $Au^{198}$ were found to be inferior to those made with $Tc^{99m}$ and $Hg^{197}$ in the resolution of surface defects in the liver phantom. This was true regardless of the depth of phantom underlying the defect. As the defect depth was increased, its resolution in the phantom containing $Au^{198}$ or $I^{131}$ was comparable to that obtained with $Hg^{197}$ but inferior to that obtained with $Tc^{99m}$. Kuhl[3] believes septal penetration of collimators by both these high energy isotopes to be significant and apparently this is the reason for the inferior resolutions of $Au^{198}$ and $I^{131}$ with the collimators used in this experiment. The above results apply to both scintillation camera and rectilinear scanner studies; the only notable difference in resolution between these two detector systems was the slightly better resolution of small surface defects using the scanner.

Caution must be exercised in applying these results to actual liver scanning. First of all, respiratory motion will tend to blur the sharpness of discrete filling defects in the liver. In addition, patient motion as well as variable thickness of overlying tissue, including ribs, serves to decrease resolution.

## SUMMARY

A series of experiments were performed to evaluate the Anger scintillation camera (Nuclear Chicago) and the rectilinear scanner (Picker Magnascanner) as devices for detecting defects in a liver phantom containing a solution of 1 of the following 4 radioactive isotopes: $Tc^{99m}$, $Hg^{197}$, $I^{131}$, and $Au^{198}$.

The experimental results are given in detail.

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# RECTILINEAR LIVER SCANNING WITH TECHNETIUM 99m SULFIDE COLLOID*

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UTILIZATION of technetium 99m sulfide colloid (TcS) for liver scanning holds the promise of improved resolution and greater scanning speeds as compared to currently employed agents.[2-4] Comparison of TcS and colloidal $Au^{198}$ was carried out on 25 unselected patients to determine the feasability of replacing the latter with the former agent for liver scanning.

The following factors were examined: (1) the waiting period between injection of TcS and maximal liver radioactivity; (2) the reproducibility of the TcS as indicated by the liver to blood pool radioactivity ratios; (3) the total liver scanning time; and (4) the subjective scan quality.

## METHODS

The TcS was produced by the method of Larson and Nelp[3] utilizing pertechnetate 99m ($Tc^{99m}O_4^-$) from a sterile generator.* Each generator was initially eluted on Monday and then daily thereafter until Friday when it was discarded. Twenty minutes were required each morning to elute the $Tc^{99m}O_4^-$ and make and calibrate† enough colloid for 2 or 3 scans. The "flocculent precipitate" observed by Larson and Nelp in their final product was frequently present in our preparation of the colloid.

With the patient in the supine position, a 2 by 2 inch sodium iodide (Tl) crystal probe‡ was placed at skin level over the left precordium (blood pool, BP), while a similar probe was placed in the right anterior axillary line directed toward the central body axis just superior to the right rib margin (liver, L). Radioactivity was recorded by a dual strip chart recorder. Three millicuries of TcS were administered intravenously to each patient.

The liver to blood pool (L/BP) ratio may be used as an index of the quality of the colloid in the absence of severe liver disease and abnormal portal blood flow. The L/BP ratio data were taken from the plateau portion of each curve (Fig. 1). A "good" quality colloid gives high liver and low blood pool radioactivity; *i.e.* the L/BP ratio is high. Colloid preparations yielding a high L/BP ratio were concomitantly found to exhibit a higher fraction of the technetium 99m in the TcS position on descending paper chromatography (95 per cent methanol solvent).[3] Although the L/BP ratio is variable depending on probe position, geometry and liver disease, it is reproducible within limits suitable for application here. On three occasions dual batches of the TcS were made, one employing filtered

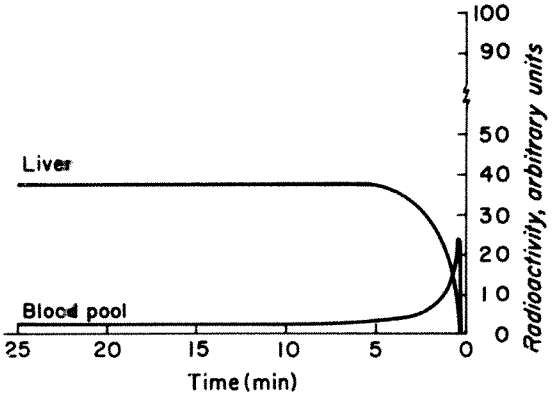



FIG. 1. A diagrammatic representation of the radioactivity recorded from the liver and blood pool probes *vs.* time following the intravenous injection of TcS.

* Technetope Generator Sterile, E. R. Squibb and Sons, Inc., New York.
† Ion chamber, Baird Atomic Inc., Valley Stream, New York.
‡ Twinprober, Picker X-ray Corporation, White Plains, New York.

* From the Department of Nuclear Medicine, The Princess Margaret Hospital and University of Toronto.
This study was supported by a grant from the Ontario Cancer Treatment and Research Foundation.

TABLE I

| Time of Generator Elution | L/BP Ratio | |
|---|---|---|
| | Nonfiltered eluate | Filtered eluate |
| First day (Monday) | 2.1 | 2.9 |
| Second day (Tuesday) | 2.8 | 4.6 |
| Third day (Wednesday) | 4.4 | 7.6 |

(0.45 $\mu$ filter pore size) $Tc^{99m}O_4^-$ eluate, and the other employing unfiltered eluate. First the TcS prepared from the unfiltered eluate was administered yielding a curve qualitatively similar to Figure 1. After the plateau had been reached (approximately 10 minutes), a second injection of TcS (filtered eluate) was then given and a second curve was obtained. The plateau of the first curve served as a background and was subtracted from the second curve. Thus the L/BP ratios could be determined for each preparation on the same patient (Table 1).

When the radioactivity recorded from the liver probe had remained at its plateau level for about 10 minutes, the patient's liver was then scanned with a rectilinear photoscanner equipped with a 5 inch sodium iodide (Tl) crystal probe with a 163 hole lead (fine) collimator.* At the completion of the scan 1 $\mu$c/kg. of colloidal $Au^{198}$ was injected intravenously into the same patient. After waiting approximately three quarters of an hour, the $Au^{198}$ scanning was carried out employing the same scanner, but utilizing a 31 hole lead (coarse) collimator and appropriate spectrometer settings for $Au^{198}$ to exclude the technetium 99m photons. Line spacing was set at 0.3 cm.

The TcS and colloidal $Au^{198}$ scans were performed at 150 cm./min. and 30–40 cm./min., respectively. These speeds were chosen to provide a minimum of 800 counts/cm.$^2$ measured at the point of maximum liver radioactivity; however, for the TcS scans more favorable statistics were often present (greater than 1,000 counts/cm.$^2$ maximum) depending upon the quality of the colloid.

The radiation dose to the liver and whole body from $Tc^{99m}$ is reported to be one third that of $Au^{198}$ for the doses of radioactivity employed.[6]

* Magnascanner V, Picker X-ray Corporation, White Plains, New York.

## RESULTS

The radioactivity recorded from the liver probe reached a plateau in 17 minutes or less. No patients with known severe parenchymal liver disease or abnormal portal blood flow were examined, thus making 17 minutes a conservative estimate of the plateau time. The average time to reach the plateau was 10 minutes. When the liver radioactivity had reached its

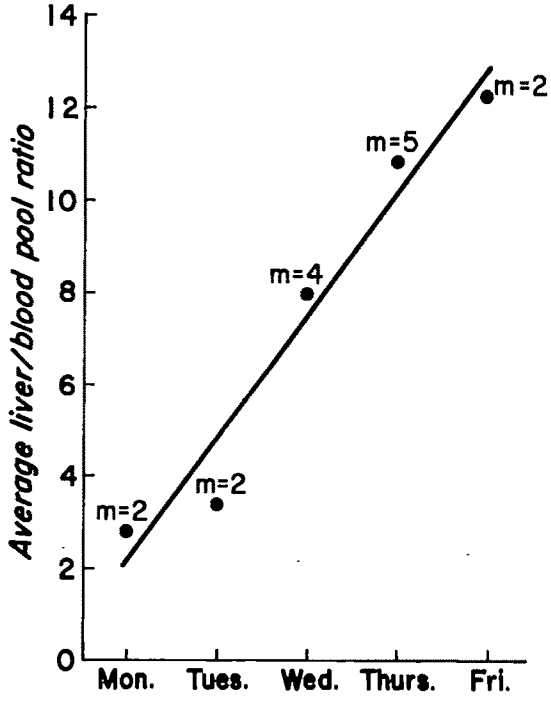



FIG. 2. The average daily liver to blood pool (L/BP) ratio *vs.* the day of the week for a 3 week period. The number of patients averaged for each point shown = m.

plateau value, the blood pool curve had simultaneously reached its plateau (Fig. 1).

The most remarkable finding was that the L/BP ratio varied rather consistently with the day of the week. Uniformly, on Monday (the day of initial generator elution) it was lowest and steadily rose with repeated elution. Figure 2 shows the average ratio for each day of a 3 week period. It was noted that on Mondays and occasionally on Tuesdays a white precipitate appeared in the pertechnetate eluate (presumably alumina[5]) and this cleared with increasing elution throughout the week.

Although filtration offered some improvement in colloid quality, as exhibited in Table 1, it was not as effective as repeated elutions (Fig. 2). These data indicate that a filterable agent was only in part responsible for the formation of a poor quality colloid. The agent responsible for the formation of poor quality TcS was also in solution (possibly excess $Al^{+3}$ or $Mo^{+6}$) and could be flushed from the column with successive elutions. A qualitative spot test for $Al^{+3}$* performed on over half the pertechnetate eluates showed highest $Al^{+3}$ concentration on Mondays diminishing as further elutions were performed during the week. Methods to remove $Al^{+3}$ and $Mo^{+6}$ are available but were not examined in this study.[2]

The $Tc^{99m}$ scans required one half to one third of the scanning time of the $Au^{198}$ scans (ranges 13 to 25 *vs.* 42 to 87 minutes) for the conditions employed.

As the study was designed, it should have favored improved resolution from the $Tc^{99m}$ (better collimated photons, finer collimator, and occasionally better statistics). However, on comparison with the consecutively performed $Au^{198}$ scan, no such improved resolution was obvious. No anatomic verification was available in any case, but the consecutively performed scans never yielded conflicting impressions. In many instances the $Tc^{99m}$ scans more clearly depicted the liver periphery, (usually associated with >1,000 counts/cm.$^2$ maximum) particularly over the often confusing and patchy edges of the left lobe. In about one third of the patients, the spleen appeared more prominently on the $Tc^{99m}$ scan than on the $Au^{198}$ scan. This may be due to variations in the colloid

* Aluminon, British Drug Houses Ltd., Toronto, Ontario, Canada.

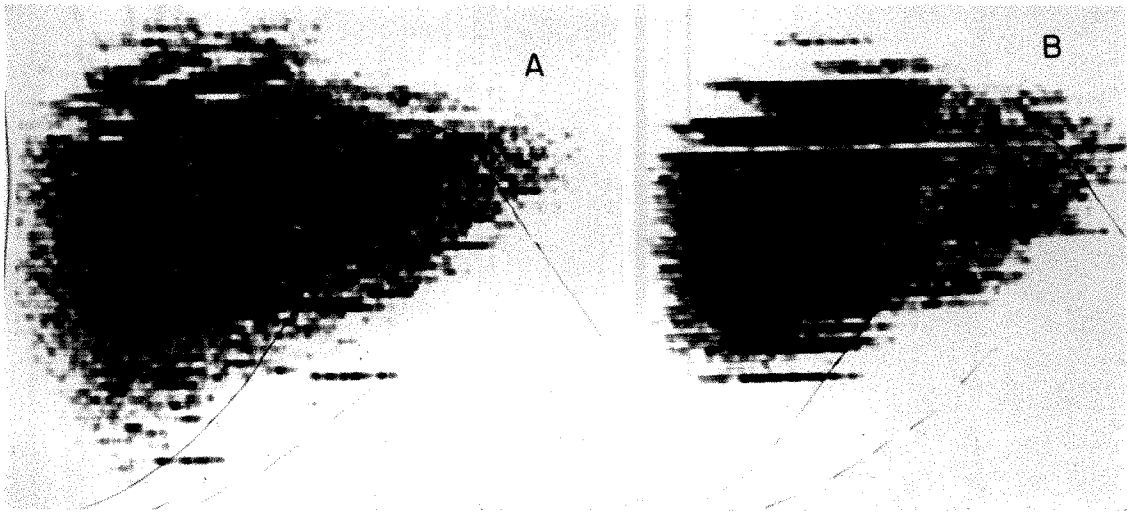



FIG. 3. The liver scans pictured above were recorded from the same patient about 45 minutes apart. The colloidal $Au^{198}$ scan (*A*) shows marked respiratory artifacts most prominent along its superior border. The TcS scan (*B*) demonstrates these same respiratory artifacts to an even greater degree yielding ragged liver borders and whole lines of absent photo-recording within the liver.

quality and particle size and/or due to the patient's disease process. Imaging of the spleen in the course of liver scanning may in some instances be an advantage and in other instances be a drawback.

One serious problem became evident during this study. Respiratory movement (or other movements) of the liver may be seen on a $Au^{198}$ scan but due to the slower speed of the probe offers minimal distortion of the over-all scan appearance. At 150 cm./min. the same respiratory motion produces a disrupting artifact (seen in about one third of the TcS scans) and when these occurred repeatedly on the same scan, the appearance was distracting (Fig. 3, *A* and *B*).

## CONCLUSIONS AND SUMMARY

Once recognized, the problems suggested by this study are, at least in part, solvable. Initial washing of the $Tc^{99m}$ generator either by the manufacturer or by the recipient should yield uniformly high quality TcS. The short scanning time is desirable and provides time for lateral views. The post-injection waiting period to commence scanning for TcS is not dissimilar from that of $Au^{198}$ colloid and should be at least 30 minutes to allow for abnormalities of liver blood flow.

Respiratory liver movement limits the resolution of a rectilinear scan at the speeds used with $Tc^{99m}$ as pointed out by Harper *et al.*[2] Perhaps the best solution to this problem is to use some variation of a gamma camera with shorter exposure times.[1] For the majority of workers with rectilinear scanners sedation, an abdominal binder, and increased patient cooperation may be of some value.[2] A 31 hole lead collimator would probably yield resolution not significantly different from that observed with the 163 hole lead collimator for TcS liver scanning because of the limitations imposed by liver movement. The coarser collimator would yield higher count rates (better statistics than utilized for this study) and still allow rapid scanning. However, the desirability of more rapid rectilinear liver scanning will remain uncertain until the liver motion problem is solved.

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# THE APPLICATION OF COLLOIDAL RADIOGOLD AND RADIOIODINATED ROSE BENGAL IN HEPATOBILIARY DISEASE*

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THE literature is replete with the functional and scanning aspects of colloidal radiogold ($Au^{198}$) and radioiodinated rose bengal ($RBI^{131}$) in diagnosing hepatobiliary disease. The rate of $Au^{198}$ disappearance from the blood has been shown to be a parameter of liver perfusion.[10,11] Rose bengal is distributed in a manner comparable to bromsulphalein (BSP) and its rate of uptake[2] or clearance from the blood is an index of polygonal cell function.[5,6,10] The distinction between extrahepatic and hepatocellular jaundice may be made by monitoring the abdomen for the time of entry of rose bengal into the intestine.[1,5,9] Recently there have been reports on serial liver and abdomen scannings to visualize the distribution and excretion of rose bengal in order to differentiate the type of jaundice.[2,8] Characteristic patterns have also been described with $Au^{198}$ scans.[4]

During the last 4 years we have been using the combination of $RBI^{131}$ retention and excretion and $Au^{198}$ retention as a means of evaluating hepatobiliary disease. The acquisition of the gamma-ray scintillation camera made it feasible to add serial 3 or 4 $RBI^{131}$ liver and abdomen scans in a 24 hour interval to the original approach. The present report is an assessment of the combined radionuclide study in the evaluation of liver perfusion, and differentiation of polygonal cell disease from obstructive choledochopathy.

## TECHNIQUE

Both the $RBI^{131}$ and $Au^{198}$ retention curves are obtained by placing a 20° diverging collimator associated with a 2 inch scintillation crystal against the side of the head encompassing the ear. The disappearance of the intravenously injected radionuclide is recorded on a rectilinear strip chart. A ratio of the net 20 minute to the 5 minute count rate for $RBI^{131}$ is used as an index of clearance and called the "20 minute $RBI^{131}$ retention." $Au^{198}$ is cleared more rapidly from the blood stream and only a 3 minute period of observation is necessary, but it takes about a minute post injection for the curve to settle after the initial bolus. The "4.5 minute $Au^{198}$ retention" is calculated as a ratio of the net 4.5 minute to the 1.5 minute count rate.

Prior to initiating serial rose bengal scans, 25 $\mu$c $RBI^{131}$ containing about 0.1 mg. carrier was used. This was followed by 200 $\mu$c $Au^{198}$ which was used to obtain the retention curve and liver scan. The present sequence calls for a 200 $\mu$c $RBI^{131}$ retention curve and serial scans of the liver and abdomen at approximately 30 minutes, 3 hours, 6 hours, and 24 hours. This is followed by a 200 $\mu$c $Au^{198}$ retention study and liver scanning.

## CLINICAL MATERIAL

Some 450 patient charts were reviewed and a definitive diagnosis was obtained in 309 $RBI^{131}$ and 311 $Au^{198}$ retention curves. Livers were judged normal only if the bilirubin, alkaline phosphatase, cephalin flocculation, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), and BSP retention were negative.

This series was categorized into the following groups: normal, acute hepatitis with jaundice, obstructive jaundice, acute

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Supported in part by the John A. Hartford Foundation.

TABLE I

LIST OF CONDITIONS STUDIED WITH SERIAL $I^{131}$ ROSE BENGAL LIVER SCANS

| Condition | No. of Patients |
|---|---|
| Normal | 26 |
| Extrahepatic obstruction | |
| Partial | 12 |
| Complete | 8 |
| Acute hepatitis | 12 |
| Liver containing tumor | 5 |
| Cirrhosis | |
| Compensated | 12 |
| Decompensated | 9 |
| Fatty metamorphosis | 5 |
| Pancreatitis | 3 |
| Bile peritonitis | 2 |
| Congenital biliary atresia | 2 |
| Granulomatous hepatitis | 1 |
| Total | 97 |

congestive heart failure, liver containing tumor, fibrotic liver disease without jaundice, and fibrotic liver disease with jaundice.

All cases with fibrotic liver disease were proved by needle biopsy, laparotomy, or clinically obvious disease. This group included postnecrotic cirrhosis, nutritional cirrhosis, cardiac cirrhosis, chronic hepatitis, hemochromatosis, biliary cirrhosis, fatty liver with fibrosis, and amyloid disease.

A definitive diagnosis was obtained in 97 patients who had the entire radionuclide study consisting of $RBI^{131}$ and $Au^{198}$ retention curves, $Au^{198}$ liver scan, and serial $RBI^{131}$ liver and abdomen scans (Table 1).

## RESULTS AND DISCUSSION

Figure 1 shows the histograms of 120 $RBI^{131}$ and 116 $Au^{198}$ retention curves in normal patients. Approximately 95 per cent of the patients had a 20 minute $RBI^{131}$ retention of less than 60 per cent and a 4.5 minute $Au^{198}$ retention of less than 70 per cent. These values have been used as the upper limits of normal in the evaluation of disease.

The 4.5 minute $Au^{198}$ retention in obstructive jaundice was less than 70 per cent in 27 out of 28 patients, and the 20 minute $RBI^{131}$ retention was greater than 60 per cent in 23 out of 24 patients (Fig. 2). The maximum retention of 86 per cent was observed in 2 patients with complete obstruction of the common duct.

The findings in acute hepatitis were similar to those found in obstructive jaundice. All 19 patients had 20 minute $RBI^{131}$ retentions greater than 60 per cent, and 16 out of 19 patients had 4.5 minute $Au^{198}$ retentions of less than 70 per cent (Fig. 3). The 2 patients with $Au^{198}$ retentions exceeding 80 per cent had acute yellow atrophy and a corresponding $RBI^{131}$ retention exceeding 95 per cent.

The $RBI^{131}$ retention in fibrotic liver disease without jaundice ranged from 40 per cent to 90 per cent with a median of 65 per cent (Fig. 4) compared to a median of 50 per cent in the normal group. The $Au^{198}$ retentions varied from 50 per cent to 100 per cent.

All 12 patients with $Au^{198}$ retentions of less than 70 per cent had large fatty livers with some fibrosis on histologic examina-

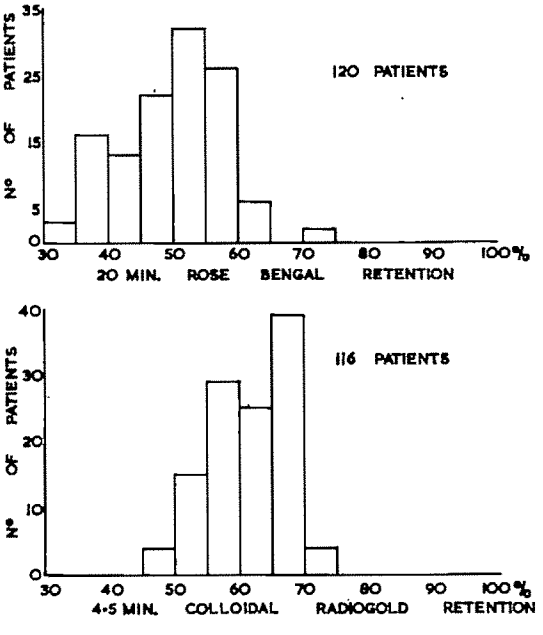



FIG. 1. Histogram of $RBI^{131}$ and $Au^{198}$ retentions in patients with normal livers.

tion. Most of these patients had a history of high alcohol intake and the observed findings probably represented early nutritional cirrhosis, but these cases were not detected by the $Au^{198}$ function study.

All patients with fibrotic liver disease with jaundice had $RBI^{131}$ and $Au^{198}$ retentions greater than 60 per cent and 70 per cent, respectively (Fig. 5).

The presence of tumor in the liver rendered unpredictable $RBI^{131}$ and $Au^{198}$ retention values (Fig. 6).

Most patients in acute congestive heart failure had normal $Au^{198}$ retention curves, but 7 patients had values between 70 per cent and 85 per cent. Two of these patients were re-examined during remission and their $Au^{198}$ retention values reverted to normal.

In the group of 97 patients studied with serial $RBI^{131}$ liver scans, one consistent observation was a uniform distribution and decrease of activity in normal livers and those affected by acute hepatitis. All cases of acute hepatitis exhibited activity in the intestine, save for 1 patient with acute yellow atrophy, who had a $RBI^{131}$ retention of 100 per cent. Most complete obstructions had $RBI^{131}$ retentions between 80 per cent and 86 per cent (the lowest was

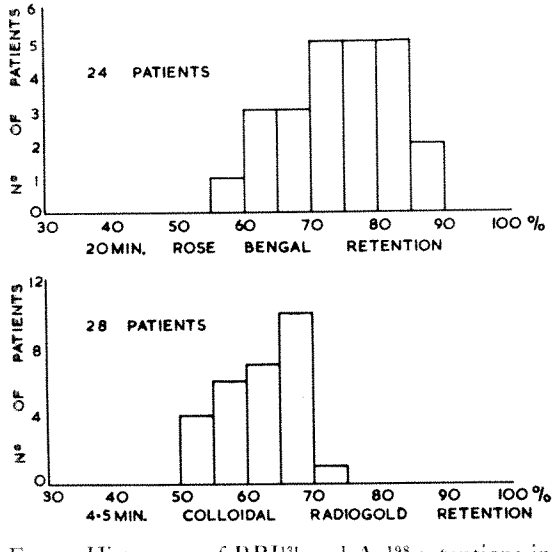



FIG. 2. Histogram of $RBI^{131}$ and $Au^{198}$ retentions in patients with obstructive jaundice.

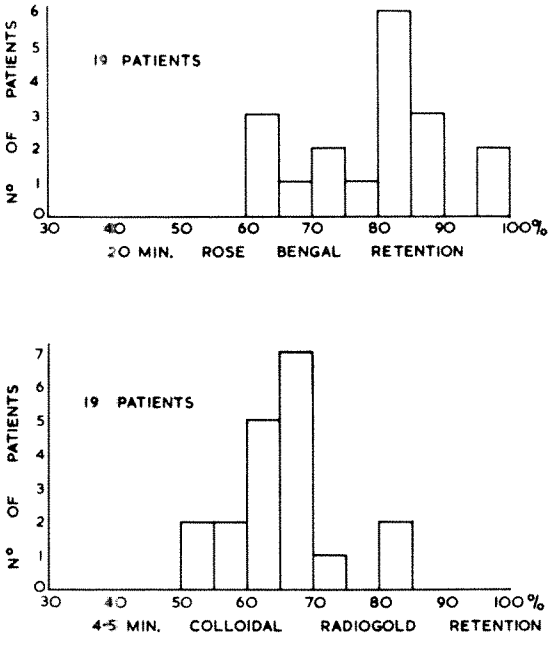



FIG. 3. Histogram of $RBI^{131}$ and $Au^{198}$ retentions in patients with acute hepatitis and jaundice.

72 per cent), and there was no activity detected in the intestine. Only the kidneys and urinary bladder contained $I^{131}$ in the scan of the abdomen. Therefore, a $RBI^{131}$ retention greater than 86 per cent strongly favors polygonal cell disease over extrahepatic obstruction whether or not there is detectable excretion, and a retention between 80 per cent and 86 per cent associated with activity in the intestine also supports a diagnosis of polygonal cell disease.

The indeterminate group is comprised of those patients who are icteric, have a $RBI^{131}$ retention less than 80 per cent and exhibit activity in the intestine during the 24 hour observation period. The size of this group may be reduced by an analysis of the serial $RBI^{131}$ and $Au^{198}$ liver scans. A focal accumulation of $RBI^{131}$ in the liver adjacent to the porta hepatis indicates a dilated biliary tree (Fig. 7). A changing relative concentration of activity within the liver with time supports regionally impaired drainage due to obstruction in one of the major hepatic biliary radicles (Fig. 8, *A* and *B*).

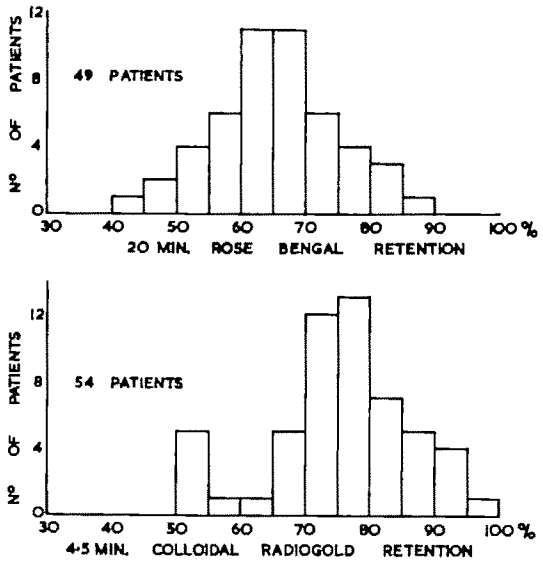



FIG. 4. Histogram of $RBI^{131}$ and $Au^{198}$ retentions in patients with fibrotic liver disease and no jaundice.

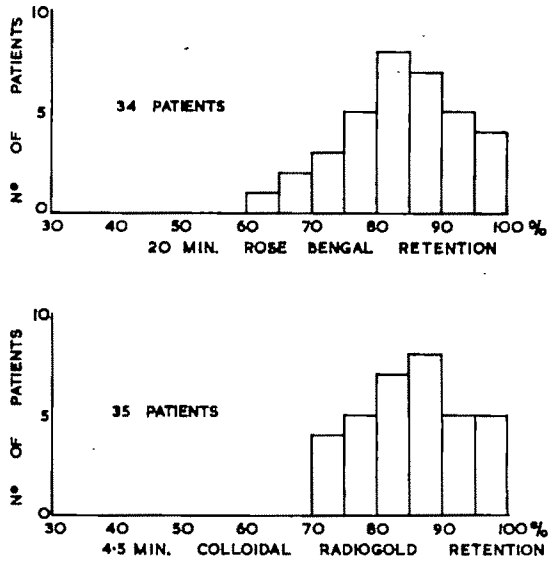



FIG. 5. Histogram of $RBI^{131}$ and $Au^{198}$ retentions in patients with fibrotic liver disease and jaundice.

The addition of the serial $RBI^{131}$ scans permitted a diagnosis of partial obstruction in 9 out of 12 cases which would have been missed if reliance were placed solely on retention and excretion of $RBI^{131}$ with the dual probe method.[7] Two of the false negative scans had a common duct diameter of 2 cm. and the third had a diameter of 3 cm. All 3 patients had an elevated alkaline phosphatase with no evidence of hepatocellular disease as determined by the SGOT and SGPT. In essence, if a dilated biliary tree or differential drainage within the liver is observed, a diagnosis of obstruction can be made, otherwise it remains indeterminate.

The finding of a "cold" region adjacent to the porta hepatis in the $Au^{198}$ scans is not a useful sign of surgical jaundice, as it could be due to tumor or be a normal variation. When the concentration of $RBI^{131}$ in the dilated biliary tree is the same as the adjacent parenchyma, the $Au^{198}$ scan will more clearly define the parenchymal concentration and aid in the diagnosis of obstructive choledochopathy.

Serial $RBI^{131}$ liver scans may also be useful in uncovering bile peritonitis. The following is a case in point:

This 80 year old male was admitted to the Montreal General Hospital with an acute onset of obstructive jaundice. At laparotomy a carcinoma of the bile ducts was found. A T tube was placed in the right hepatic duct and the wound closed. Postoperatively the patient

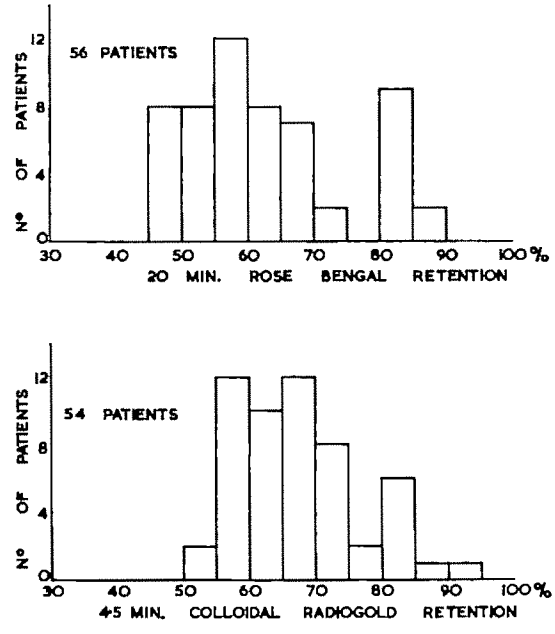



FIG. 6. Histogram of $RBI^{131}$ and $Au^{198}$ retentions in patients with primary and metastatic tumor of the liver.

developed a high fever. The bilirubin was 4.3 mg. per cent and the alkaline phosphatase was increasing. Serial $RBI^{131}$ liver scans (Fig. 9, *A–C*) and abdomen scan (Fig. 9*D*) showed a marked reduction in concentration in the right lobe relative to the left initially, but by 24 hours the situation was reversed. The radioactive material was also seen in the transverse and descending colon. After ascertaining that the T tube was patent a diagnosis of a bile leak was made, and this was later confirmed at a second laparotomy.

Two cases of biliary atresia were studied and they differed in the distribution of rose bengal. A 3 month old female infant showed good uniform concentration of $RBI^{131}$ in the liver which persisted for the 24 hour observation period, but at no time was there excretion into the intestines. The second patient was a 2 1/2 year old male. At 5 minutes (Fig. 10*A*) the $RBI^{131}$ was seen in the liver and cardiac blood pool. By 1 hour (Fig. 10*B*) the cardiac blood pool and liver concentration were markedly reduced and activity was observed gathering in the kidneys. At 4 hours (Fig. 10*C*) only a small amount of activity remained in the liver and the kidneys were clearly outlined. At no time was $I^{131}$ detected in the intestine. It would seem that the older child deiodinated the $RBI^{131}$ much more rapidly.

Recently 4 patients with toxic hepatitis were studied. In 3, the toxic hepatitis was due to chlorpromazine, and in the 4th it was due to a combination of cancer chemo-

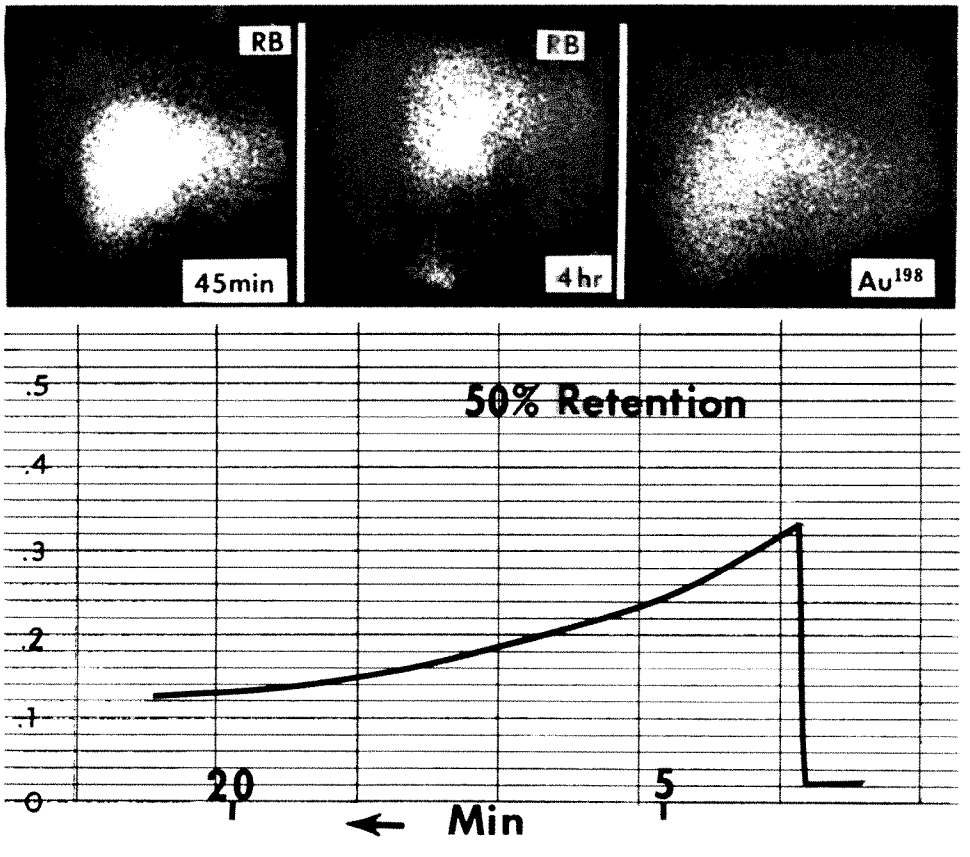



Fig. 7. A 35 year old female with partial obstruction of the common duct by a single stone. Bilirubin 9.2 mg. per cent, alkaline phosphatase 30 units (Bodansky), and SGOT 500 mg. per cent. $RBI^{131}$ retention is normal, but the 45 minute and 4 hour $RBI^{131}$ scans show an increased accumulation of activity in the dilated biliary tree. The $Au^{198}$ liver scan depicts the nonbiliary distribution of activity. Excretion of $RBI^{131}$ into the intestine is seen at 4 hours.

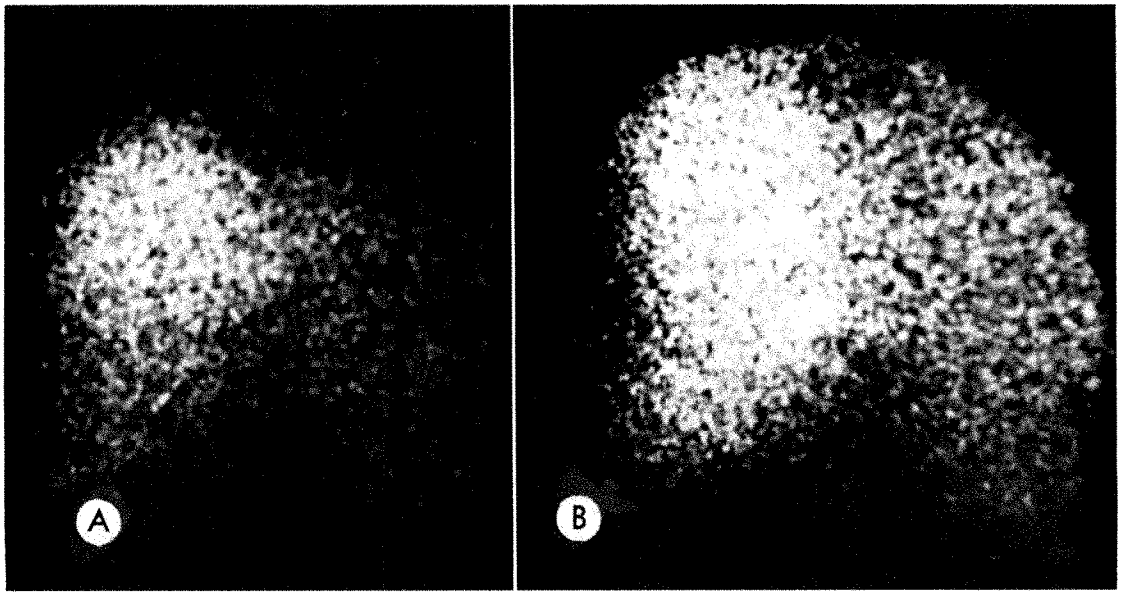
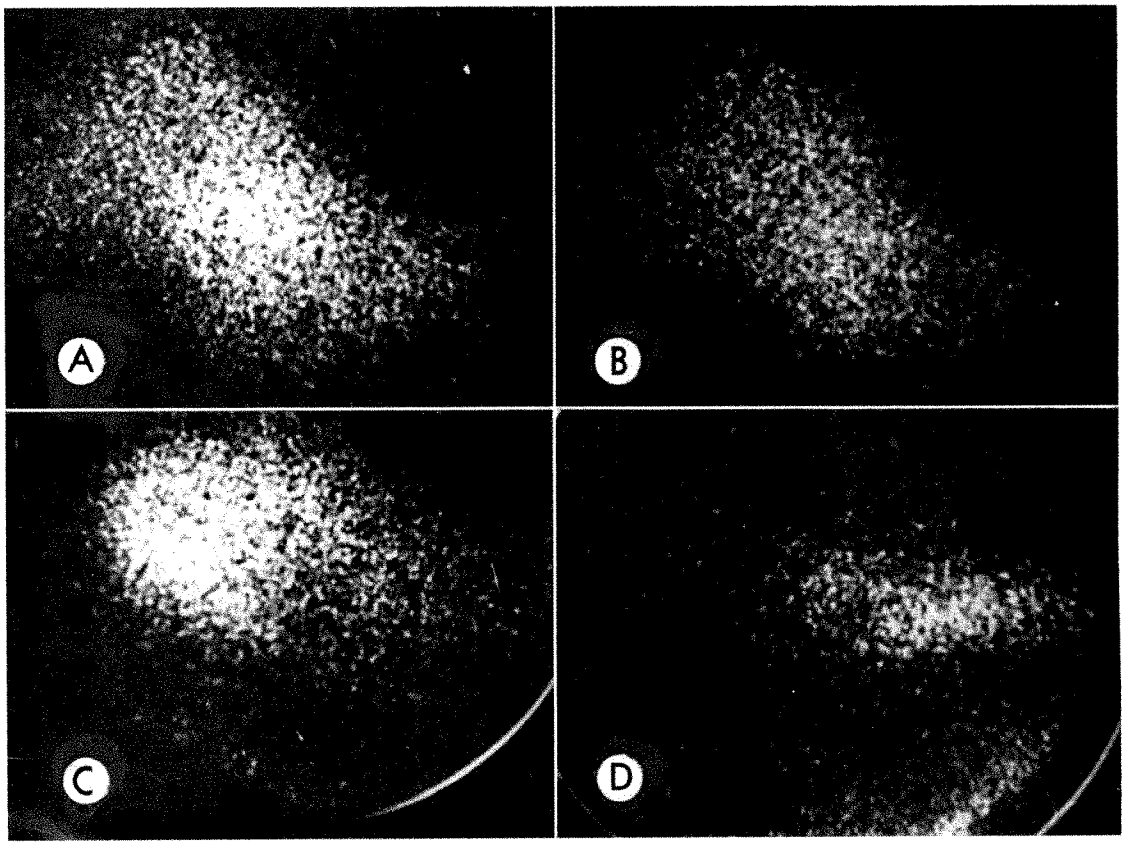



FIG. 8. A 61 year old female found to have multiple stones in the choledochus and both hepatic biliary ducts. Bilirubin 1.4 mg. per cent, and the $RBI^{131}$ retention 63 per cent. $RBI^{131}$ scans at 45 minutes (*A*) and $5\frac{1}{2}$ hours (*B*) show a difference in distribution, probably a result of a reduced flow of bile from the lower half of the right lobe, secondary to a partially obstructing stone. At no time was a dilated biliary tree seen, and there was good excretion into the intestine.



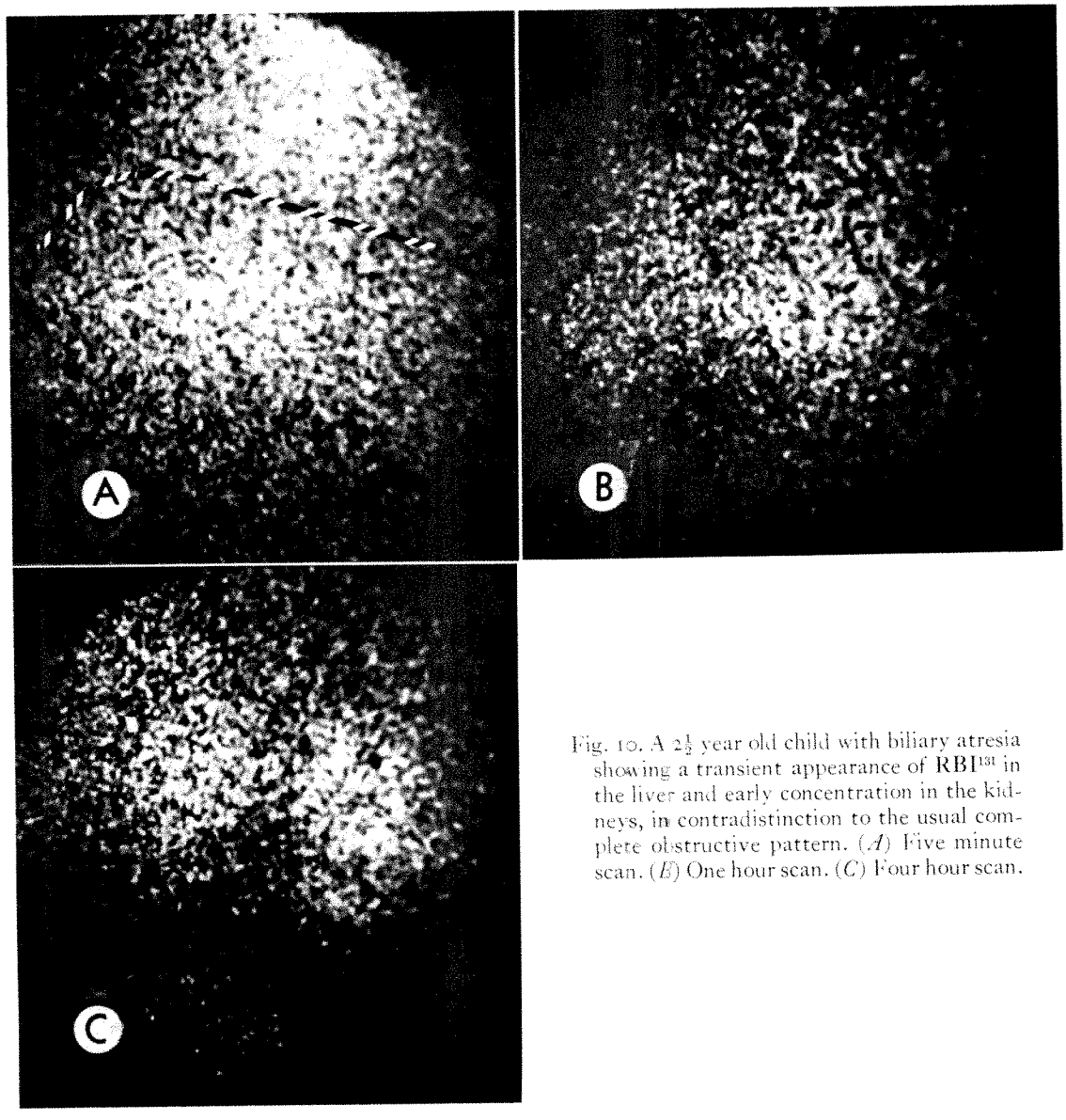



Fig. 10. A 2½ year old child with biliary atresia showing a transient appearance of $RBI^{131}$ in the liver and early concentration in the kidneys, in contradistinction to the usual complete obstructive pattern. (*A*) Five minute scan. (*B*) One hour scan. (*C*) Four hour scan.

←

Fig. 9. An 80 year old male with carcinoma of the biliary ducts and extension into the right lobe of the liver. Serial $RBI^{131}$ liver scans at 1 hour (*A*), 4 hours (*B*), and 24 hours (*C*), and abdomen scan at 24 hours (*D*) show a progressively increasing concentration of activity over the right lobe of the liver. This was due to a bile leak extending over the surface of the liver.

TABLE II

SUMMARY OF RESULTS OF $RBI^{131}$ RETENTION AND SERIAL LIVER SCANS

| $RBI^{131}$ Retention (%) | Excretion into Intestine in 24 hr. | Distribution of $RBI^{131}$ in the Liver | Diagnosis |
|---|---|---|---|
| 86–100 | +ve or −ve | Uniform | Polygonal cell disease |
| >80 | +ve | Uniform | Polygonal cell disease |
| ≤86 | −ve | Uniform or increased in region of porta hepatis | Complete extrahepatic obstruction |
| ≤80 | +ve | Increased in region of porta hepatis or nonuniform drainage | Partial extrahepatic obstruction |
| 45–90 | +ve or −ve | Persistent hypoactive areas with $RBI^{131}$ or $Au^{198}$ | Intrahepatic tumor |
| <80 | +ve | Uniform | Indeterminate group |

therapeutic agents in a child treated for neuroblastoma. All patients had 20 minute $RBI^{131}$ retentions of less than 80 per cent, and normal 4.5 minute $Au^{198}$ retentions. One patient with chlorpromazine jaundice did not show evidence of excretion of rose bengal into the gut within 24 hours of administration, whereas the other 3 did. Thus a distinction between intrahepatic cholestasis due to drug poisoning and a complete extrahepatic obstructive jaundice may be impossible to make, as exemplified by our single case, without recourse to careful history.

## SUMMARY

A radionuclide study is described for the evaluation of hepatobiliary disease. The study consists of $Au^{198}$ retention and liver scans, and radioiodinated rose bengal ($RBI^{131}$) retention and serial scans of the liver and abdomen. Twenty minute $RBI^{131}$ and 4.5 minute $Au^{198}$ retentions are given for normals, acute hepatitis, obstructive jaundice, fibrotic liver disease with and without jaundice, acute congestive heart failure, and liver containing tumor.

$RBI^{131}$ retentions greater than 86 per cent indicated polygonal cell disease, whether or not excretion into the intestine was detected (Table II). Complete extrahepatic obstruction was present when the $RBI^{131}$ retention was 86 per cent or less, and excretion into the intestine was not observed in the scans of the abdomen. Serial $RBI^{131}$ liver and abdomen scans may exhibit the presence of a dilated biliary tree or nonuniform drainage in the liver, and thus distinguish partial obstruction from hepatocellular disease. An indeterminate group remains which consists of a $RBI^{131}$ retention less than 80 per cent, excretion into the intestine, and a uniform distribution of activity in the liver with $Au^{198}$ and $RBI^{131}$.

Intra-abdominal bile spill can be diagnosed with serial $RBI^{131}$ scans. The findings in 2 cases of biliary atresia are described. A 3 month old infant with biliary atresia showed persistence of $RBI^{131}$ in the liver and no excretion into the intestine which is the usual picture in obstruction, whereas a $2\frac{1}{2}$ year old child showed only a transient appearance of $RBI^{131}$ in the area of the liver and a higher concentration in the kidneys at 1 and 4 hours.

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# RADIOISOTOPE LIVER SCANS IN PEDIATRICS*

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SEVERAL reports have described the radioisotope liver scanning as useful to demonstrate both liver structure and function. $Au^{198}$ colloid, $I^{131}$ rose bengal, and most recently $Tc^{99m}$ colloid have been used.[1,2,4,5,6,7,8,9,10,13,14,15,16,19]

It is the purpose of the author to illustrate, by specific examples, the usefulness of this method in various conditions encountered in pediatrics.

## MATERIAL

Within the past two years, we have performed approximately 250 liver scannings in children less than 16 years of age. Diagnoses have included primary and metastatic neoplasia, hepatitis, cirrhosis, biliary atresia, periductal inflammation, hemangiomata, eventration of the diaphragm and splenic mass.

## METHODS

Three $\mu$c/kg. body weight of $Au^{198}$ colloid or $I^{131}$ tagged rose bengal are injected intravenously. The former is administered from 1/2 to 3 hours prior to scanning, while the latter is injected 15 minutes prior to the start of the examination.

A Picker Magnascanner, equipped with a thallium activated 3×2 inch sodium iodide crystal, a 19 hole lead shielded collimator, and a pulse height analyzer, is used. The upper and lower "window" settings for both isotopes are 361 and 461 kev. respectively. The average count rate is 8,000 counts per minute. The scan speed is 30 cm. per minute and the line spacing is 0.3 cm. Probe high voltage of 531 volts is a function of both the isotope and the individual unit.

Following injection, the patient is placed in the supine position. Anatomic landmarks are recorded on the teledeltos paper scan and are: the xiphoid, the last palpable anterior ribs, and the superior iliac crest. Following completion of the examination, these landmarks are transferred to the simultaneously recorded photoscan.

Despite the young age of the patients, sedation is only rarely required.[17] Careful restraint and the constant attention of the staff oriented to the pediatric patient suffices, despite the length of the examination which may vary from 20 to 50 minutes per scanning.

The anterior scanning is routine. A lateral scanning with the right side towards the crystal, and/or a posterior scanning with the patient in the prone position are performed if indicated.

The following cases were selected to illustrate the appearance and clinical application of the liver scan in certain conditions:

### I. PRIMARY AND METASTATIC NEOPLASIA

The liver scan may demonstrate the existence and the extent of a liver neoplasm.

CASE I. R. B., a 4 year old boy entered The Children's Hospital Medical Center with a mass in the right abdomen. Physical examination revealed a firm and hard mass. A clinical diagnosis of Wilms' tumor was made. Intravenous pyelograms demonstrated that both kidneys were normal but were compressed by a mass in the region of the liver (Fig. 1, *A* and *B*). $Au^{198}$ scan of the liver disclosed that the right lobe was involved by an inhomogeneous replacement of normal liver tissue (Fig. 2). Moreover, this process seemed to extend beyond the midline and involve the left lobe of liver. At surgery, a juvenile hepatoma was found to arise in the right lobe of liver, extend beyond the midline and involve the left lobe.

CASE II. R. F., a 14 year old boy, was first seen at another hospital for a large right intra-

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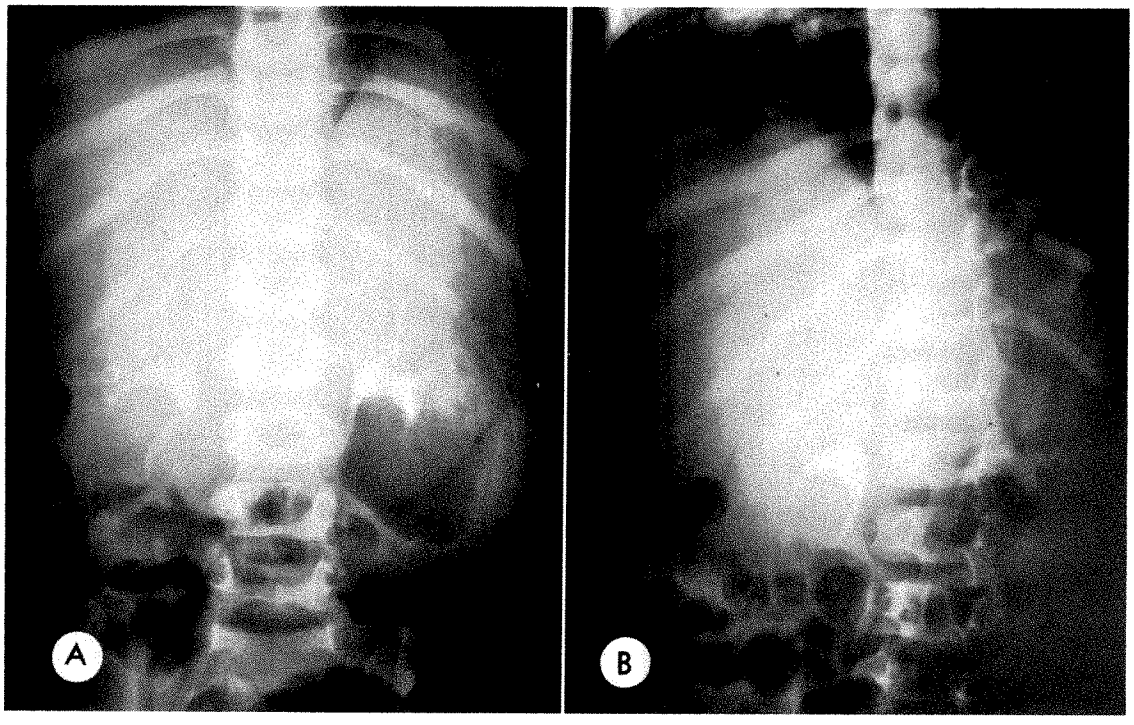



FIG. 1. Case 1. (*A*) Anterior and (*B*) oblique intravenous pyelograms. The kidneys are intrinsically normal but compressed by a mass in the region of the liver.

abdominal mass. Exploration revealed a grossly enlarged liver without abnormality in texture or appearance. Because of cardiac arrest at this time, a biopsy was not done. He was shortly thereafter transferred to The Children's Hospital Medical Center where an intravenous pyelogram revealed displacement of the right kidney and hepatic flexure of the colon by a right abdominal mass. A nodule in the left lower lung field was considered a metastasis from a probable hepatoma (Fig. 3). Liver scan revealed that the right lobe of liver was replaced by a process which spared a rim of functioning liver tissue. The left lobe of liver appeared to be uninvolved (Fig. 4).

Two needle aspirations revealed normal liver tissue. Before laparotomy could be undertaken, he died of a pulmonary embolism.

At postmortem examination, the right lobe of liver was replaced by a juvenile hepatoma which arose deep within the liver substance. This grew towards the surface of the liver but spared a 3.5 cm. rim of liver tissue circumferentially. The left lobe of liver was not involved by tumor.

It would seem that the rim of normal liver

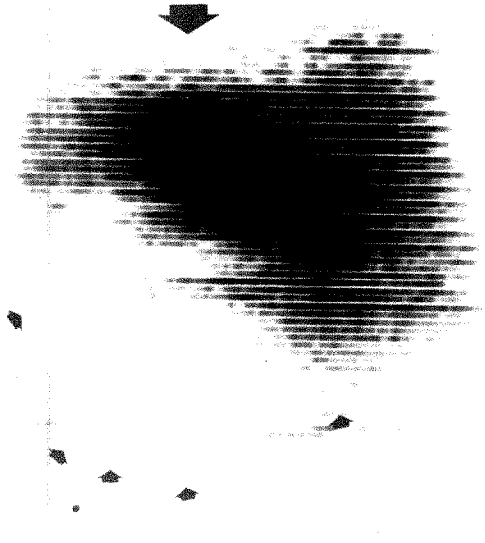


FIG. 2. Case 1. $Au^{198}$ liver scan. There is a nodular infiltrative process replacing much of the right lobe of liver with extension beyond the midline and involving the left lobe. A diagnosis of hepatoma was made and confirmed at surgery. The small arrows demarcate the palpable mass in the right upper quadrant. The large arrow indicates the xiphoid.



FIG. 3. Case II. Intravenous pyelogram and barium enema examination. There is noted to be displacement of the kidney and hepatic flexure of the colon inferiorly by a mass in the region of the liver. A nodule is noted in the left lower lung field.

tissue spared from the neoplastic process accounted for the appearance on the scan, the failure to note neoplasia at the first surgical procedure, and the two normal liver biopsies which were apparently too superficial.

CASE III. S. B., a 12 year old girl, entered The Children's Hospital Medical Center with jaundice and hepatosplenomegaly. At the age of 3 years, she had had a liver biopsy consistent with "portal cirrhosis" at a time when she was noted to have jaundice, anorexia and epistaxis. Three years later, a portal-caval shunt was performed because of gastrointestinal bleeding. Thereafter, she was clinically well except for icterus until her present admission when she developed enlargement of the liver. On physical examination, the liver was nodular to palpation. At this time she also complained of right pleuritic pain and weight loss.

A liver scan revealed diffuse replacement of the left lobe of liver with a nodular pattern of infiltration in the right lobe (Fig. 5). Lung metastases were noted at this time and she died shortly thereafter.

At postmortem examination, she was found to have "hepatocellular carcinoma" consisting of large nodules, mostly located in the left lobe of liver but with extension into the right lobe.

CASE IV. J. L., an 8 year old girl, was operated at another hospital for a left Wilms' tumor. Later she had developed a mass in the right upper quadrant. Liver scan (Fig. 6) revealed replacement of the right lobe of liver with relative sparing of the left lobe. At surgery, total replacement of the right lobe of liver by metastatic tumor was found. The left lobe was free of disease.

CASE V. A. H., an 11 year old girl, was referred from another hospital after surgery for inoperable dysgerminoma which was metastatic to pelvic and retroperitoneal lymph nodes. No liver involvement was noted at the time of surgery and none was suspected clinically. $Au^{198}$ liver scan was normal (Fig. 7*A*). Following irradiation to the pelvis and abdomen, the

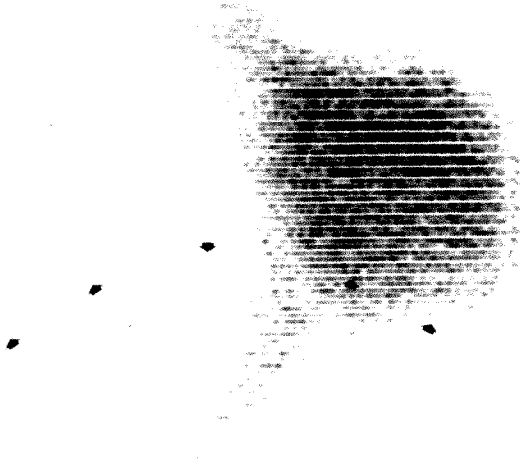


FIG. 4. Case II. $Au^{198}$ liver scan. There is noted to be replacement of normal functioning liver tissue in the right lobe. There is a rim of functioning tissue circumferentially. The left lobe is uninvolved. At postmortem examination a hepatoma involving the right lobe was confirmed. The arrows demarcate the last palpable anterior ribs.

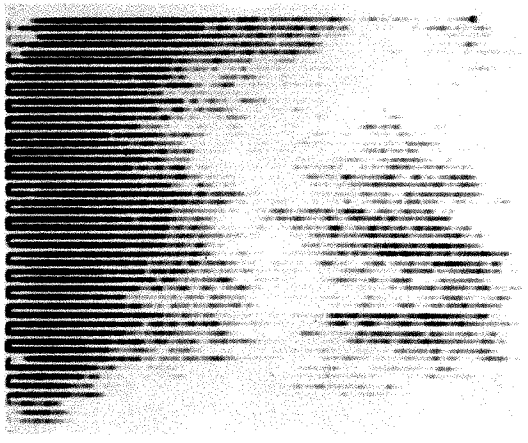


Fig. 5. Case III. $Au^{198}$ liver scan. This shows a diffuse replacement of left lobe of liver by a nodular process with infiltration in the right lobe. At postmortem examination primary liver carcinoma was found to arise in the left lobe and to involve the right lobe.

masses regressed. She was well for the next 3 months when she developed recurrence in the pelvis and a right upper quadrant mass. Liver scan at this time showed a defect in the superior border of the liver (Fig. 7*B*). She died within a few weeks of this scan. At postmortem examination, a metastatic dysgerminoma was found as a well circumscribed mass involving the right lobe of liver.

Case VI. J. S., a 4 year old boy, was operated for a left retroperitoneal embryoma. At surgery,

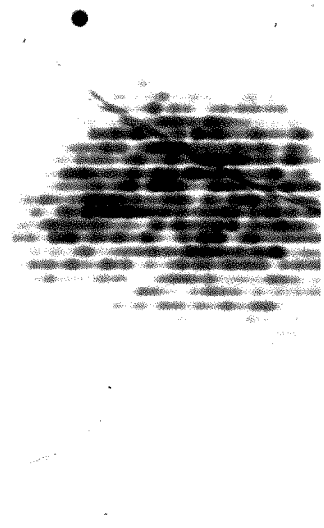


Fig. 6. Case IV. $Au^{198}$ liver scan. There is noted to be diffuse replacement of the right lobe of liver by metastatic Wilms' tumor with relative sparing of the left lobe. This was confirmed at surgery. The dotted line demarcates the palpable mass in the right upper quadrant.

the liver appeared to be free of tumor. He was well for the next 4 months when he developed right upper quadrant pain and fever. Chest roentgenogram, which previously had been within normal limits, now showed an elevated right hemidiaphragm.

Liver scan revealed a defect in the superior border of the liver with compression of liver

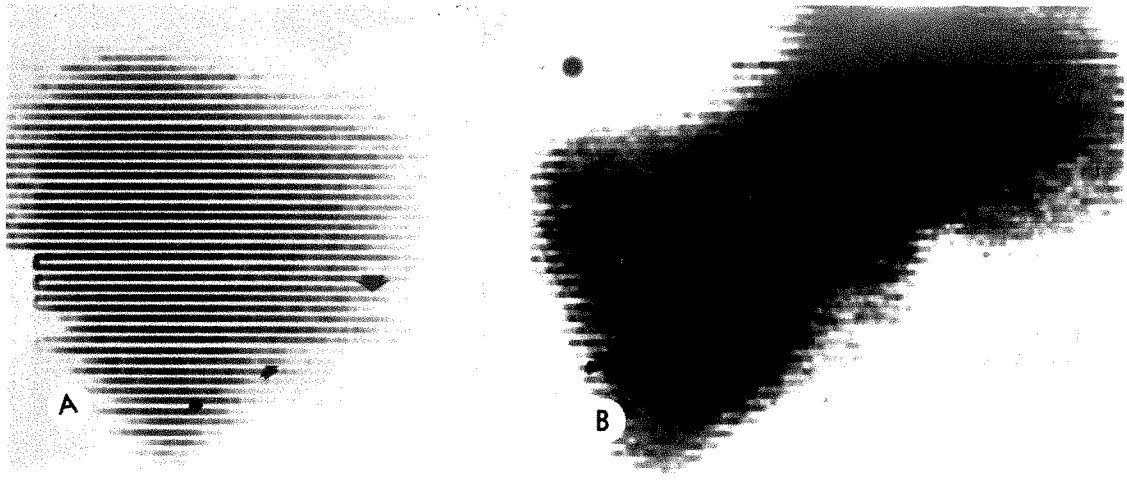



Fig. 7. Case V. (*A*) $Au^{198}$ liver scan. This appears to be within normal limits. The large arrow indicates the xiphoid; the small arrows demarcate the last palpable right anterior rib. (*B*) Three months later, there is noted to be replacement of liver tissue in the superior portion of the right lobe. At postmortem examination, metastatic dysgerminoma was found in this area.

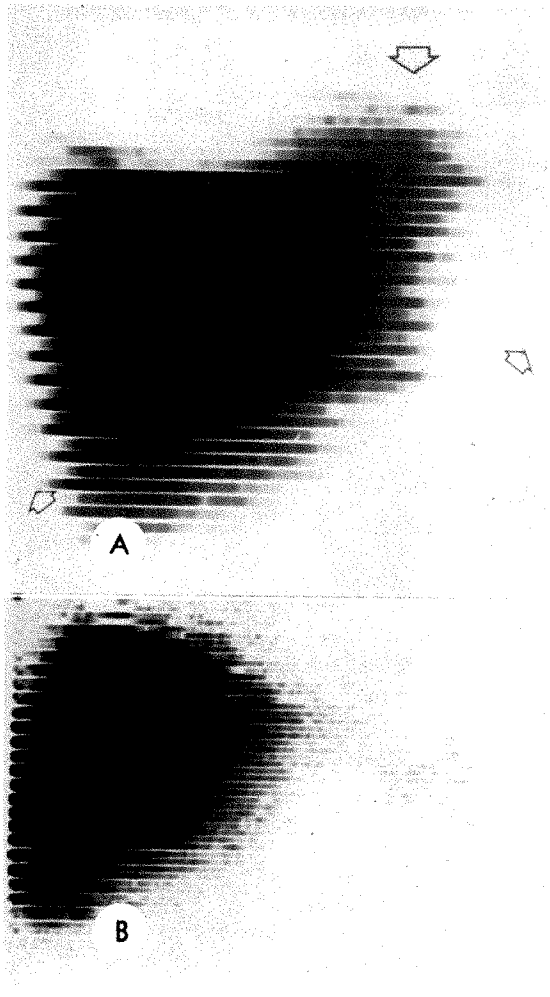



FIG. 8. Case VI. (*A*) $Au^{198}$ liver scan. There is noted to be a defect in the superior border of the liver. Liver tissue is compressed inferiorly and medially. At surgery, metastatic retroperitoneal embryoma was found in this location. The arrows demarcate the xiphoid and last palpable anterior ribs. (*B*) Following combined irradiation and chemotherapy, the defect in the liver is noted to have regressed. The liver has now assumed a more normal configuration.

tissue inferiorly and medially (Fig. 8*A*). At surgery, a mass was found to be located between the liver and right hemidiaphragm and to have invaded the superior border of the liver. Following combined irradiation and chemotherapy, we were able to demonstrate that the defect had regressed (Fig. 8*B*).

CASE VII. M.P. was 2 years of age when seen first at another hospital because of a mass in the right upper quadrant of the abdomen. At surgery, a right adrenal neuroblastoma was found to be inoperable and he was transferred to The Children's Hospital Medical Center. On admission, a liver scan (Fig. 9*A*) revealed a defect due to liver invasion by the neuroblastoma of the right adrenal. Following combined therapy with irradiation and chemotherapy, the right upper quadrant mass diminished and the liver scan revealed disappearance of the defect (Fig. 9*B*).

CASE VIII. P.B., a 14 year old boy, was seen first for Hodgkin's disease involving the cervical, supraclavicular and mediastinal lymph nodes. No evidence of dissemination was present at diagnosis and abdominal lymphangiography at this time was within normal limits. Following therapy, he was well for the next year when he developed symptoms of generalized disease and was found to have involvement of the retroperitoneal lymph nodes by repeat lymphangiography. No clinical evidence of hepatic involvement was present and the

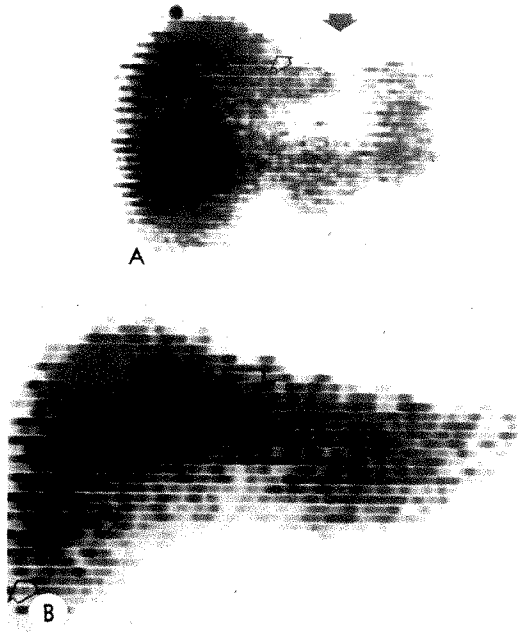



FIG. 9. Case VII. (*A*) $Au^{198}$ liver scan. There is noted to be a defect at the junction of right and left lobes of liver due to invasion by right suprarenal neuroblastoma. The black arrow indicates the xiphoid; the white arrow demarcates the last palpable right anterior rib. (*B*) Disappearance of the defect is shown 10 weeks later, following combined radiation and chemotherapy.

liver function tests were negative. A liver scan was considered unremarkable (Fig. 10*A*). Following therapy, he remained well for the next 3 months when he developed jaundice and hepatosplenomegaly. A repeat liver scan now revealed diffuse hepatic involvement and marked enlargement of the spleen (Fig. 10*B*). He died of dissemination within a few weeks. At postmortem examination, the liver and spleen were grossly enlarged and showed typical infiltration by Hodgkin's granuloma.

Case IX. Patient S.B. was a 6 year old boy when treated first for Hodgkin's disease of the supraclavicular and mediastinal lymph nodes. He remained well for the next 2 years when he developed symptoms of generalized disease. Lymphangiography revealed involvement of the retroperitoneal lymph nodes.

A liver scan showed possible involvement by a localized nodular lesion in the region of the porta hepatis (Fig. 11*A*). His symptoms subsided on further therapy and he was well for the next 6 months. He then developed signs of liver involvement with jaundice and clinical enlargement of the liver. A repeat liver scan revealed diffuse infiltration of the liver (Fig. 11*B*).

Following irradiation of the liver, the process seemed to improve as demonstrated by the liver scan (Fig. 11*C*). In addition, his jaundice diminished, his liver mass regressed, and his liver function tests reverted to more normal levels. Nevertheless, he died within a few weeks of disseminated disease. At postmortem examination there was diffuse involvement of the liver by Hodgkin's disease.

Case X. K.K. was a 6 year old girl when admitted to The Children's Hospital Medical Center with mediastinal lymphosarcoma. During her clinical course, a mass was noted in the right abdomen. At this time, liver scan revealed involvement of the liver by probable lymphosarcoma (Fig. 12*A*). Following irradiation, the mass clinically regressed and the liver scan documented this regression (Fig. 12*B*). She died within a few weeks of the last scan and at postmortem examination there was a necrotic tumor nodule in the area of the liver as demonstrated by the liver scan.

Case XI. $Au^{198}$ scan of a $5\frac{1}{2}$ year old boy with acute lymphocytic leukemia, who developed liver enlargement during his clinical course, revealed this enlargement with inhomogeneous uptake due to diffuse infiltration (Fig. 13). Following irradiation to the liver, the liver diminished in size, thus giving presumptive evidence of leukemic involvement.

## II. INFLAMMATION

The liver scan is useful not only to demonstrate abnormality of distribution of the isotope in inflammatory processes, but also to evaluate progression or healing of the disease at a later time.

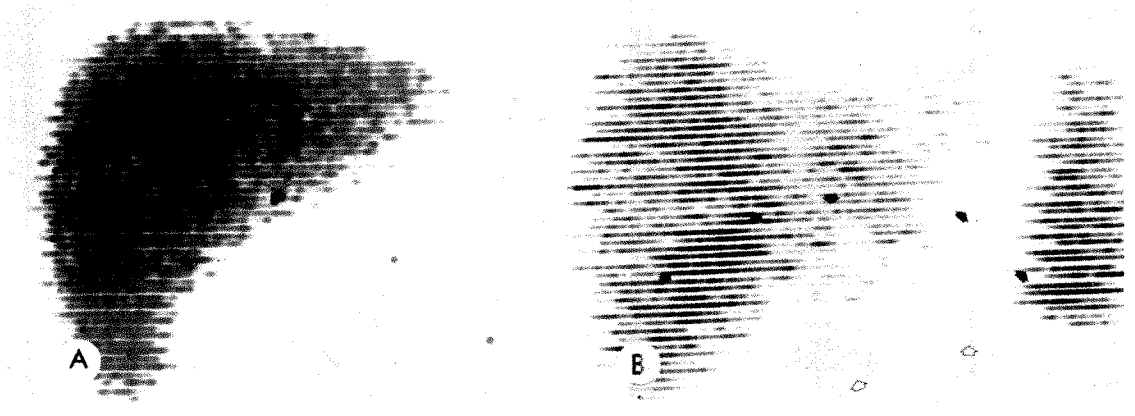



Fig. 10. Case VIII. (*A*) $Au^{198}$ liver scan is considered to be within normal limits in this patient with Hodgkin's disease. (*B*) Three months later, there is noted to be diffuse irregularity in uptake of isotope throughout the liver and marked enlargement of the spleen. At postmortem examination, there was Hodgkin's infiltration of both liver and spleen. The black arrows demarcate the last palpable ribs; the white arrows demarcate the palpable edge of the enlarged liver.

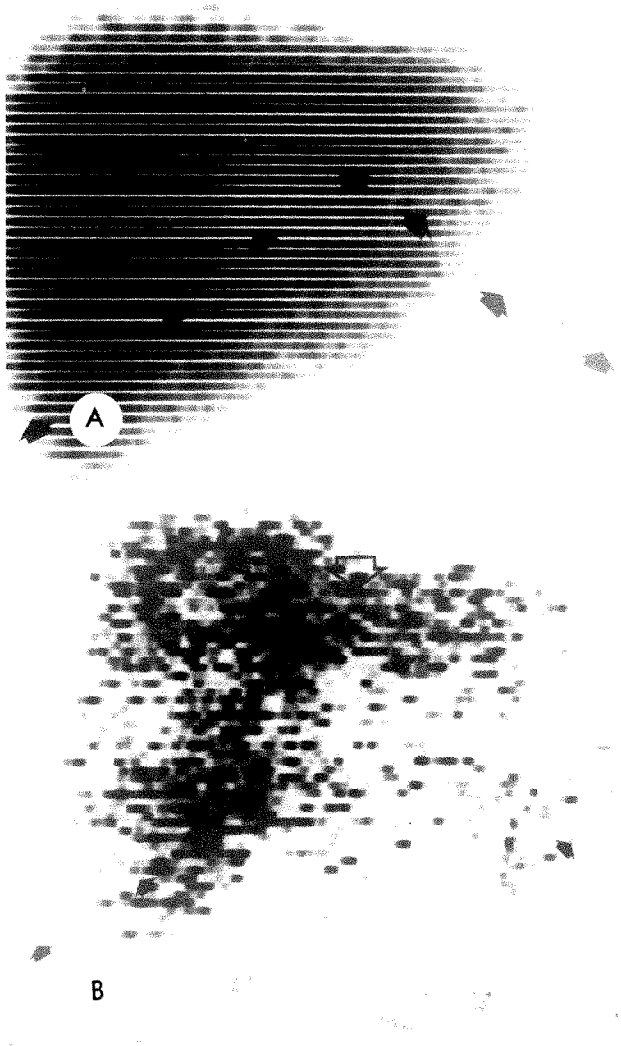



FIG. 11. Case IX. (*A*) $Au^{198}$ scan shows an area of diminished uptake in the region of the gallbladder which is considered to be within normal limits. The black arrows demarcate the last palpable anterior ribs. (*B*) Repeat $Au^{198}$ scan at 6 months shows diffuse infiltration of the liver and diminished uptake of isotope. The black arrows demarcate the last palpable anterior ribs; the white arrows demarcate the palpable edge of the enlarged liver. (*C*) Following irradiation of the liver, the process has improved as noted by increased isotope uptake.

CASE XII. G.S., a 17 year old boy, had had a blood transfusion at the time of pulmonary surgery for metastatic Ewing's sarcoma. He was well for the next 4 months when he developed dark urine and jaundice. His liver function tests revealed evidence of parenchymal disease. A diagnosis of serum hepatitis was made. A liver scan at this time revealed diffuse inhomogeneous distribution of $Au^{198}$ colloid (Fig. 14*A*). He improved over the next few months and a repeat liver scan at 3 months reverted to normal (Fig. 14*B*). His liver function tests were now also within normal limits.

CASE XIII. J.P., a $13\frac{1}{2}$ year old boy, was well until 3 days prior to admission when he developed fever of 101°F., malaise, jaundice and dark urine. On examination his spleen was palpable 10 cm. below the left costal margin. The total bilirubin was 7.7 mg. per cent and SGOT 570. An upper gastrointestinal series and intravenous pyelogram demonstrated splenic enlargement without hepatomegaly (Fig. 15, *A* and *B*). Liver scan revealed diffuse abnormality of uptake in the liver and enlargement of the spleen (Fig. 15*C*). A diagnosis of hepatitis was made and confirmed by biopsy which revealed "subsiding hepatitis."

### III. LIVER FUNCTION ($I^{131}$ ROSE BENGAL)

$I^{131}$ rose bengal is useful to demonstrate liver function.[2,5,6,8]

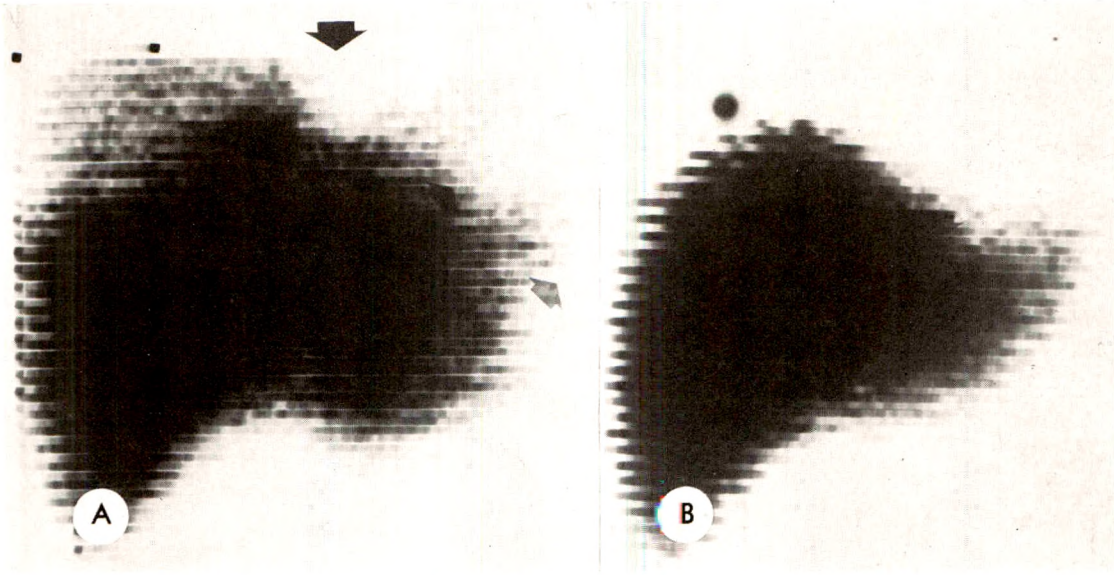



Fig. 12. Case x. (*A*) $Au^{198}$ liver scan. There is a defect in the liver due to lymphosarcoma. The arrows demarcate the xiphoid and the last palpable anterior ribs. (*B*) Following irradiation the process has regressed.

Case xiv. B.C. was $1\frac{1}{2}$ years old when he had surgery for a hepatoma involving the right lobe of liver. The left lobe of liver was found to be uninvolved and the right lobe was resected. The common and left hepatic ducts were found to be patent. Six days later he developed an elevated bilirubin of 3.8 mg. per cent mostly in the direct form. Abdominal distention was present. There was also an elevated temperature and increase in the white blood cell count. Shortly thereafter, a right upper quadrant bowel abscess was drained. Within a few days, his bilirubin continued to rise to as high as 10 mg. per cent with primarily a direct fraction and there was no evidence of bile in the stool.

Two weeks later, $Au^{198}$ liver scan revealed the remaining left lobe of liver to have assumed much of the anatomic position of the right lobe. The left lobe of liver, by scan technique, was unremarkable (Fig. 16, *A* and *B*). Following the injection of $I^{131}$ rose bengal, excretion into the bowel was noted (Fig. 16, *C* and *D*). It thus appeared that the process was subsiding and further surgery was possibly not necessary at this time.

A diagnosis of periductal inflammation was made. Within the next few weeks his clinical signs abated and the laboratory tests returned to normal. He is well at 1 year.

### IV. LIVER POSITION

Demonstration of the position of the normal liver can at times be quite helpful in ruling out abnormal masses.



Fig. 13. Case xi. $Au^{198}$ liver scan of a $5\frac{1}{2}$ year old boy with acute leukemia. The liver is noted to be grossly enlarged and there is diffuse inhomogeneous uptake of the isotope throughout. The arrows demarcate the last palpable anterior ribs.

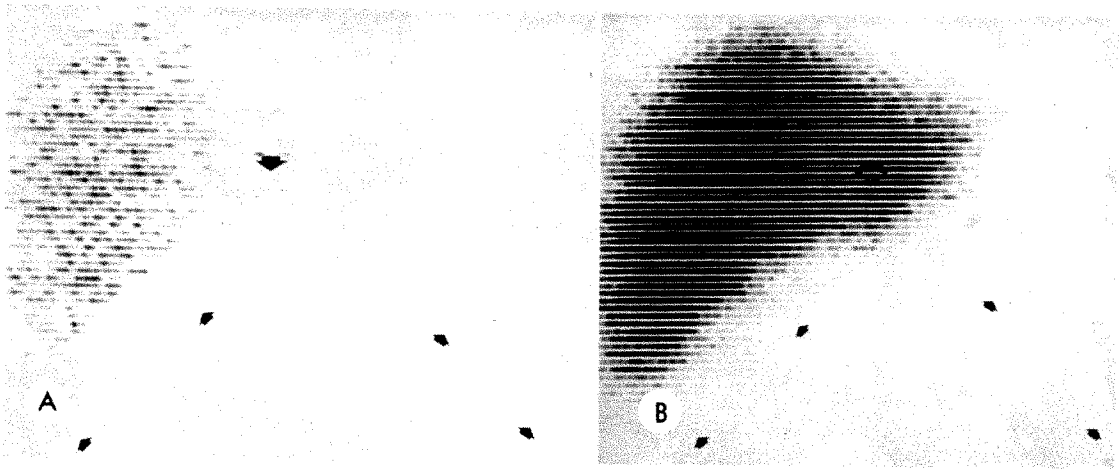
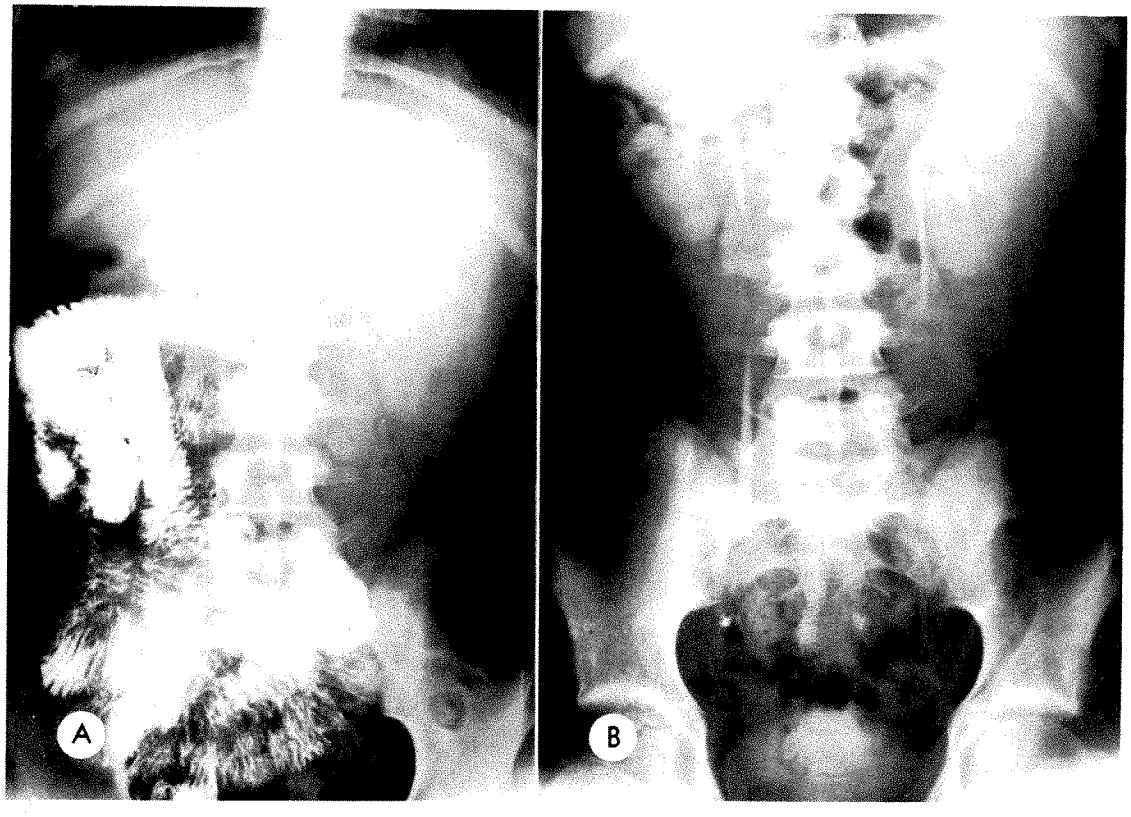



FIG. 14. Case XII. (*A*) $Au^{198}$ scan of the liver shows diffuse inhomogeneous distribution of the isotope. The arrows demarcate the xiphoid and the last palpable anterior ribs. (*B*) At 3 months, the scan has reverted to normal.



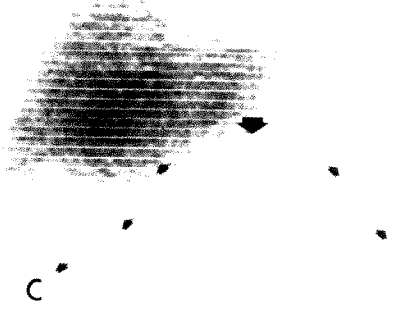




FIG. 15. Case XIII. There is a mass in the left abdomen in the region of the spleen on both (*A*) the upper gastrointestional roentgenogram and (*B*) on the intravenous pyelogram. (*C*) The liver scan of this patient revealed diffuse abnormality of uptake in the liver and enlargement of the spleen. On biopsy, subsiding hepatitis was found. The arrows demarcate the xiphoid and the last palpable anterior ribs.

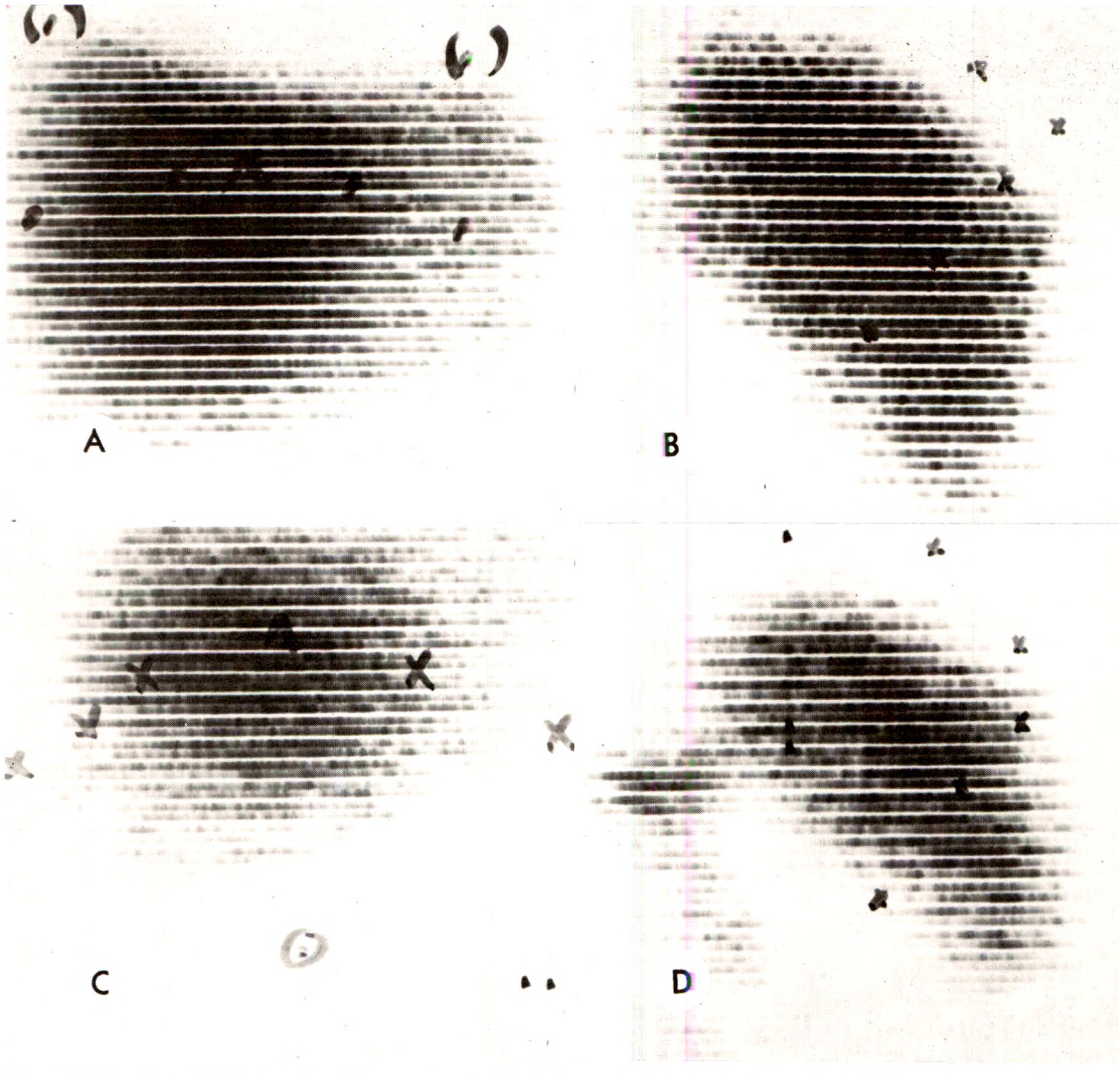



Fig. 16. Case XIV. $Au^{198}$ scans in (*A*) anterior and (*B*) lateral projections show the left lobe of liver to have assumed much of the anatomic position of the right lobe. The marks signify the last palpable anterior ribs and the xiphoid on the anterior scan; on the lateral scan, the marks signify the last palpable rib on the right and the posterior abdominal wall. (*C* and *D*) Following injection of $I^{131}$ rose bengal, there is noted to be excretion into the bowel at 4 hours.

Case XV. L.A.C. was 9 months of age when admitted to The Children's Hospital Medical Center with fever of 101°F. and vomiting. Routine chest roentgenogram revealed an elevated left hemidiaphragm and a barium swallow roentgenogram demonstrated a mass between the stomach and diaphragm (Fig. 17, *A* and *B*). $Au^{198}$ liver scan revealed this mass to be a normal left lobe of liver (Fig. 17, *C* and *D*). Surgery, which might have been undertaken to evaluate this "mass," was therefore not performed.

### V. Enlargement of the Spleen

At the time of the examination of the liver by $Au^{198}$ colloid, we have been able, in certain cases, to evaluate the presence and structure of the spleen.

Case XVI. M.O. was 14 years of age when she entered The Children's Hospital Medical Center with a mass in the left abdomen. She was entirely asymptomatic and the only laboratory abnormality was a platelet count of 74,000. An upper gastrointestinal series and intravenous

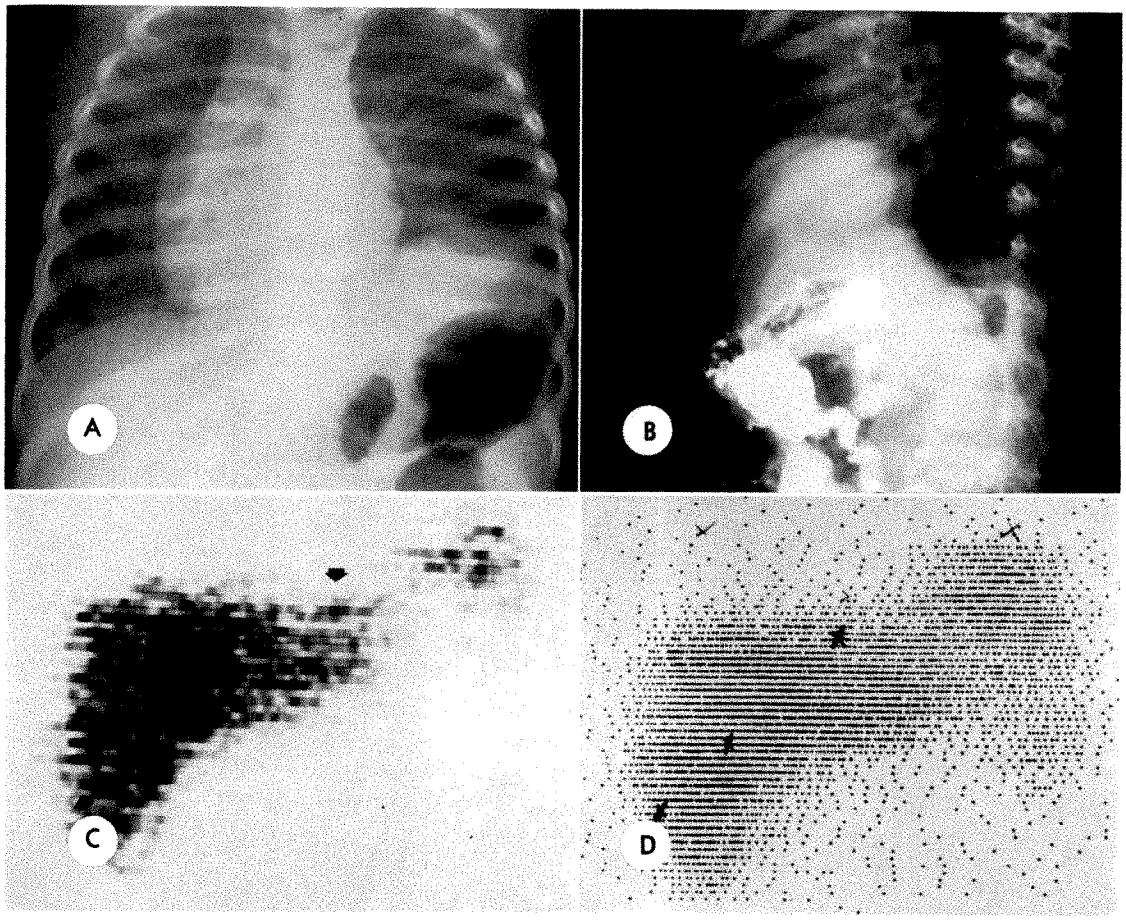



FIG. 17. Case XV. (*A* and *B*) Roentgenograms of the chest and barium swallow, reveal the left hemidiaphragm to be elevated. There is a soft tissue density between barium outlined stomach and the diaphragm. (*C*) $Au^{198}$ photoscan and (*D*) simultaneously obtained teledeltos paper scan reveal the "mass" under the left hemidiaphragm to be normal left lobe of liver. The arrow demarcates the xiphoid on the photoscan; on the paper scan, the last palpable rib on the right is demarcated.

pyelogram revealed displacement by a mass consistent with an enlarged spleen (Fig. 18, *A* and *B*). The clinical diagnosis of splenic cyst was made.

$Au^{198}$ scan showed the liver to be intrinsically normal but to exhibit a concave defect along the medial border (Fig. 18*C*). A scan over the mass in the left abdomen demonstrated a markedly enlarged spleen with multiple filling defects (Fig. 18*D*). A diagnosis of splenic cyst seemed confirmed.

At surgery, the spleen was involved by a solitary large cyst whose walls showed trabeculation. This latter finding would explain the appearance on the scan where the possibility of multiple cysts was raised.

CASE XVII. C.K. was 9 years of age when she sustained abdominal trauma. Physical examination at that time revealed no evidence of intra-abdominal mass. Over the next 6 months, she complained of intermittent epigastric pain but had no associated symptoms or signs. Six months later, on routine physical examination, a mass was palpated in the left upper quadrant and she was admitted to The Children's Hospital Medical Center.

On admission, physical examination was within normal limits except for a soft firm rounded nontender mass in the left upper quadrant. Vital signs were within normal limits and laboratory examinations were not remarkable.

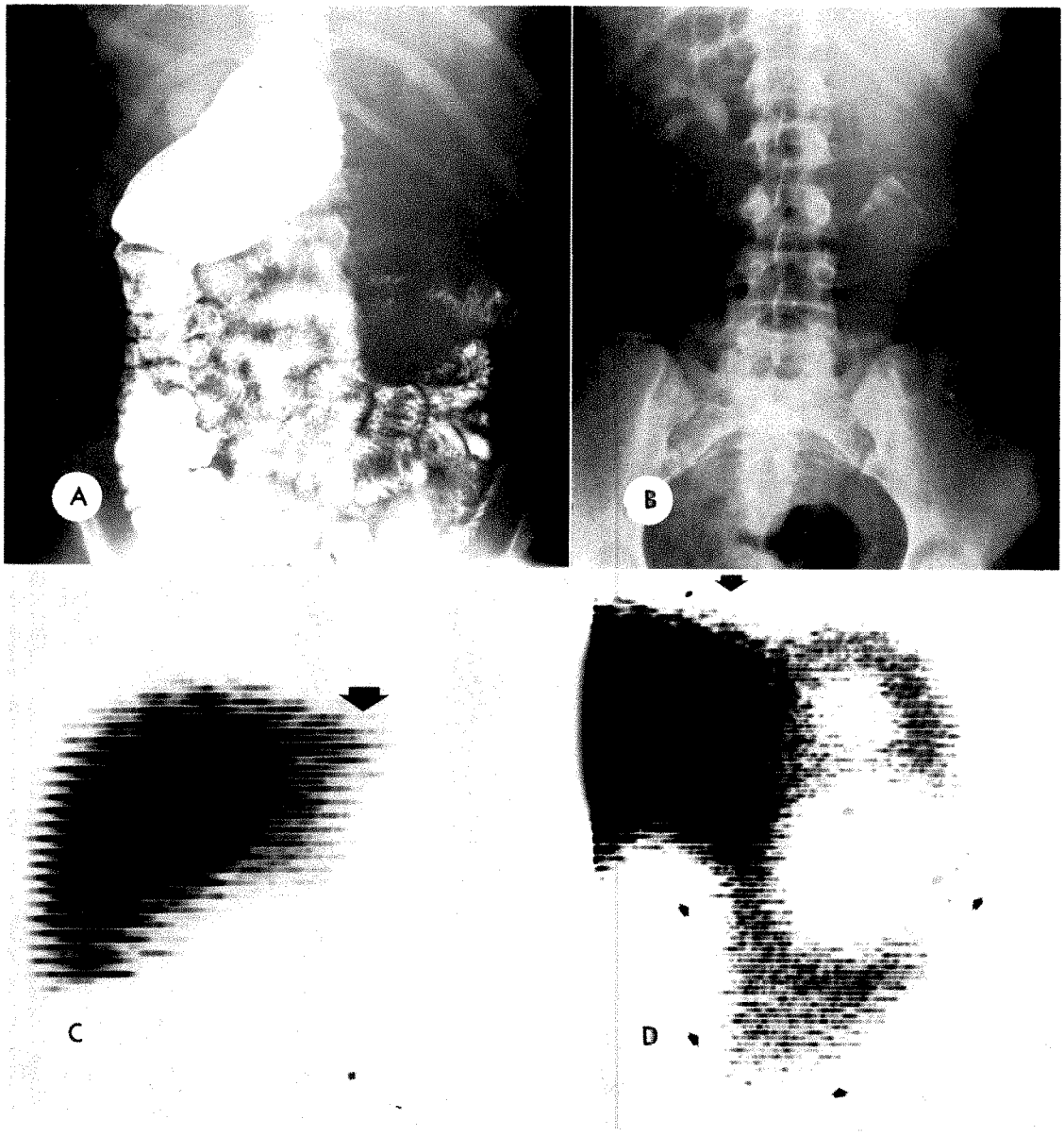



FIG. 18. Case XVI. (*A*) Gastrointestinal series and (*B*) intravenous pyelogram show the spleen to be grossly enlarged with displacement of the stomach and small bowel medially and the kidney inferiorly. (*C*) $Au^{198}$ scan shows the liver to be intrinsically normal. There is a slight concave defect along its medial border. The arrow demarcates the xiphoid. (*D*) The scan over the left abdominal mass shows the spleen to be markedly enlarged. There are multiple filling defects throughout. The diagnosis of splenic cyst was confirmed at surgery. The large black arrow demarcates the xiphoid; the light arrows demarcate the last palpable anterior rib on the left; the small black arrows demarcate the palpable mass in the left abdomen.

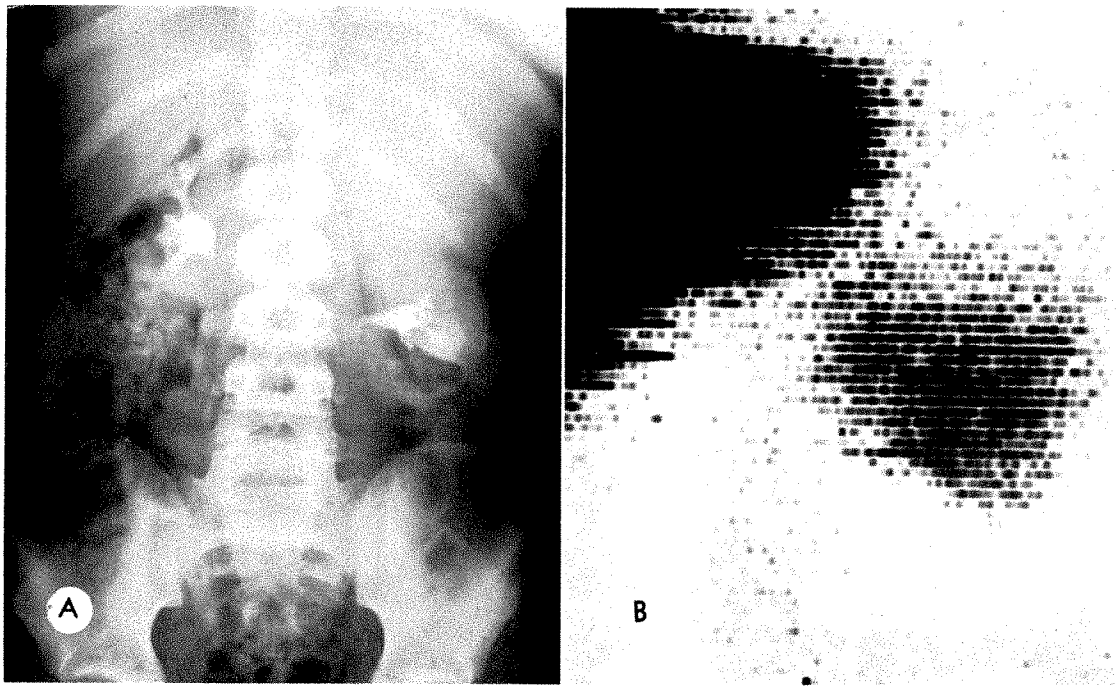



FIG. 19. Case XVII. (*A*) Intravenous pyelogram demonstrates a mass in the left upper quadrant which displaces the left kidney inferiorly and medially. (*B*) A scan over the left abdomen after the injection of $Au^{198}$ colloid demonstrates functioning splenic tissue which is displaced inferiorly by a nodular process in the upper portion of the spleen. At surgery, a hemorrhagic splenic cyst was found.

Intravenous pyelogram showed evidence of a left upper quadrant mass which displaced the left kidney medially and inferiorly (Fig. 19*A*). $Au^{198}$ colloid scan of the liver showed no abnormality. As part of the same examination, a scan over the left upper quadrant revealed splenic tissue to concentrate the $Au^{198}$ colloid. The spleen was noted to be displaced inferiorly by a lobulated process in its upper portion (Fig. 19*B*).

A diagnosis of splenic cyst was made. At operation, a large hemorrhagic cyst in the superior portion of the spleen was found.

CASE XVIII. P.H. was 20 months of age when she entered with lethargy and fever of 104°F. Physical examination revealed her spleen to be enlarged to the pelvic brim. Her hematocrit was 19 per cent, her platelet count was 79,000 and her reticulocyte count was 4.6 per cent. Thymol flocculation was 4 plus, thymol turbidity 2 plus, cephalin flocculation 2 plus. All other laboratory tests were unremarkable.

$Au^{198}$ liver scan showed no abnormality (Fig. 20*A*). A scan over the left abdomen demonstrated a markedly enlarged spleen which concentrated the $Au^{198}$ colloid throughout (Fig. 20*B*). No evidence of splenic cyst was noted.

At surgery, liver biopsy revealed slight increase in portal fibrous tissue and periportal fatty metamorphosis of liver cells. The spleen was enlarged and on section revealed infiltration by a process related to histiocytosis.

## DISCUSSION

We have found the radioisotope scan useful to demonstrate both the existence and the extent of involvement of the liver by primary or metastatic neoplasm. Such an examination can be employed to evaluate the results of therapy and the regression or progression of the disease as noted in the cases cited. Thus, it is most helpful as a baseline liver scan in all patients with malignancy to better estimate any changes in the scan in the patient's future clinical course.

Others have noted the usefulness of isotopes in the evaluation of the spleen.[16,17,18,19,20,21,22] We have found that

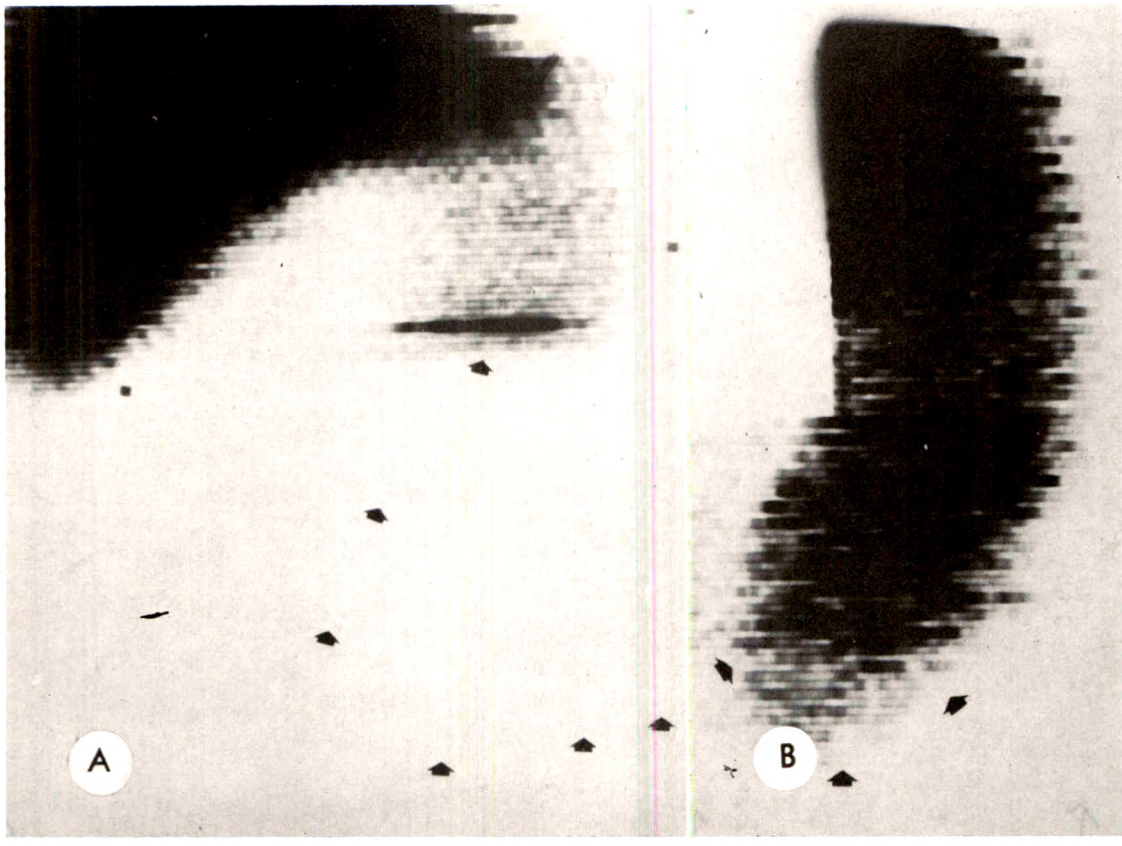



FIG. 20. Case XVIII. (*A*) $Au^{198}$ scan of the liver shows no abnormality. The arrows demarcate the palpable edge of the left abdominal mass. (*B*) A scan over the left abdominal mass reveals the spleen to be markedly enlarged. The isotope is distributed evenly throughout without evidence of intrinsic filling defect.

during scanning of the liver, we may at times also be able to evaluate the spleen. By adjusting the voltage on the scanner and calibrating to the count rates over the splenic area, a satisfactory scan of the spleen may be obtained as exemplified in the 2 cases described. The liver scan is obtained prior to the splenic scanning by the usual method of calibration to the count rate over the liver tissue.

## SUMMARY

Selected cases from approximately 250 liver scans in children less than 16 years of age are described. Using this procedure, we have attempted to demonstrate in certain disease states the appearance of the scan, its correlation with either operative or postmortem findings, and its value in the clinical management of some patients.

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I should like to acknowledge with thanks the permission to Drs. Nathan Brody and William Gage to reproduce the roentgenograms used in Figure 18, *A* and *B*.

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# ELIMINATION OF MAGNIFICATION ERRORS IN COMPARISON ROENTGENOGRAMS USED WITH RADIOISOTOPE SCANS*

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CARDIAC scanning with technetium 99m was begun at this Medical Center in January 1965 and later modified to the method described by Witcofski and Bolliger.[4] Both technetium 99m as the pertechnetate and technetium 99m labeled albumin have been used for cardiac scanning. Both methods have been found to be superior to the use of RISA (iodine 131) due to the larger doses which can safely be given.[3]

The use of technetium 99m albumin demonstrates a marked increase in the resolution of the cardiac blood pool.[1,3] The limiting factor at this point is the roentgenogram that is necessary for comparison of cardiac size. It is necessary that the cardiac silhouette as seen on the roentgenogram be as nearly life-size as possible. This is the size which the blood pool on cardiac scanning will demonstrate.

The method used by Wagner *et al.*[2] consistently produced roentgenograms in which the cardiac silhouette was magnified even with a 6 foot target film distance (Fig. 1, *A* and *B*). These roentgenograms usually produced a reasonably close correlation in size of the transverse diameter of the heart at the exact midplane of the roentgen-ray exposure field. The vertical diameter was always much greater than the vascular pool as seen on the cardiac scan.

The problem of excessive magnification

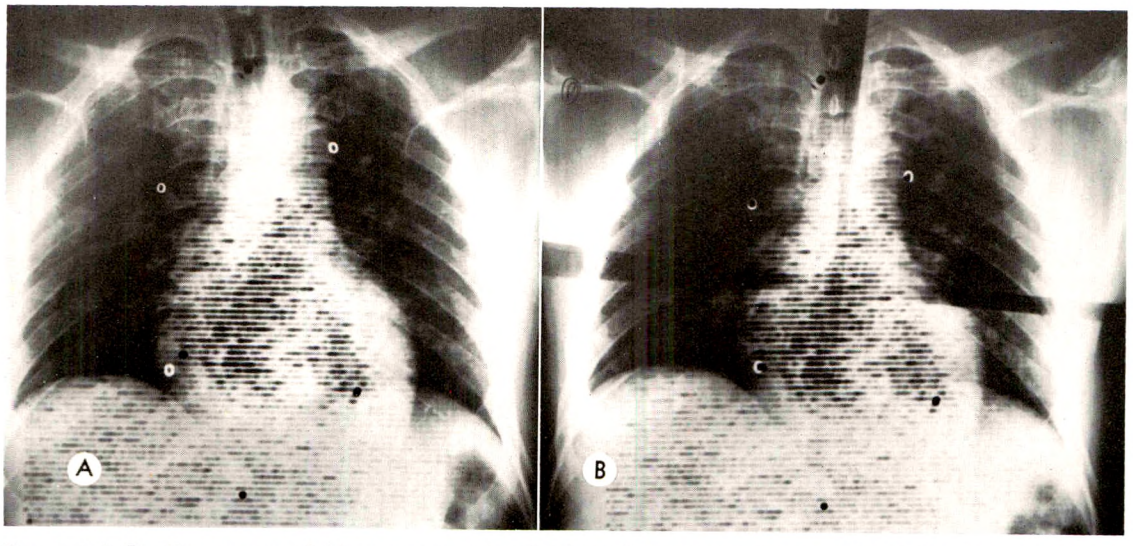



FIG. 1. (*A*) Cardiac scan overlaid on a conventional 6 foot chest roentgenogram. Notice that the 4 quadrant markers (the small black dots) do not coincide, but are actually within the 4 quadrant markers identified on the chest roentgenogram (the white circles). (*B*) The same cardiac scan of *A*, overlying a chest roentgenogram taken utilizing the 4 quadrant technique. Notice that the black scan markers and the white roentgen-ray markers coincide. Comparison 6 foot roentgenograms were made on the first 20 patients done utilizing the 4 quadrant technique. The average vertical dimension measured at the markers was 13 per cent greater and the average transverse dimension was 10 per cent greater on the routine 6 foot roentgenogram as compared to the actual scan markers.

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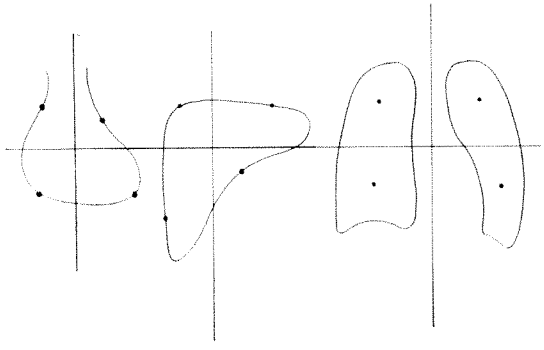


FIG. 2. Line drawings illustrating the segmentation of activity over the heart, liver and lungs into 4 quadrants. The black dots represent the areas where photo markers are placed on the scan and the roentgen-ray beam is centered when the exposure of the quadrant is made.

of the roentgenographic silhouette as compared to the scan dimensions also creates considerable error when an attempt is made to compare iodine 131 labeled macroaggregate albumin lung scans, and colloidal gold 198 liver scans with the companion roentgenograms.

To eliminate the problems of magnification error on the companion roentgenograms the following technique (utilizing a 4 quadrant exposure) was developed for taking the roentgenograms.

## DESCRIPTION OF TECHNIQUE

The patient is positioned and scanning is carried out utilizing conventional scanning techniques. At the completion of scanning, a determination of the outline of the organ to be studied is made from the tap scan. The center of the area of activity is then photo-marked (Fig. 2). Two lines are drawn through the center of activity which divide the area of interest into 4 quadrants. The central portion of each quadrant is then explored with the scanner and the lateral border of the heart or liver under study is determined. This border is obvious since the activity falls rapidly as the scanner passes from an area of high activity into an area of background. A marker dot is made on the film at this point and a corresponding ink dot is made on the patient. This process is repeated in the other 3 quadrants.

The patient's exact position at the time of scanning is reproduced under the roentgen-ray machine. Lead markers are placed on the 4 central quadrant dots.

To minimize magnification errors as much as possible, a maximum tube film distance should be obtained. Ideally, this could be 6 feet or greater.

We have found that it is best to place the patient on the floor lying on a standard 14×17 inch grid cassette with the roentgen-ray tube positioned as far above the patient as possible. The film quality is not as good as when the table Bucky grid is used, but we feel that the lower magnification factor offsets this minor disadvantage.

A special three legged, lead topped table, with one quadrant removed has been designed (Fig. 3). The table is placed over the patient. The roentgen-ray central beam is centered (by means of the collimator cross-

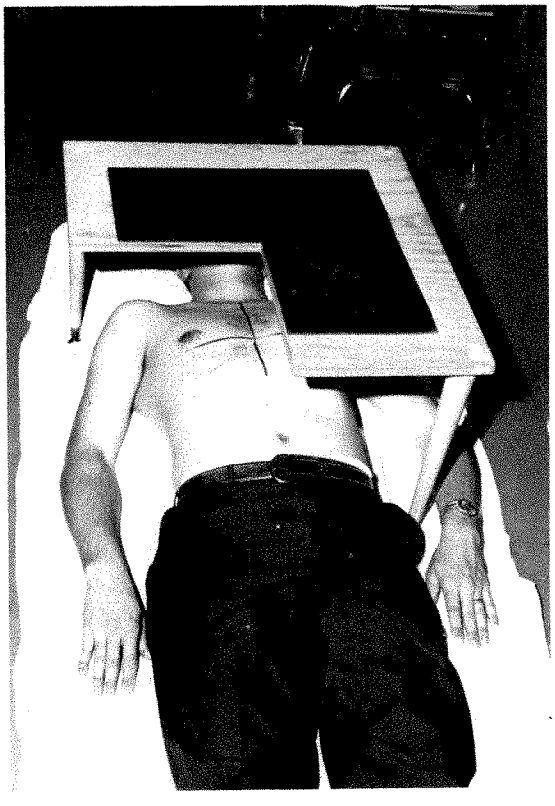


FIG. 3. Special lead top table to shield the 3 other quadrants from roentgen-ray exposure while each single quadrant is being roentgenographed.

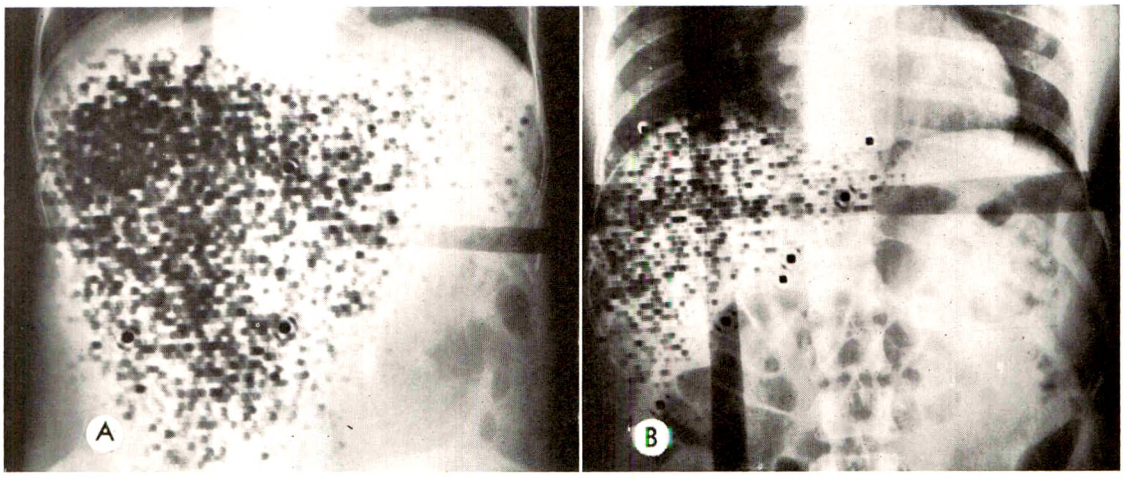



FIG. 4. (*A*) Liver scan obtained on a patient with carcinoma, metastatic to the liver with marked ascites. Notice that there is fluid between the dome of the liver and the dome of the diaphragm as indicated by radioactive free space. (*B*) Liver scan obtained on a patient with a small normal liver. Note that the liver borders correspond with the dome of the diaphragm and the bowel gas patterns. The extra black dots along the lower margin of the liver indicate the bony costal margin.

hairs) over one of the quadrant central markers. The table is positioned so that the edge of the collimator light beam falls along the edges of the inner quadrant lines.

An exposure of this quadrant is made. The table is then carefully shifted so that the opening is over one of the remaining quadrants, the tube is centered over the quadrant central marker, and a second exposure is made. This is repeated on the last 2 quadrants. Neither the patient nor the film cassette may be moved during this procedure. The patient is instructed to hold his breath in shallow inspiration while the exposures are being made. This insures that the diaphragm, heart and other internal organs will be in approximately the same position on the roentgenogram as when the scan was made. Usually a slight blurring, due to the overlap along the bisecting lines, is noted; however, this blurring is minimized by carefully positioning the table and the collimator light.

Due to the fact that the majority of the roentgen rays passing through each quadrant are nearly perpendicular to the frontal plane of the body, the 4 quadrant technique yields roentgenograms with virtually no magnification. By having the exact center of the roentgen-ray beam on the border of the organ to be studied the borders will be life-size.

The roentgenographic film is compared to the photoscans. The photo markers on the isotope scan and the lead markers on the roentgenogram should coincide and, therefore, the silhouette cast by the heart, lungs or liver on the roentgenogram coincide with minimal distortion or overlap with the isotope pattern on the scan (Fig. 4, *A* and *B*).

SUMMARY

A new "four quadrant" technique for taking roentgenograms to compare with radioisotope scans is presented. The technique allows more exact superimposition of the roentgenographic silhouette and the radioisotope organ scan outline. This greatly facilitates the radioisotope scan interpretations in heart, lung and liver scans.

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# NONVISUALIZATION OF GALLBLADDER CAUSED BY HIATAL HERNIA*

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A PATIENT with a normal gallbladder and liver rarely has unsuccessful roentgenographic visualization of the gallbladder if an adequate amount of opaque medium is ingested and the roentgenographic examination is properly conducted. In the following case, the gallbladder was not visualized because the cholecystographic medium was retained in a large hiatal hernia. A gallbladder of normal size and appearance was demonstrated on intravenous cholangiography the next day.

## REPORT OF CASE

A 62 year old woman was admitted to the Ochsner Clinic on February 11, 1964, complaining of low back pain, which had become increasingly worse for several years. On examination, obesity, slight fever, pulmonary congestion, anemia, arteriosclerosis, and spinal osteoporosis were noted. Electrocardiography indicated old myocardial infarction.

Roentgenograms of the chest on February 12 showed a large area of opacity behind the heart, consistent with a diagnosis of hiatal hernia (Fig. 1, *A* and *B*). Gastrointestinal roentgenograms proved this to be an incarcerated hernia 9.5 cm. long containing large mucosal folds (Fig. 2). This hernia was presumed to be the cause of bleeding, although no ulceration could be seen gastroscopically and extensive colonic diverticulosis offered an alternate possible site of bleeding.

Cholecystography, after the patient had taken 6 tablets of telepaque on February 25, 1964, resulted in nonvisualization of the gallbladder (Fig. 3). In a roentgenogram of the thoraco-abdominal area almost all the opaque medium was trapped in the large hiatal hernia (Fig. 4). On the next day, normal biliary ducts and a normal-sized gallbladder in which no stones were visible, were demonstrated in cholangiograms made after intravenous injection of 20 cc. of cholografin (Fig. 5).

On February 27, 1964, unrelated to the

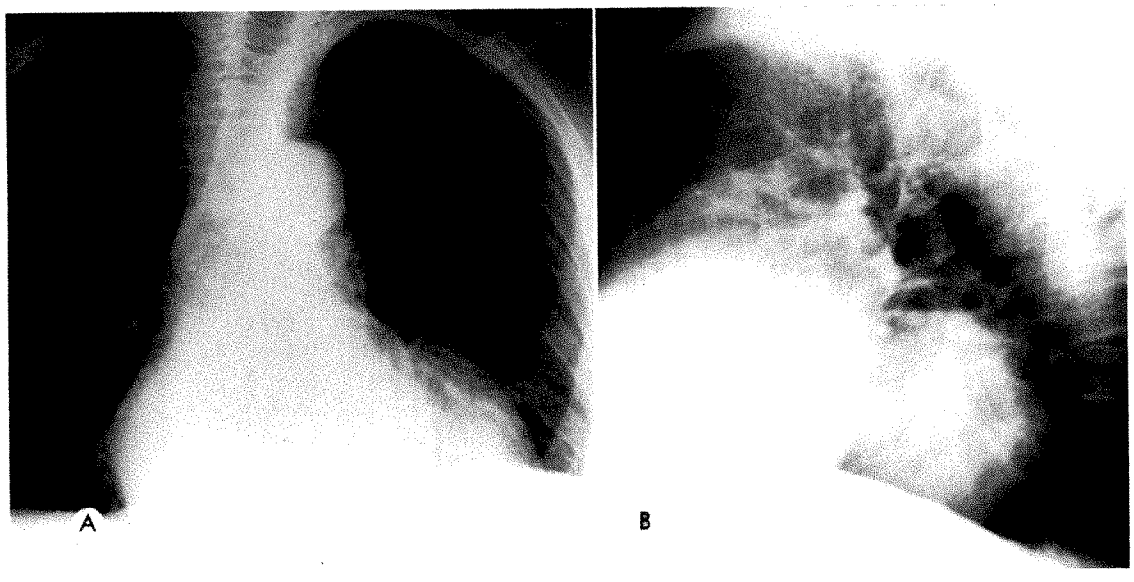



FIG. 1. (*A*) Shadow of double density related to heart visualized in anteroposterior roentgenogram of chest. (*B*) Lateral view indicates large hiatal hernia almost completely filled with fluid.

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FIG. 2. A large, incarcerated hiatal hernia is demonstrated by gastrointestinal roentgenography.

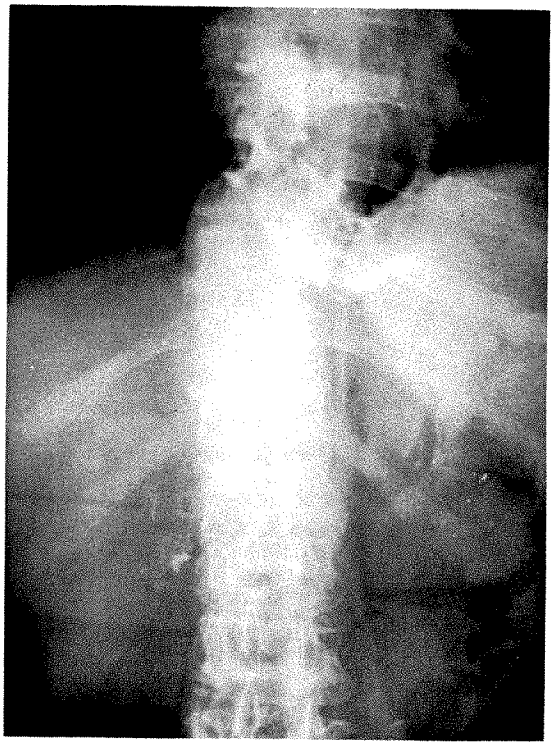


FIG. 4. Roentgenogram of thoraco-abdominal area shows retained cholecystographic medium in the hiatal hernia.

roentgenographic examinations, acute thrombosis of the right brachial artery occurred. An embolus presumed to have arisen from a mural thrombus of the heart was successfully removed. Medical treatment relieved the pain and anemia. The patient was discharged on April 9, 1964. She was well when seen on November 13, 1966, except for back pain which was believed due to osteoporosis.

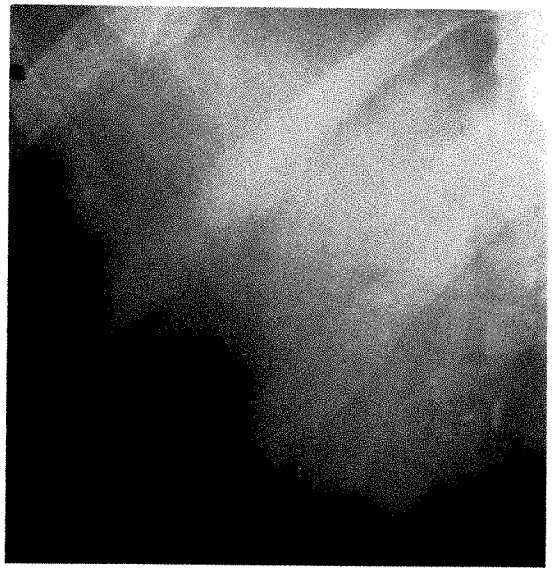


FIG. 3. No visualization of gallbladder on cholecystography after ingestion of telepaque.

## DISCUSSION

Cholecystography is so widely used, and so reliable, that physicians must be aware of the few prerequisites for successful visualization. In Shehadi's[3] review, listing 16 causes for nonvisualization, retention of the contrast medium in a hiatal hernia was not included. Various diseases of the liver, biliary ducts, and gallbladder account for most of these failures. Prehepatic diversion of the opaque medium is a relatively rare cause, if one excludes failure to ingest the medium, or emesis of it after ingestion. Lake[1] recorded an instance of contrast material trapped in an upper esophageal diverticulum, and one of us[2] reported a case in which the opaque me-

dium was retained in an epiphrenic esophageal diverticulum. Although reports of the opaque medium retained in the stomach because of obstructive disease in the pyloric area have been published,[4,5] we are aware of no previous cases of hiatal hernia causing unsuccessful visualization of the gallbladder.

## CONCLUSION

This case is reported to call attention to this possible cause of nonvisualization of the gallbladder. Hiatal hernias are common and will become more common as the average age of our citizens increases. If the patient does have an incarcerated hernia, the roentgenographic examination should include at least one roentgenogram that will exclude the possibility of retention of opaque medium in the hernia.

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FIG. 5. Normal gallbladder visualized in cholangiogram after intravenous injection of contrast medium.

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# PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY*

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JAUNDICE or icterus is the condition in which there is a hyperbilirubinemia with deposition of bile pigments in the tissue of the body. It is manifested clinically by yellow, orange, or even green discolorations of the skin, sclera, and mucous membranes. Roentgenographically, the jaundiced patient can become a diagnostic problem in that oral and intravenous methods of visualization of the gallbladder and common duct are no longer applicable. Since the contrast medium will not enter the biliary collecting system the patient is usually clinically evaluated by his history, course and chemistry profile. The important differential diagnosis rests between intrahepatic disease and extrahepatic block of the biliary tree. Although this is often clearly drawn, it is not uncommon for the differential diagnosis to become more difficult as the patient's course progresses. Percutaneous transhepatic cholangiography has been extremely helpful in visualizing patency of the biliary tract or the site of mechanical obstruction.[1–10]

It has been a valuable diagnostic aid since many of these patients are quite ill and thus, when normal biliary tree anatomy has been demonstrated, extremely poor candidates for surgery have been spared unnecessary exploratory laparotomy. If the patient has a high biliary pressure and mechanical obstruction with gross dilatation of the common duct and hepatic radicles, a surgical procedure is performed shortly after the diagnostic procedure.

Calculi and tumor, either primary or metastatic, are common causes of block. In the newborn group atresia can be demonstrated. Primary tumors encountered are carcinoma of the pancreas, ampulla of Vater, bile ducts and duodenum.

Stricture formation secondary to inflammatory disease or surgery has a significant incidence.

When patency of the common duct and main hepatic ducts is shown, the common causes of the intrahepatic block are hepatitis, cirrhosis and cholangitis.

All percutaneous cholangiography is performed in the Radiology Department utilizing the special procedure room. Premedication may be ordered but it is usually unnecessary. The patient is placed supine and an 8 inch No. 18 or 20 gauge needle is placed below the costal margin in the mid clavicular line and directed posteriorly, superiorly and medially. During this insertion, and during all future maneuvering of the needle, the patient is requested not to breathe or move. A polyethylene extension tube is attached to the needle so that aspiration and injection may be made without touching the needle. This prevents laceration of the liver capsule. Aspiration of bile is continuously attempted as the needle is advanced and withdrawn in the liver bed. All manipulations are performed under direct television control. When bile is obtained the duct is emptied as much as possible and then contrast material is injected to fill the biliary tree. The more dilated the biliary tree, the easier it is to enter the bile duct on initial probing. If after two passes into the liver bed no bile is aspirated, small amounts of contrast material are injected in an attempt to identify a biliary duct. The contrast material may pool in the parenchyma, or enter a vein or artery and may be quickly removed. When a bile duct is entered the familiar biliary tree picture is obtained. After complete filling of the bile ducts and common duct multiple spot roentgeno-

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grams are taken with the needle in place. The needle is then removed and overhead exposures are made.

The two major complications of percutaneous cholangiography are interperitoneal bleeding and bile leakage with resultant peritonitis. In most cases we have scheduled the procedure early in the morning of the planned laparotomy day. If patency of the biliary tree is established and surgery is cancelled the patient must be observed carefully. We have had no difficulty in this situation. In general, when the main biliary branches and common bile duct are open, the pressure is low and bile leakage is not a significant problem.

The following case histories were selected to demonstrate the broad applicability of the procedure.

## REPORT OF CASES

Case 1. This was the second hospital admission of a 60 year old woman who entered with the chief complaint of jaundice of 3 weeks' duration.

The patient had had a left radical mastecomy 6 months prior to this admission. The pathologic report was adenocarcinoma with metastatic disease to axillary lymph nodes. At that time, the liver was not palpable. The alkaline phosphatase was 5.2 King Armstrong units and the bilirubin was 0.7 mg. per cent. The patient did well postoperatively and underwent radiation therapy. Following this, she was discharged and was well for 6 months until 3 weeks prior to this admission, when she noted weakness, anorexia, and a 19 pound weight loss accompanied by nausea.

On the second admission, laboratory work-up revealed a bilirubin of 16 mg. per cent, alkaline phosphatase of 27 King Armstrong units and a prothrombin time of 43 per cent of normal.

Percutaneous transhepatic cholangiography showed a normal common duct with free flow of material into the small bowel (Fig. 1). The clinical impression at this time was severe hepatitis or overwhelming metastatic carcinoma to the liver. She expired shortly after admission.

Autopsy revealed a markedly icteric white female. There was a large liver, firm in consistency, almost entirely replaced by small nodules of tan firm tissue. The liver microscopically was almost completely replaced by metastatic adenocarcinoma. Blockage of small biliary radicles within the liver parenchyma was present.

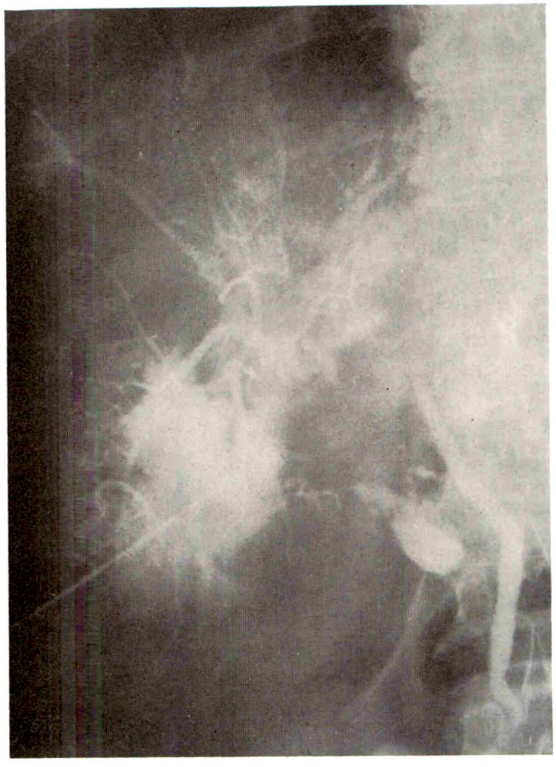


Fig. 1. Case 1. Percutaneous transhepatic cholangiography showed a normal common duct with free flow of contrast material into the small bowel.

*Comment.* Of interest is the massive metastatic disease to the liver without involvement of other organ systems. The most commonly involved metastatic site of breast carcinoma is the lung in approximately 75 per cent of patients. The liver is involved in approximately 61 per cent of patients. Many workers feel that lung metastases must almost always occur before blood born metastases can be delivered to other parts of the body. Percutaneous transhepatic cholangiography localized the mechanical block to an intrahepatic site thus removing the need for a laparotomy. Note the marked stretching of the bile ducts presumably due to metastatic tumor (Fig. 1).

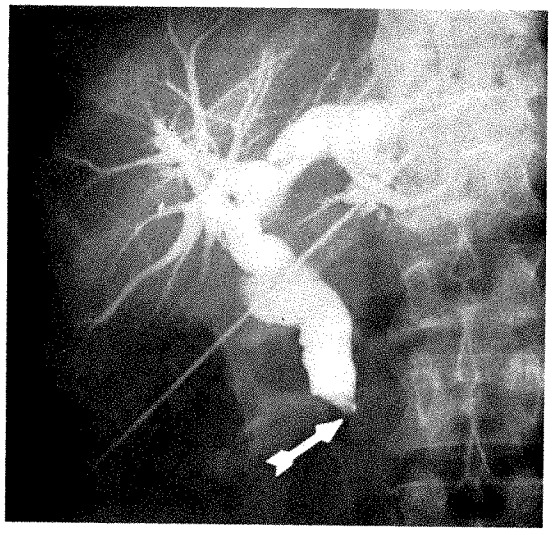


FIG. 2. Case II. Percutaneous transhepatic cholangiogram showed a dilated common duct and hepatic radicles with no evidence of a block. Marked spasm (arrow) was present at the ampulla of Vater; however, good emptying was noted during the percutaneous transhepatic cholangiography.

CASE II. This was the fifth hospital admission of a 74 year old man who entered because of abdominal pain.

The diagnosis of chronic cholecystitis and cholelithiasis was established 4 years prior to this admission. At that time, cholecystectomy and common duct exploration were performed. Stones were removed. The patient did well until 1 year ago when he experienced episodes of epigastric cramping pain subsiding spontaneously.

During this admission cholangiography was not entirely satisfactory. A percutaneous transhepatic cholangiogram showed a dilated common duct and hepatic radicles with no evidence of a block. Marked spasm was present at the ampulla of Vater (Fig. 2); however, good emptying was noted during the percutaneous transhepatic cholangiography.

The patient was re-explored and no obstruction was found. A T-tube was left in place (Fig. 3). The patient's postoperative course was complicated by chest pathology including pneumonia and pleural effusion. The patient finally succumbed following a complicated course including choledochojejunal fistula and transverse colostomy. Pulmonary infection and overwhelming septicemia appeared to be the terminal events.

*Comment.* Because of the recent onset of acute abdominal pain and the previous history of cholelithiasis, it was felt that a percutaneous transhepatic cholangiogram would be helpful in better defining the pathologic anatomy. Stones were definitely ruled out with the procedure. Some degree of cholangitis was probably present. Spasm of the ampulla of Vater was well demonstrated (Fig. 2). Chronic dilatation of the duct was probably on the basis of recurrent cholelithiasis.

CASE III. This 60 year old woman was in good health until 3 months prior to admission when she developed malaise, generalized weakness and anorexia. Two weeks prior to admission the patient became so weak that she fainted and sustained scalp lacerations. Two days later she noted the development of jaundice, nausea and vomiting, and some abdominal swelling. In the last few months she had noted the loss of 10 to 15 pounds of weight. She had also

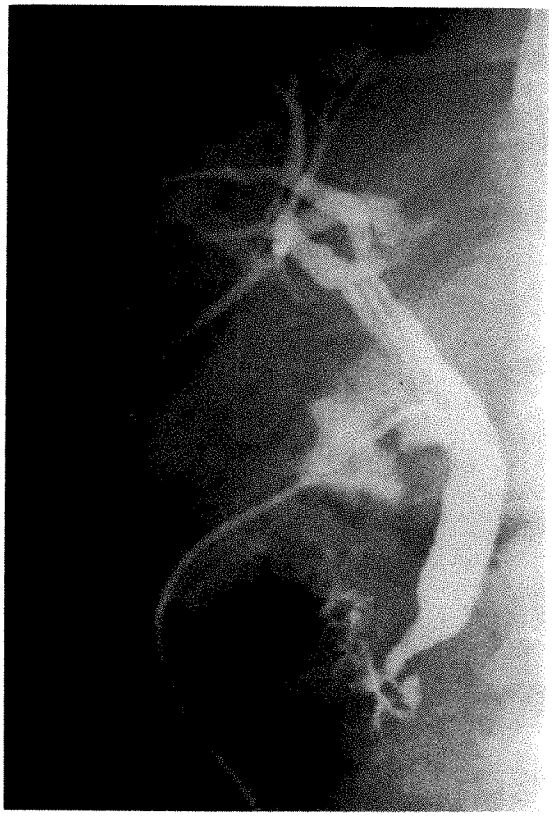


FIG. 3. Case II. The patient was re-explored and no obstruction was found. A T-tube was left in place.

noticed in the last few weeks a light color of her stool which she described as clay, and a darkening of urine.

On physical examination she was markedly jaundiced, but in no apparent distress. Her liver was huge, irregular and nodular. The impression following work-up was metastatic carcinoma to the liver with possible mechanical obstruction of the common duct. Bilirubin was 21 mg. per cent.

A percutaneous transhepatic cholangiogram showed no evidence of obstruction (Fig. 4).

Liver biopsy was performed. The pathologic examination revealed Laennec's cirrhosis with intrahepatic cholestasis.

*Comment.* Percutaneous transhepatic cholangiography clearly removed the need to explore the common duct to verify obstruction or patency. Good filling of the gallbladder was obtained as well.

Case IV. This 59 year old man entered the hospital with the complaint of jaundice of 10 days' duration accompanied by nausea and vomiting.



Fig. 4. Case III. Percutaneous transhepatic cholangiography clearly removed the need to explore the common duct to verify obstruction or patency. Good filling of the gallbladder was obtained as well.



Fig. 5 Case IV. Percutaneous transhepatic cholangiogram revealed a complete block of the common duct. The block appeared to be in the distal portion of the common duct suggesting carcinoma of the pancreas or ampulla of Vater.

The patient had a 3 month history of pain in the back, and left upper quadrant. The pain was intensified by alcohol. During this 3 month period, nausea and vomiting became more frequent until intense nausea developed 10 days prior to admission.

Physical examination revealed an enlarged tender liver. Bilirubin and alkaline phosphatase values were markedly elevated.

Percutaneous transhepatic cholangiogram revealed a complete block of the common duct (Fig. 5). The block appeared to be in the distal portion of the common duct suggesting carcinoma of the pancreas or ampulla of Vater.

At operation, a 6 cm. by 6 cm. rock-hard mass of the head of the pancreas was found involving the common bile duct. The gallbladder was markedly dilated. A gastric antrectomy with end to side gastrojejunostomy, hemipancreatectomy with end to end pancreaticojejunostomy, duodenectomy, cholecystectomy, resection of the distal common duct and end to side choledochojejunostomy were performed.

The pathology was adenocarcinoma of the head of the pancreas with invasion of the wall of the common duct and secondary obstruction.

*Comment.* Demonstration of the site of block can be very helpful in planning the

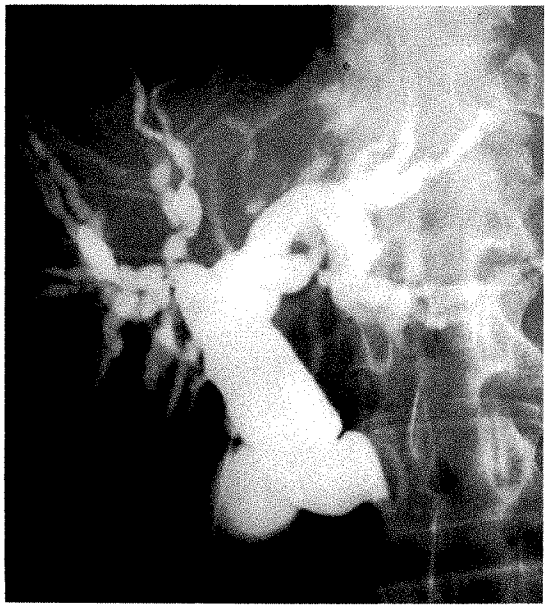


FIG. 6. Case v. Percutaneous transhepatic cholangiogram revealed complete obstruction of the common bile duct with marked dilatation of the biliary system. No contrast material advanced into the duodenum. The point of obstruction strongly suggested carcinoma of the pancreas or ampulla of Vater.

surgical approach, in evaluating the etiology of the block, and in anticipating the degree of surgery needed.

CASE V. This 73 year old white man was well until 2 days prior to admission when, while making breakfast, he lost his balance and fell backwards, injuring his back. Back pain persisted and he entered the hospital. During this period his urine had become darkened and his eyes were somewhat yellow.

Physical examination revealed a jaundiced patient with a three-finger breadth enlarged liver. The clinical picture suggested obstructive jaundice. Bilirubin was in the range of 28 mg. per cent and alkaline phosphatase was 22 King Armstrong units.

Preoperatively, a transhepatic cholangiography was performed. Complete obstruction of the common bile duct with marked dilatation of the biliary system was demonstrated. No contrast material advanced into the duodenum. The point of obstruction strongly suggested carcinoma of the pancreas or ampulla of Vater (Fig. 6).

At laparotomy, a golf ball sized mass in the head of the pancreas was found, obstructing the dilated common bile duct. A choledochojejunostomy was performed.

The pathologic report was carcinoma of the pancreas.

*Comment.* Carcinoma of the biliary tree and pancreas can cause obstruction long before a mass is felt clinically. The roentgenographic appearance of carcinoma involving the common duct is variable. The biliary tree is usually dilated. At the site of the tumor there may be a pressure defect seen indenting the dilated common duct. The distal filled tip of the common duct is most often pointed.

CASE VI. This 60 year old man was admitted for the first time with a 2 week history of jaundice and light stools. The patient in the last 6 months had lost 40 pounds, supposedly while dieting. His liver was slightly enlarged with a sharp edge. No nodules were felt. The patient's alkaline phosphatase was 43 King Armstrong units and bilirubin was 11 mg. per cent. It was felt on evaluation that intrahepatic obstruction was the most likely diagnosis.

Liver biopsy was performed and the pathologic examination revealed acute and chronic pericholangitis and biliary stasis of the liver. Intrahepatic obstruction was considered most likely.

As a result of the pathologic impression, a

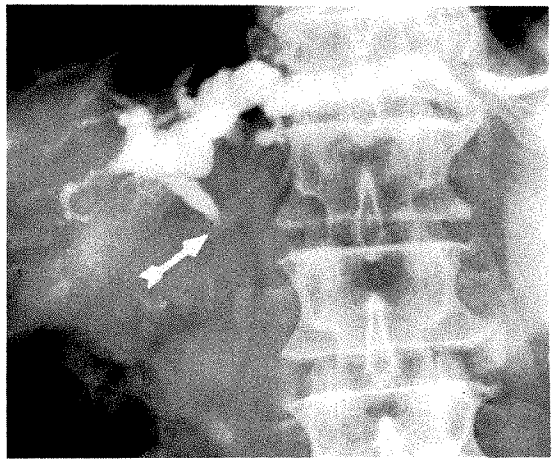


FIG. 7. Case VI. Percutaneous transhepatic cholangiogram was obtained and revealed complete obstruction of the common duct (arrow). At operation, carcinoma of the gallbladder with secondary involvement of the common duct was found.

percutaneous transhepatic cholangiography was performed and revealed complete obstruction of the common duct (Fig. 7).

At operation, carcinoma of the gallbladder with secondary involvement of the common duct was found. A T-tube was placed to re-establish drainage (Fig. 8).

*Comment.* Percutaneous transhepatic cholangiography is obviously a two-edged procedure. On the one hand, it may be used preoperatively to rule out the need for laparotomy, but on this occasion it was instrumental in documenting the need for operative decompression of the biliary tree.

Case VII. This was the second hospital admission of a 59 year old Russian man who entered with jaundice. He had a 3 week history of epigastric and right upper quadrant pain and anorexia for 1 month. He had lost 9 pounds in the preceding period of time. Laboratory data at that time revealed obstructive jaundice with bilirubin of 20.6 mg. per cent and alkaline phosphatase of 28.2 King Armstrong units.

A transhepatic percutaneous cholangiogram was obtained and demonstrated complete obstruction of the common duct (Fig. 9).

At laparotomy a tumor of the head of the pancreas was found with direct extension to the porta hepatis and celiac lymph nodes.

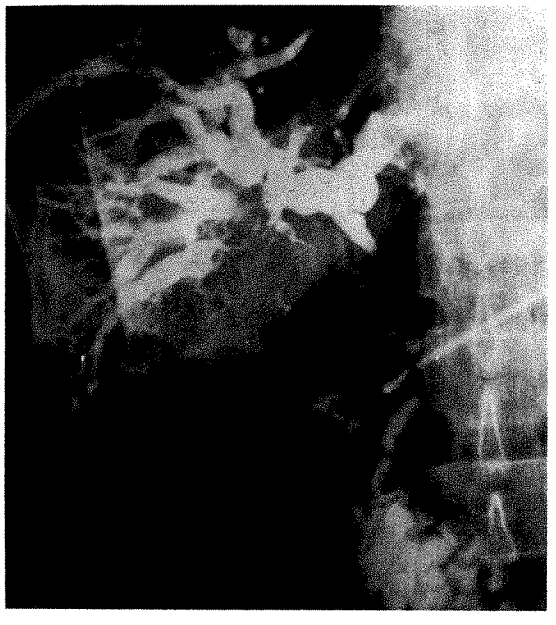


Fig. 8. Case VI. A T-tube was placed to re-establish drainage.



Fig. 9. Case VII. Transhepatic percutaneous cholangiography was performed and demonstrated complete obstruction of the common duct (arrow). At laparotomy a tumor of the head of the pancreas was found with direct extension to the porta hepatis and celiac lymph nodes.

Cholecystojejunostomy and jejunojejunostomy were performed.

The pathologic diagnosis was adenocarcinoma of the pancreas with metastases to the lymph nodes.

The patient expired on the twelfth postoperative day.

*Comment.* Once again the amount of surgery needed was predicted by the findings on cholangiography. Note contrast material placed along the liver edge during probing of the liver bed.

Case VIII. The patient's illness began with a feeling of dull discomfort beneath the right rib cage, diarrhea, and fever. In 1 month he noted that his stools had become lighter in color, his sclerae were icteric, and his urine had turned rather dark. During this period the aching under the right costophrenic angle remained fairly constant. He was seen at the hospital and admitted for evaluation.

During his hospital stay he remained icteric with a bilirubin of 5 to 7 mg. per cent and a progressive elevation of alkaline phosphatase to 40–50 King Armstrong units. It was felt clini-

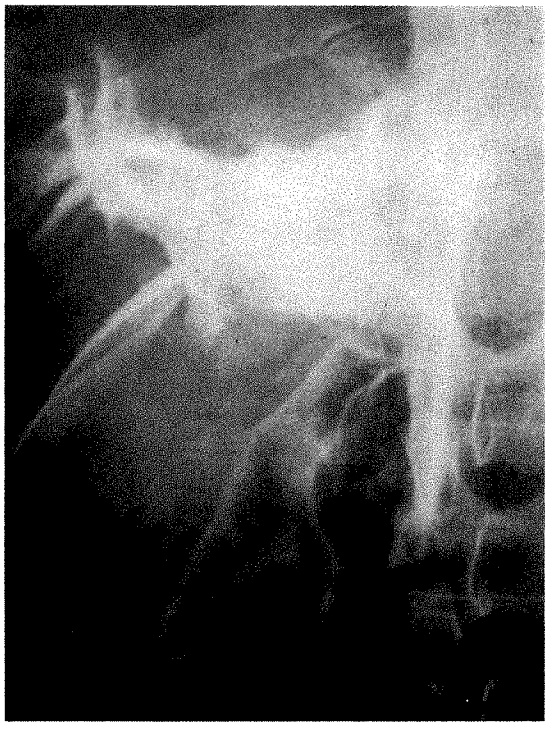


FIG. 10. Case VIII. Percutaneous transhepatic cholangiography was attempted. The examination was not satisfactory. Despite several attempts to fill the biliary tree no ductal system was visualized. Small injections into the liver parenchyma were to no avail.

cally that this was an obstructive jaundice due to an intrahepatic process.

The possibility of an extrahepatic obstruction was considered and a percutaneous transhepatic cholangiography was attempted. The examination was not satisfactory (Fig. 10). Despite several attempts to fill the biliary tree, no ductal system was visualized. Small injections into the liver parenchyma were to no avail. The patient was taken to the operating room.

The common duct was encased in a mass of firm tissue causing severe narrowing (Fig. 11). The more peripheral ducts in the liver bed showed a similar change (Fig. 12).

Pathologic examination of a biopsy specimen of the common duct showed a severe degree of mononucleate inflammatory cell infiltration of the wall with fibrosis. The liver showed parenchymal cholestasis with bile plugs in the canaliculi. Chronic inflammatory change throughout the parenchyma was noted.

*Comment.* Chronic pericholangitis and fibrosis can result in narrowing of all the bile ducts causing extreme technical difficulty in percutaneous transhepatic cholangiography.

CASE IX. This 62 year old Puerto Rican man was admitted with the chief complaint of epigastric pain. The patient had well documented diabetes with severe peripheral vascular disease. Multiple surgical procedures had been performed in the past, including sympathectomy, aortic grafts, prostatectomy, partial gastrectomy for duodenal ulcer, and cholecystectomy.

Twenty-four hours prior to admission he experienced the sudden onset of epigastric pain which he felt was similar to his previous ulcer pain. He had some nausea and vomiting of clear material. His urine was dark but without blood. His stool was of a normal brown color.

Physical examination was unremarkable except for some generalized abdominal tenderness. Alkaline phosphatase was elevated to 27 King Armstrong units. Total bilirubin was 6.7 mg. per cent. The differential diagnosis rested

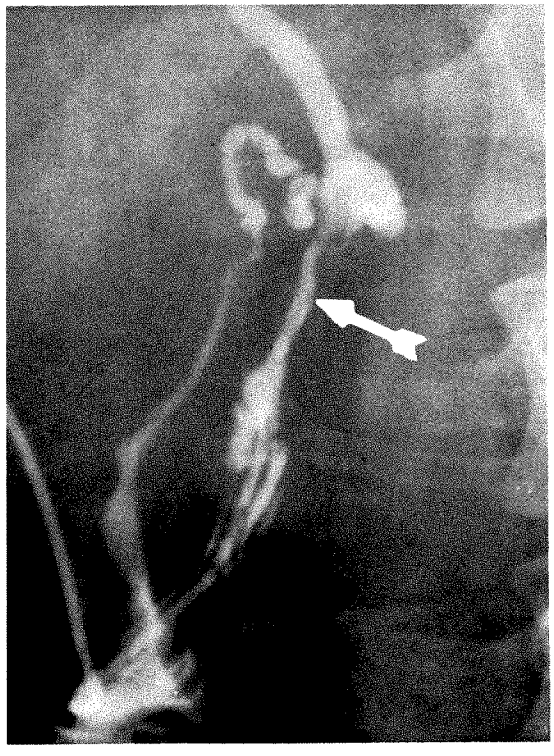


FIG. 11. Case VIII. The common duct was encased in a mass of firm tissue causing severe narrowing (arrow).

between extrahepatic block and ascending cholangitis with intrahepatic block.

At cholecystectomy 6 months before, the liver had been enlarged and a liver abscess had been drained. In light of this hepatic abscess history the possible diagnosis of recurring cholangitis certainly seemed to be a good one. Following subtotal gastrectomy marked distortion of the relationship of duodenum to common duct can occur.

Transhepatic cholangiography showed the duodenum and common duct pulled upward and medially (Fig. 13). The proximal loop of the gastroenterostomy literally followed the normal common duct position. The common duct was not obstructed.

At operation the common duct appeared to be patent but higher and more medial in position than usual. Liver biopsy was performed.

The pathologic examination revealed acute ascending cholangitis and pericholangitis with abscess formation in the parenchyma of the liver.

*Comment.* The common duct may show a marked variation in position following surgery in the area. Gastrointestinal series is recommended in all patients some time prior to percutaneous transhepatic cholangiography.

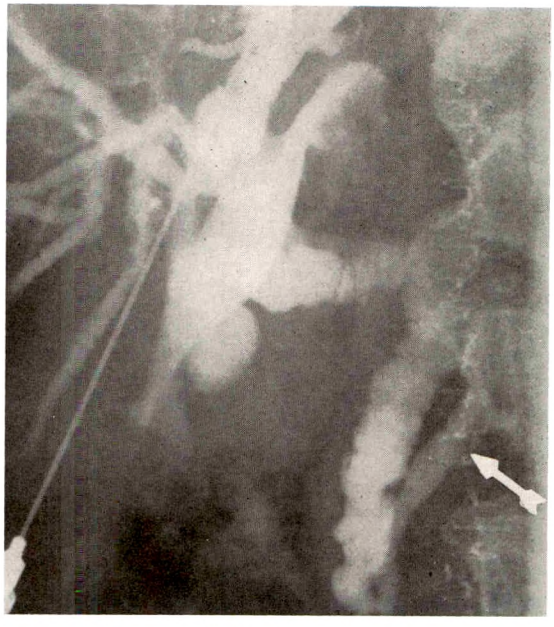


Fig. 13. Case IX. Following subtotal gastrectomy marked distortion of the relationship of duodenum to common duct can occur. Percutaneous transhepatic cholangiography showed the duodenum and common duct (arrow) pulled upward and medially. The proximal loop of the gastroenterostomy literally followed the normal common duct position.



Fig. 12. Case VIII. The more peripheral ducts in the liver bed showed similar change.

Case X. This was the second hospital admission of a 49 year old man who entered with the chief complaint of weight loss, anorexia and abdominal pain of 1 week's duration. The patient first came to the hospital 5 months before with a 2 week history of obstructive jaundice.

Percutaneous transhepatic cholangiography was unsuccessful.

The patient was explored and was found to have a common duct stricture approximately $1\frac{1}{2}$ cm. above the ampulla of Vater. Pericholangitis was noted at the time of the operation. A cholecystectomy, choledochostomy and jejunostomy with placement of a T-tube through the stenotic portion of the common duct for external drainage were performed. Stool and urine returned to normal color. T-tube cholangiogram was normal. The patient was discharged to be followed by the surgical clinic with a T-tube in place. He has been lost to follow-up.

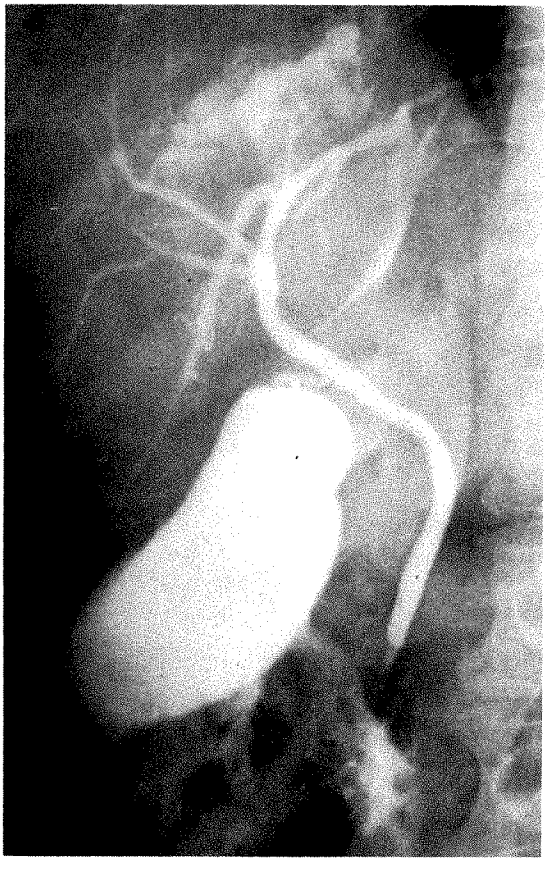


FIG. 14. Case XI. Transhepatic percutaneous cholangiography revealed a normal biliary tree with no evidence of obstruction. Note the excellent filling of the gallbladder.

*Comment.* Pericholangitis can result in severe stenosis of the duct system. Narrowed ducts create a difficult problem in performing percutaneous transhepatic cholangiography.

CASE XI. This was the second hospital admission of a 64 year old laundry employee who was admitted for evaluation of obstructive jaundice and to be treated for what appeared to be liver failure. His previous admission 3 months prior to this had ended with the diagnosis of alcoholic nutritional cirrhosis. Since that time he had had continual itching and elevated bilirubin values.

Physical examination revealed a jaundiced patient with an enlarged firm liver. Many signs of Laennec's cirrhosis were present.

To rule out the possibility of an extrahepatic obstruction component to this disease due possibly to a gallstone, transhepatic percutaneous cholangiography was performed. This revealed a normal biliary tree with no evidence of obstruction. Note the excellent filling of the gallbladder (Fig. 14).

*Comment.* Demonstration of a normal common duct removes a need in many cases for operation. If open biopsy of the liver has also been contemplated, then a great deal of surgery on the common duct can be saved.

CASE XII. This was the first hospital admission of a 50 year old woman who presented with chief complaint of diarrhea and weight loss of 4 weeks' duration. For this period, the patient had experienced soft, light colored stools and severe general pruritus. Dark colored urine was also noted.

On admission to the hospital the patient was noted to be severely jaundiced. The liver was firm, nontender and enlarged. Laboratory examinations revealed a markedly elevated alkaline phosphatase and bilirubin.

Preoperatively, a percutaneous transhepatic cholangiography was performed. Marked dila-

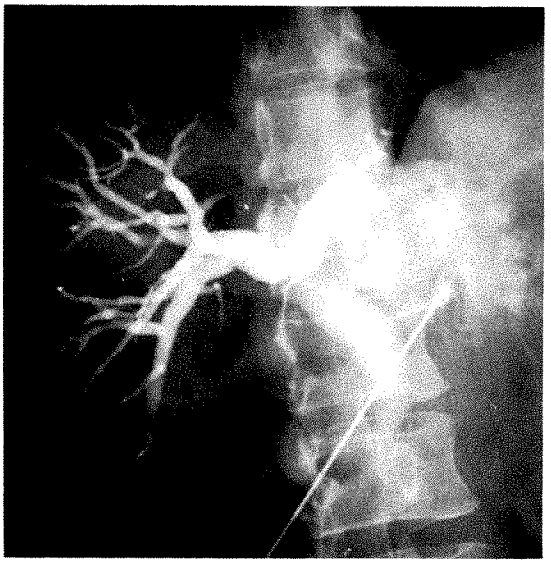


FIG. 15. Case XII. Preoperatively, a percutaneous transhepatic cholangiogram was obtained. Marked dilatation of the right and left hepatic ducts was noted. Abrupt block was present at the junction of the right and left common hepatic ducts, and a small amount of contrast material was seen to pass into a more distal, markedly dilated common duct, which was completely obstructed with no contrast material entering the intestinal tract.

tation of the right and left hepatic ducts was noted. An abrupt block was present at the junction of the right and left common hepatic ducts, and a small amount of contrast material was seen to pass into a more distal, markedly dilated common duct, which was completely obstructed with no contrast material entering the intestinal tract (Fig. 15). It was felt that the most likely diagnosis was carcinoma of the pancreas, ampulla of Vater, common duct, or possible metastatic lymph nodes to the porta hepatis; calculi were less likely.

At operation a large mass in the region of the pancreas was found. Biopsy was taken. No peritoneal or hepatic metastases were noted.

The pathologic report was based on a lymph node biopsy from the lesser sac. The diagnosis was metastatic adenocarcinoma, probably primary in the pancreas.

Case XIII. This was the second hospital admission of this 89 year old female with the chief complaint of jaundice for 2 months. The patient had been relatively well except for a previous episode of diverticulitis. Six months prior to admission, she developed mild anorexia, but was otherwise well. Four weeks prior to admission she began to feel tired, but no other symptoms arose until 2 months prior to admission when she noted jaundice. Her urine became dark and her stool became clay colored. She occasionally experienced a tight feeling in the mid epigastrium, but no definite pain.

On physical examination, the liver was enlarged. No other positive findings were obtained. The alkaline phosphatase and bilirubin values were markedly elevated.

Upon admission the diagnosis was obstructive jaundice, probably extrahepatic.

A percutaneous transhepatic cholangiogram was obtained preoperatively and revealed complete obstruction of the distal common duct (Fig. 16).

At operation, the common duct and gallbladder were markedly distended. A hard mass in the head of the pancreas was noted.

The diagnosis was adenocarcinoma involving the liver and pancreas, probably primary in the pancreas.

Case XIV. This was the second hospital admission of a 77 year old married woman who entered because of pruritus and jaundice.

One year previously, she was admitted to the hospital and underwent cholecystectomy

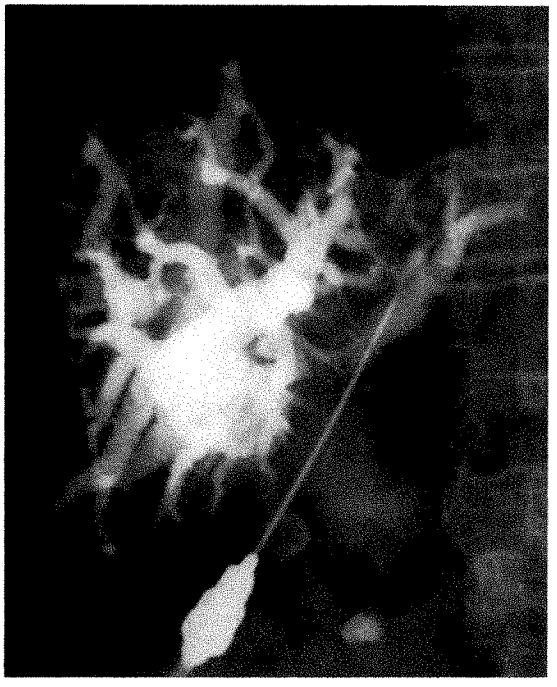


Fig. 16. Case XIII. A percutaneous transhepatic cholangiogram was obtained preoperatively and revealed complete obstruction of the distal common duct.

and common duct drainage because of an adenocarcinoma of the gallbladder with extension to the porta hepatis. At that time, the patient's bilirubin and alkaline phosphatase were elevated. At surgery, the tumor was found to involve the common hepatic duct, just proximal to the cystic duct. Decompression was accomplished.

Three months later, the patient again became jaundiced and intense pruritus developed.

On the second admission, the physical findings were limited to the abdomen which was protuberant. No fluid wave was palpable. The liver edge was 2 finger breadths below the right costal margin in the midline.

Percutaneous transhepatic cholangiogram showed complete obstruction of the common hepatic duct with massive dilatation of the bile ducts. Two immense ducts descended into the lower lateral edge of the liver (Fig. 17, 18 and 19).

*Comment.* This case clearly demonstrates how the information obtained during percutaneous transhepatic cholangiography can influence and occasionally decide the technical aspects of a procedure. The vari-

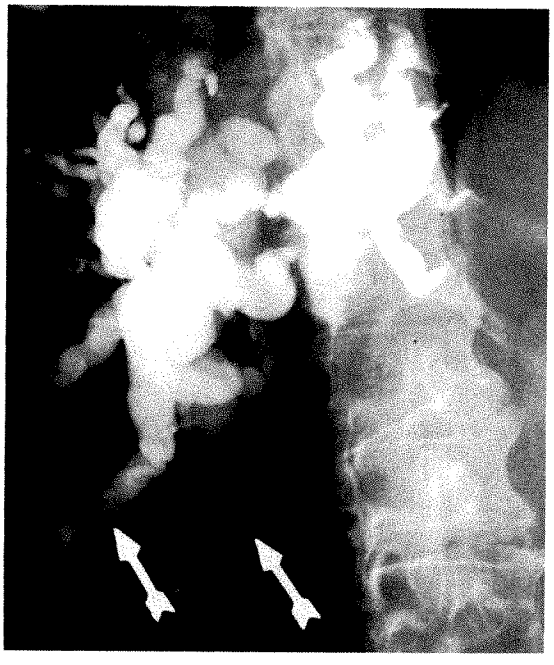


FIG. 17. Case XIV. Percutaneous transhepatic cholangiogram showed complete obstruction of the common hepatic duct with massive dilatation of the bile ducts. Two immense ducts descended into the lower lateral edge of the liver (arrows).

cose-like dilated channels in the lower liver edge anteriorly became the major anastomotic channels with the intestinal tract (Fig. 20).

CASE XV. This was the fifth hospital admission of a 64 year old woman with the chief complaint of jaundice. Previous admissions had involved two bouts of pneumonia, sinusitis and hemorrhoidectomy. Her present illness started 5 months prior to admission when she noted the onset of tiredness and anemia. In the following months, she noted a loss of appetite, but no vomiting or pain. Finally, shortly before admission, jaundice developed. At that time her urine became dark and her stools became clay colored. Weight loss in the preceding 5 months to admission had been 15 pounds.

Physical examination was unremarkable except for icterus and an enlarged, firm, nontender liver. During hospitalization, the differential diagnosis of intrahepatic liver disease *versus* extrahepatic block on the basis of calculi or tumor was a difficult one and percutaneous transhepatic cholangiography was performed.

Percutaneous transhepatic cholangiography demonstrated a large filling defect in the gallbladder suggesting calculi (Fig. 21). The common duct was not dilated and was clearly patent with contrast material flowing freely into the duodenum (Fig. 22). The biliary tree was not dilated.

Shortly after the percutaneous transhepatic cholangiography, the patient was explored. The common duct was opened and found to be widely patent. Large amounts of sludge were present in the gallbladder. The patient was discharged with the diagnosis of biliary cirrhosis and cholecystitis.

*Comments.* It can be difficult to differentiate calculi in the gallbladder from debris and bile sludge as in this case. Once again the common duct was clearly shown to be normal.

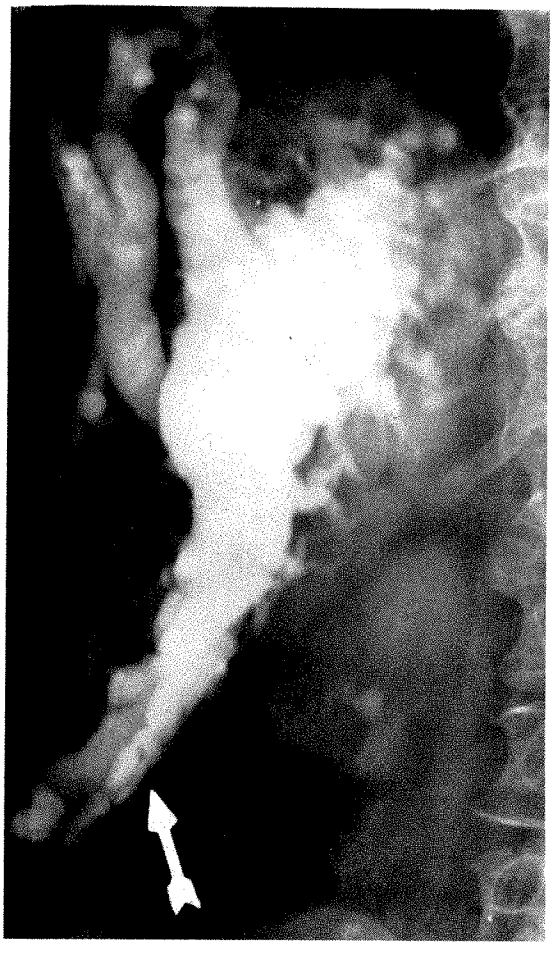


FIG. 18. Case XIV. A delayed cholangiogram showed better filling of the dilated ducts in the lower tip of the liver (arrow).



Fig. 19. Case XIV. Lateral view showed the dilated ducts to be anterior in position (arrows).



Fig. 20. Case XIV. Postoperative study demonstrates the utilization of these huge ducts in decompressing the entire biliary tree (arrow).

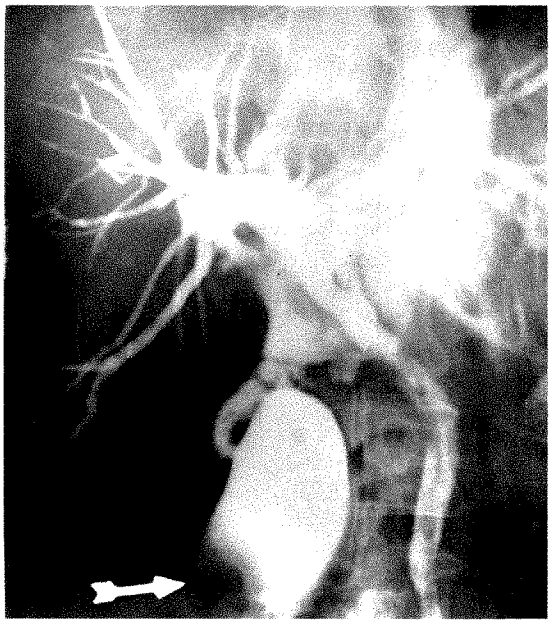


Fig. 21. Case XV. Percutaneous transhepatic cholangiography demonstrated a large filling defect in the gallbladder suggesting calculi (arrow).



Fig. 22. Case XV. A closer view of the normal common duct and the defects in the gallbladder (arrow).

SUMMARY

The radiologist has proven percutaneous transhepatic cholangiography to be a safe, valuable diagnostic method. In the very ill patient much information can be obtained with relatively little trauma or discomfort. The biliary tree and common duct can be normal with good flow of contrast material into the duodenum. In patients with marked obstruction, the point of block and the probable etiology can be demonstrated. Surgery can be planned with reliable, advanced knowledge of the pathologic anatomy to be encountered and dealt with. Anatomy that is often obscured at the operating table by overwhelming tumor or diffuse fibrosis is clearly demonstrated on the cholangiogram.

The prognosis of persistent, progressive, severe jaundice is poor regardless of the etiology. For patients in whom extrahepatic block is even slightly suspected, percutaneous transhepatic cholangiography is strongly recommended as a safe, rapid and reliable source of information regarding the pathologic anatomy present.

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# PREOPERATIVE DIAGNOSIS OF CARCINOMA OF THE GALLBLADDER BY PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY*

*By* JAMES G. McNULTY, M.B., B.Ch., B.A.O., D.M.R.D., F.F.R.
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PRIMARY carcinoma of the gallbladder is an uncommon tumor which is rarely diagnosed preoperatively. It is usually an unexpected finding at laparotomy for calculous disease of the biliary tract.

The neoplasm may develop in association with gallbladder adenoma or papilloma.[5,13] A case has been reported by Cimmino[1] in which the tumor presented as a filling defect in the fundus of the gallbladder at cholecystography. Intraductal spread of the neoplasm, which occurs in 4 per cent of cases[2] may produce a similar appearance to primary bile duct carcinoma at percutaneous transhepatic cholangiography. In the case reported the bile ducts were not infiltrated by the tumor.

## REPORT OF A CASE

A 59 year old man was admitted with a 2 year history of recurrent attacks of painless jaundice. He had been jaundiced for 4 weeks prior to admission. There was no history of weight loss or melena. Clinical examination revealed a moderate degree of icterus and smooth enlargement of the liver. The urine contained excess bilirubin and a trace of urobilinogen.

*Investigations.* Hb. 12.6 gm./100 ml.; white blood cell count 6,000 cells/cu. mm.; total serum bilirubin 5.4 mg./100 ml. (conjugated bilirubin 5.2 mg./100 ml.); total serum proteins 6.9 gm./100 ml., albumin 3.5, globulin 3.4; serum alkaline phosphatase 38 King Armstrong units.

*Liver Biopsy.* This showed portal zone expansion, ductule proliferation, and centrilobular cholestasis not involving all the lobules. There was an inflammatory cell infiltrate around some of the central veins. Large duct obstruction could not be excluded on histologic grounds.

*Percutaneous Transhepatic Cholangiography.* This was performed under local anesthesia using the technique described by Shaldon *et al.*[11] A bile duct in the left lobe of the liver was catheterized. There was considerable dilatation of the intrahepatic and common bile ducts with common duct calculi and a small calculus at the lower end of the dilated common bile duct. There was some drainage of contrast medium into the duodenum. A short segment of the cystic duct was opacified. This was markedly dilated and terminated in a concavity with a finely irregular border. The appearances were reported as being consistent with carcinoma of the gallbladder obstructing the cystic duct (Fig. 1).

*Operative Findings.* Laparotomy revealed a carcinoma of the gallbladder infiltrating the cystic duct and the adjacent surface of the liver. Calculi were present in the common bile duct and there were metastases in the omentum and peritoneum. The tumor was excised and a T tube inserted into the common bile duct with drainage to the duodenum. The jaundice regressed postoperatively and the patient was allowed home.

*Histology.* Papillary adenocarcinoma.

The patient died 4 months later from carcinomatosis.

## DISCUSSION

Carcinoma of the gallbladder is a disease of elderly women with gallstones. The latter are an important etiologic factor in its development.[7,10,14] Experimental evidence supports this view. The tumor has been induced by insertion of methylcholanthrene pellets into the gallbladder lumen in animals.[3] It is estimated that 10 per cent of patients over the age of 65 years with symptomatic gallstones will develop carcinoma of the gallbladder.[4]

Clinically the neoplasm mimics chronic inflammatory disease. Continuous pain and weight loss suggest malignancy.[12] An unusual feature of this case was the absence of

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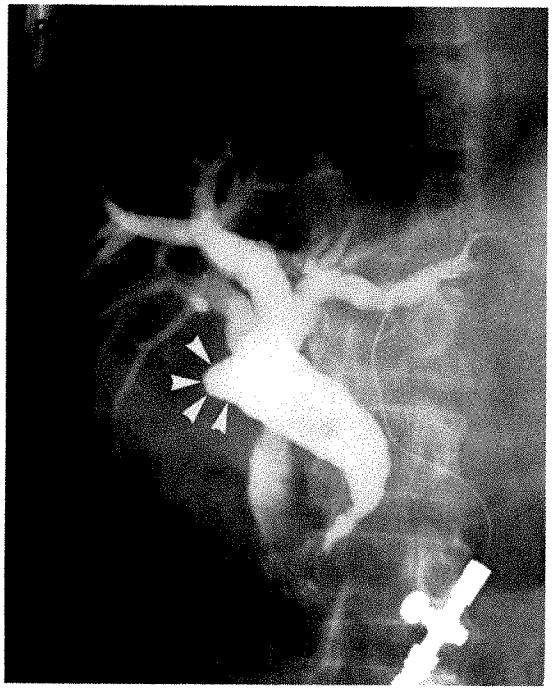


FIG. 1. Percutaneous transhepatic cholangiogram showing dilatation of the bile ducts. The common bile duct contains translucent calculi; the cystic duct obstruction produced by the gallbladder carcinoma is outlined by the arrows.

pain. Fifty per cent of cases present with jaundice as a complication.[4] The usual radiologic methods of investigation of the biliary tract fail to opacify the gallbladder when it is infiltrated by carcinoma. Barium meal may show deformity of the pylorus and proximal duodenal loop. Hypotonic duodenography, originally introduced by Liotta,[9] may show a characteristic deformity of the second part of the duodenum which may be distinguished by this technique from pancreatic and primary duodenal tumors. Three such cases are illustrated by Jacquemet, Liotta and Mallet-Guy.[8]

At percutaneous cholangiography the gallbladder is usually opacified or the cause of nonopacification is shown, especially if bile is aspirated from the ducts before contrast medium is injected. A calculus obstructing the gallbladder neck or cystic duct is partly outlined as a filling defect with a convex border by contrast medium refluxing from the common bile duct. Edema after the passage of a calculus produces complete cystic duct obstruction at its junction with the common bile duct and there is usually associated biliary pain. Carcinoma may have to be distinguished from leiomyosarcoma by histology. The latter is a very rare tumor which is occasionally reported in the gallbladder.

Since carcinoma of the gallbladder is usually found in association with biliary calculi, often with stasis and bile duct dilatation, percutaneous cholangiography will be successful in many cases. The demonstration of an irregular cystic duct obstruction as described in this patient should alert the radiologist preoperatively to the possibility of a malignant gallbladder tumor.

SUMMARY

A case of carcinoma of the gallbladder in a 59 year old man, diagnosed preoperatively by percutaneous transhepatic cholangiography, is reported.

A short segment of the cystic duct was opacified by reflux of contrast medium from the common bile duct. The cystic duct terminated in a concavity with a finely irregular border which corresponded to the intracystic duct infiltration of the neoplasm. The appearances have not been previously described and are thought to be characteristic.

The causes of nonopacification of the gallbladder at percutaneous cholangiography are discussed.

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# CHOLECYSTO-ANGIOGRAPHY*

## VISUALIZATION OF THE GALLBLADDER BY SELECTIVE CELIAC AND MESENTERIC ANGIOGRAPHY

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THE role of selective celiac and mesenteric angiography in the diagnosis of intra-abdominal disorders and especially in the evaluation of hepatic, splenic and pancreatic disease is becoming increasingly important. Boijsen and Reuter[1] in a recent article describe its value in diseases of the hollow viscera, which could not be differentiated by routine roentgenologic methods.

The purpose of this preliminary paper is to discuss the value of selective celiac and mesenteric angiography in the diagnosis of gallbladder diseases which cannot be made by routine roentgenologic means.

### TECHNIQUE

Our experience with selective celiac and mesenteric angiography is based on the investigation of 150 cases. The technique employed was essentially that proposed by Ödman,[4] using a green Kifa catheter, introduced through the femoral route or, if not possible, the axillary route. Consecutive catheterization of the celiac and mesenteric arteries was performed injecting 40–50 cc. contrast medium (Urographin-Schering, 76 per cent) into the celiac artery and about half this quantity into the mesenteric artery. The contrast medium was injected with a Gidlund syringe at a pressure of 3 kg. The exposures were made in the following sequence: 8 exposures at ½ sec. intervals; 4 at 1 sec. intervals and 10 at 3 sec. intervals. The exposure factors were: 70–80 kv., 500 ma.; 0.25 sec. Kodak Royal Blue films were used in most cases.

### THE BLOOD SUPPLY OF THE GALLBLADDER

The cystic artery is a relatively large vessel, considering the size of the organ it supplies. According to textbook descriptions, it arises from the right hepatic artery, near its origin from the common hepatic artery.[3] Shortly after its origin, the cystic artery divides into an anterior branch which is right sided and found on the free peritoneal surface of the gallbladder wall, and a posterior branch which is more left sided and found on the nonperitoneal surface of the gallbladder wall. The secondary branches of the two cystic arteries communicate and end in rich capillary plexuses.

In about 25 per cent of the cases the two main branches of the cystic artery originate separately from the right hepatic artery (Fig. 1, *A–D*). Other common variations include origin of the cystic artery or one of its branches from the common hepatic artery, from the left hepatic artery, the gastroduodenal artery or from the celiac trunk itself.

Some of the venous blood from the gallbladder is drained directly into the liver through a complicated venous capillary plexus, and some into the portal system by anastomoses with the choledochal and superior mesenteric veins.

The cystic artery, when its origin and course are normal, can easily be detected by selective celiac and mesenteric angiography. When there are variations in its origin, its detection may be difficult or sometimes even impossible.

Although the gallbladder wall has a thickness of only 2–3 mm., it can be well demonstrated during the capillary phase of angiography by virtue of the rich plexuses it contains.

It is important to appreciate that in some 25 per cent of the cases the hepatic artery itself originates partly or wholly from the superior mesenteric artery.[3] For this reason, visualization of both celiac

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and superior mesenteric arteries is necessary.

### THE NORMAL GALLBLADDER

In our earliest selective celiac and mesenteric angiographies we observed that the normal gallbladder wall was demonstrated as an egg shell thin shadow, 6–8 sec. after visualization of the cystic artery. The condition for its demonstration was that contrast medium be directed into the hepatic artery in adequate quantity and not

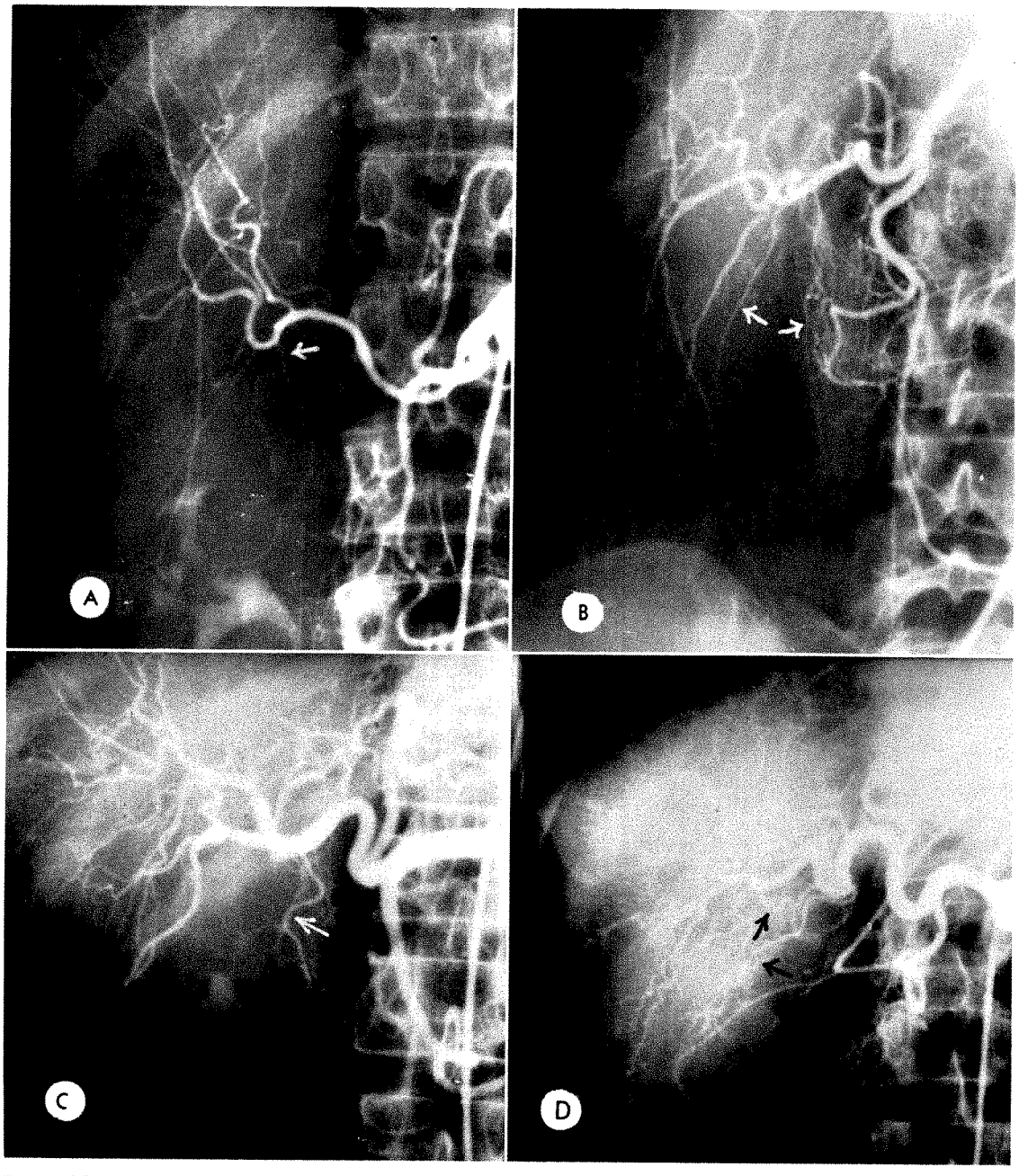



FIG. 1. Normal variations of the cystic artery. (*A*) The common cystic artery is relatively long and divides into two branches. (*B*) The two cystic arteries originate separately from the right hepatic artery. (*C*) Only a single cystic artery branch can be recognized. (*D*) A short common cystic artery divides into two tortuous branches.

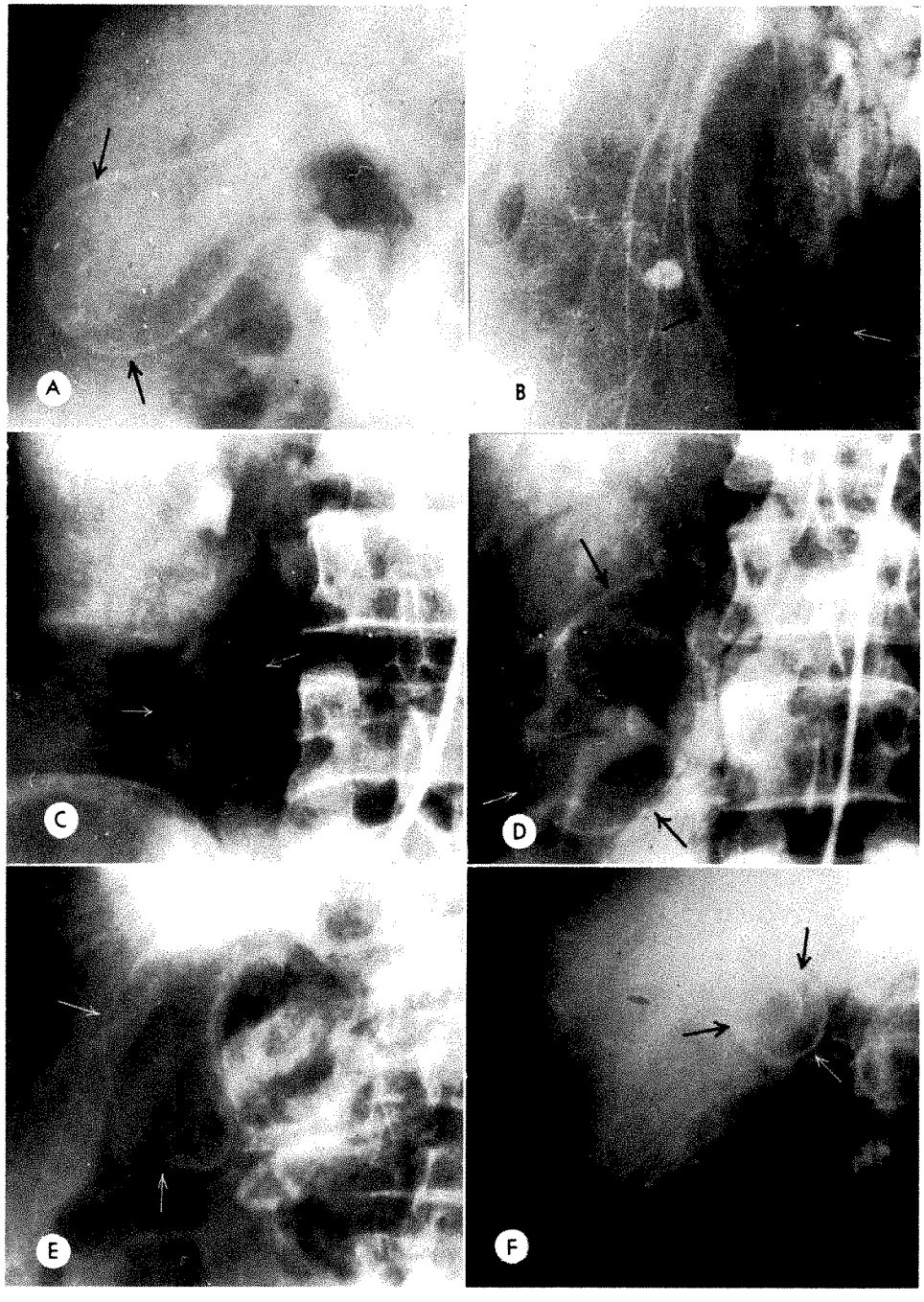



FIG. 2. (*A–F*) Capillary phase of angiography showing different shapes of the normal gallbladder wall. The white stain lateral to the gallbladder in *B* represents a small pericytoma.

shunted away by highly vascular lesions, such as neoplasms or hemangiomas in the liver parenchyma. It has been found possible to define the different shapes of the normal gallbladder, its various localizations in normal cases as well as to differentiate them from pathologic patterns (Fig. 2, *A–F*).

## THE ANGIOGRAPHIC APPEARANCE OF GALLBLADDER CARCINOMA

Carcinoma of the gallbladder was diag-

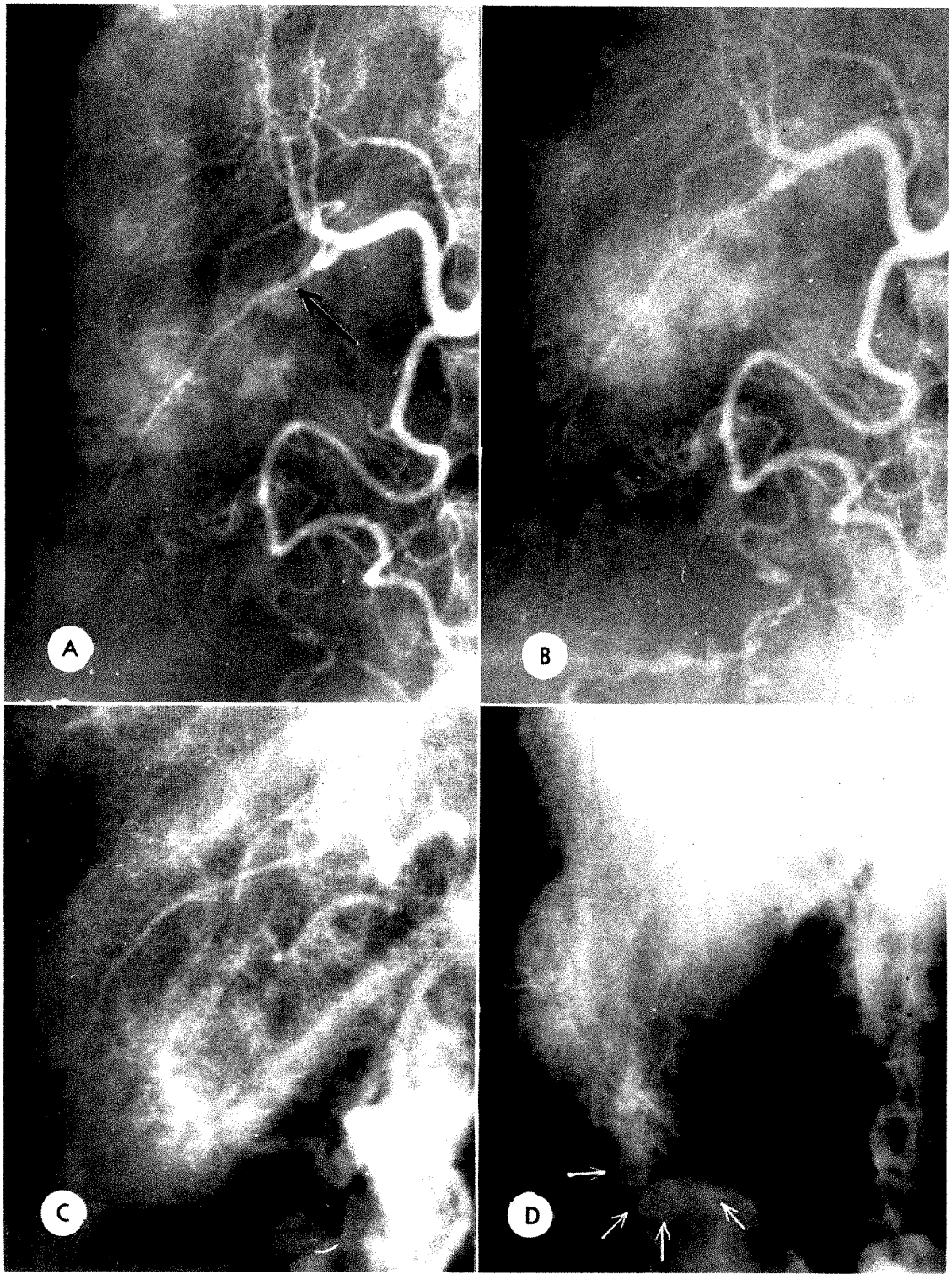



FIG. 3. The different characteristic patterns of malignant tumors of the gallbladder. (*A*) Early arterial phase. The cystic artery is enlarged and prominent (arrow). Its distal segment supplies pathologic vessels and is surrounded by pathologic staining. (*B*) Same case, 2 seconds later. The diffuse pathologic staining is more prominent and involves the entire gallbladder area. (*C*) Inoperable carcinoma of gallbladder. Large and diffuse pathologic vessels fed by the cystic artery and prominent early venous filling. (*D*) Resectable carcinoma of gallbladder. Oval shaped pathologic staining of a thickened gallbladder wall with smooth edges involving only a portion of its wall.

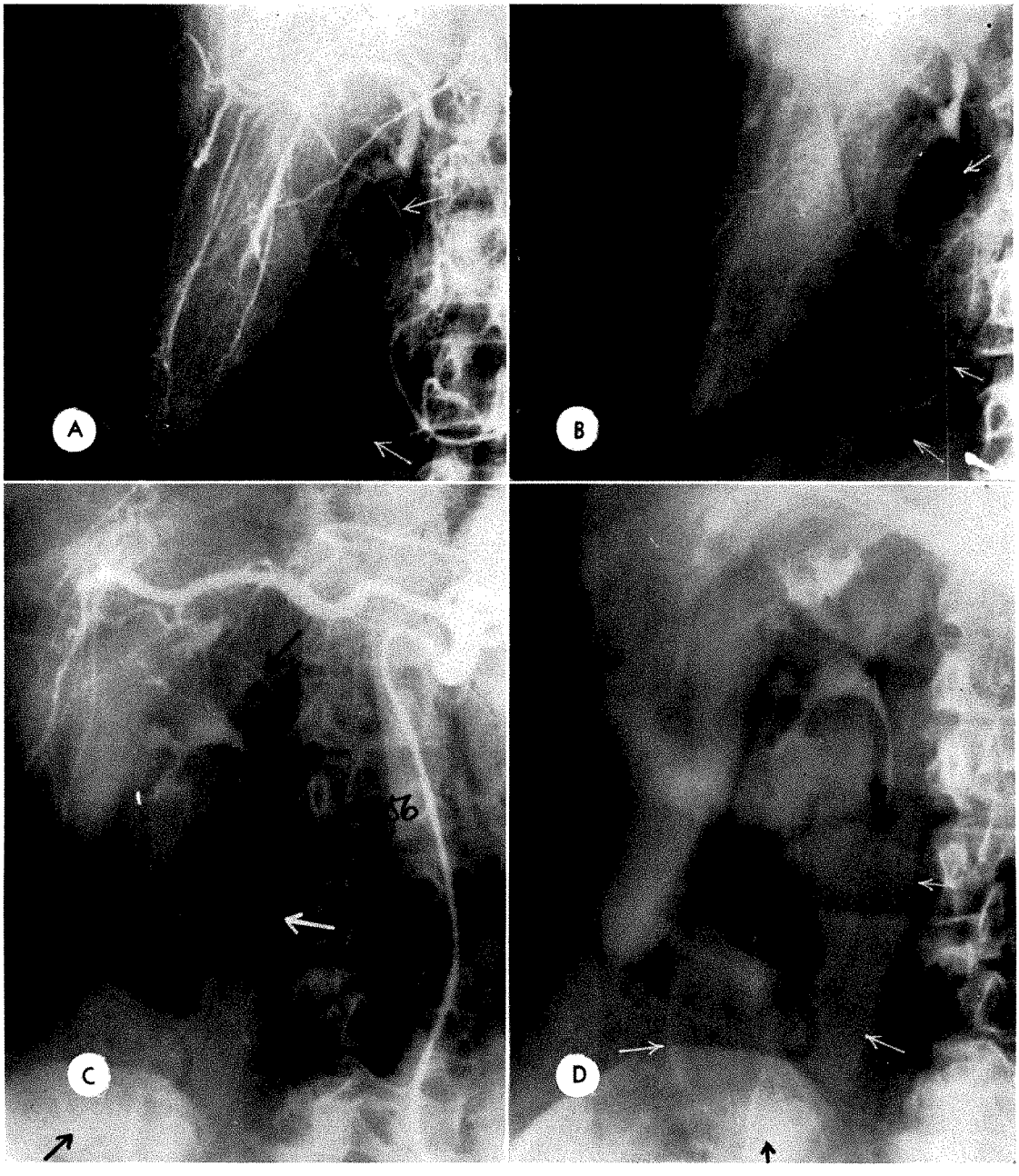



FIG. 4. Hydrops of the gallbladder (*A* and *B* pertain to one case, *C* and *D* to another case). (*A* and *C*) Arterial phase. The cystic artery is elongated and stretched having a prominent convex course. (*B* and *D*) Capillary phase. The elongated gallbladder with its paper thin wall can be clearly seen.

nosed in 5 cases—all confirmed at laparotomy or autopsy. No false-positive or, as far as we know, false-negative diagnoses were made. All 5 cases demonstrated classical angiographic signs of malignancy. In 2 cases, pathologic vessels of varying caliber originating from a prominent cystic artery, were present in the gallbladder region (Fig. 3*A*). Pathologic staining of the gallbladder wall or its environs was noted in all cases (Fig. 3, *B*, *C* and *D*). This involved the entire gallbladder area in 2 cases and only

a portion of its wall in 2 others (Fig. 3, *B* and *D*; and 5, *A* and *B*). In the fifth case widespread diffuse staining represented, as confirmed at necropsy, infiltration around the gallbladder into the adjacent liver (Fig. 3*C*). The staining appeared in the very early arterial phase, increased during the following 4 seconds and its intensity decreased only in the late portal phase. Early venous filling was demonstrated in 1 case only.

Our experience with tumors of the choledochus is limited to a single case in which pathologic vessels and localized constriction of the hepatic arteries could be demonstrated in the hilus of the liver.

### ANGIOGRAPHIC APPEARANCE OF HYDROPS OR EMPYEMA

The angiographic findings in 2 cases of hydrops and 1 case of empyema of the gallbladder were clear-cut. The cystic arteries showed a stretched concave course, surrounding the dilated gallbladder, whose paper thin wall could be recognized protruding below the margin of the liver (Fig. 4, *A–D*). In the case of empyema the demonstration of gallbladder dilatation, together with large pathologic vessels around the gallbladder and a zone of pathologic staining of the wall in its proximal portion, prompted an angiographic diagnosis of malignant neoplasm (Fig. 5, *A* and *B*). Although the surgeon's impression after careful inspection and palpation of the region was that only empyema was present, microscopic examination of the surgical specimen revealed adenocarcinoma, confirming the angiographic diagnosis.

### CHRONIC CHOLECYSTITIS

The angiographic appearance of chronic cholecystitis, with or without cholelithiasis, was studied in a group of cases in which this was usually an incidental finding. The diagnosis of chronic cholecystitis was confirmed at laparotomy, frequently performed for other disease, or at necropsy. In almost all cases a very similar angiographic appearance could be observed. The cystic artery was less readily detectable than usual and, when visualized, was smaller in caliber than normal (Fig. 6*A*). By contrast, the epiploic branches of the right gastro-epiploic artery were more

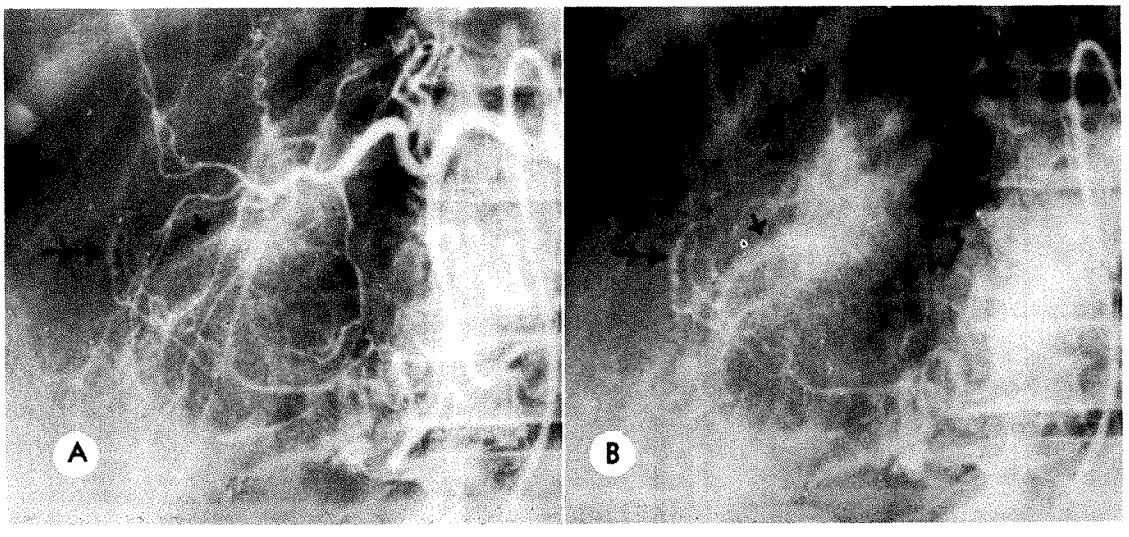



Fig. 5. Empyema associated with carcinoma of the gallbladder. (*A*) Early arterial phase. The cystic arteries are stretched and surround an enlarged gallbladder. The wall of the upper pole of the gallbladder appears thickened, as visualized by the pathologic staining (arrow). Lateral to the gallbladder, a large pathologic vessel can be seen (double arrow). (*B*) Late arterial phase. Cranial to the gallbladder a round pathologically stained shadow can be seen corresponding to involved lymph nodes (thick arrow).

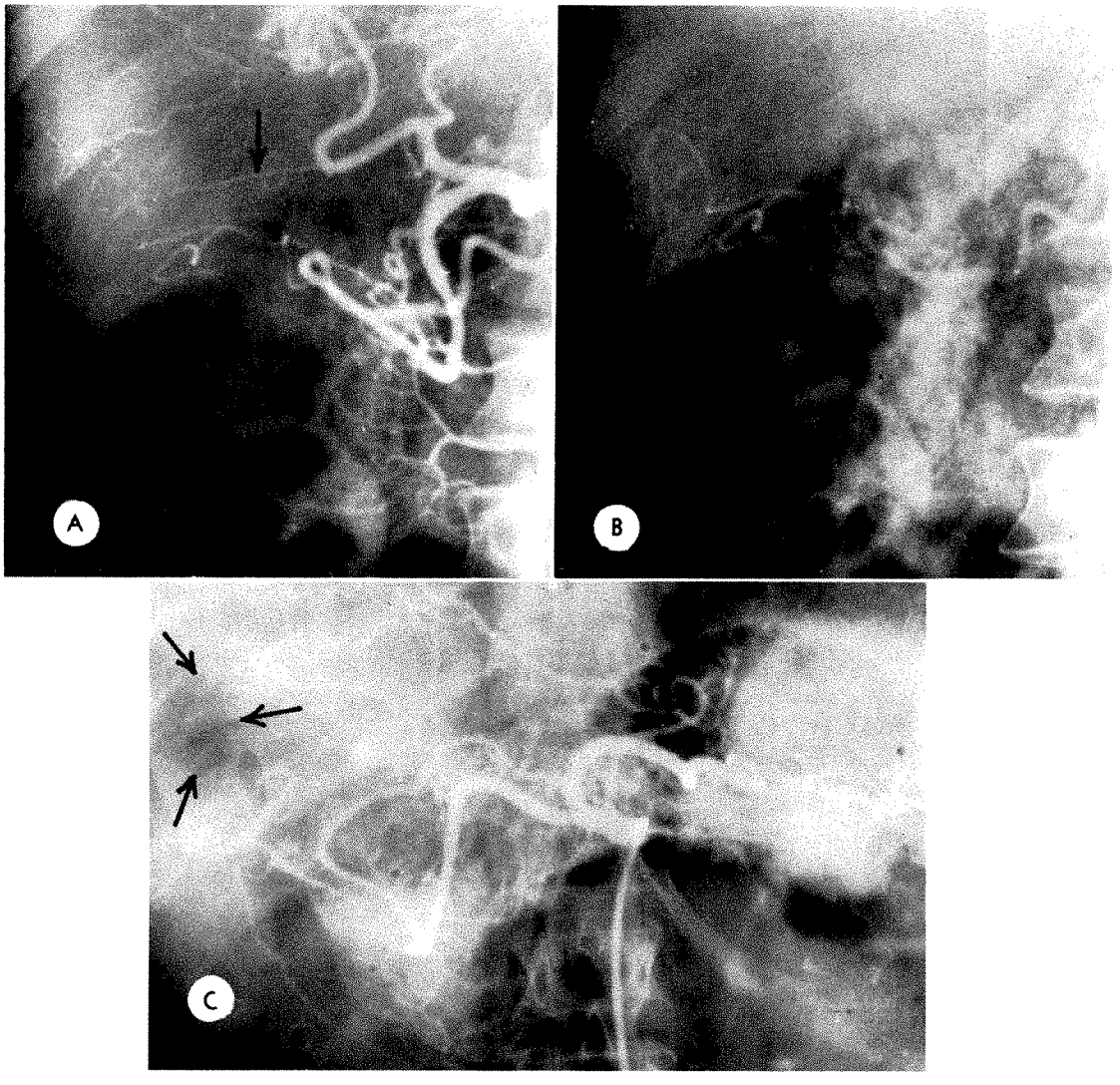



FIG. 6. Chronic cholecystitis. (*A*) Early arterial phase. The cystic artery is stretched (arrow). The gastro-epiploic artery with its epiploic branches is displaced and pulled to the right upper quadrant below the liver. (*B*) In the late arterial phase, the displaced epiploic arteries are still more prominent. The gallbladder wall cannot be demonstrated (see text). (*C*) Cholecystoduodenal fistula. The gallbladder is filled with air (arrows). The epiploic arteries are pulled in the direction of the gallbladder. The lower edge of the liver is blurred.

prominent than normal and displaced to the right, towards the lower margin of the liver, producing a bizarre and irregular appearance. In no case was a normal thin gallbladder wall demonstrated (Fig. 6*B*). Instead, the lower third of the right lobe of the liver showed irregular staining, causing blurring of its edge, beginning 3–4 seconds after injection and continuing until the late venous phase. This blurring probably is an expression of vascular channels in pericholecystic adhesions to the liver and/or omentum (Fig. 6*C*).

## DISCUSSION

Clinical, laboratory and routine roentgenologic findings often fail to reveal the pathology of the biliary tract. In obscure

cases of jaundice percutaneous transhepatic cholangiography may be helpful in visualizing extrahepatic obstruction, but should be used with caution and only on the day that operation is contemplated, because of the potential complications related to this procedure. In many cases selective celiac and mesenteric angiography has been found to be contributory by demonstrating lesions in the pancreas, liver and other organs. However, no attention has been paid to the angiographic diagnosis of gallbladder disease.

In our experience selective celiac and mesenteric angiography has elucidated many problems of biliary tract disease, having enabled us to diagnose various gallbladder diseases and differentiate malignant tumors of the gallbladder. In some of the cases in which tumor of the gallbladder has been diagnosed, biliary disease was clinically not suspected and the selective celiac and mesenteric angiography was performed in a search for some abdominal pathology or malignancy undetected by conventional roentgenologic methods. The same is true of one of the cases of hydrops, when a tumor was palpable below the right costal margin and considered to be liver metastasis. In another case a movable mass was palpable below the lower edge of the liver and angiography demonstrated a cystic lesion, probably an hydatid cyst.

The observations made with regard to chronic cholecystitis may prove helpful when angiography is performed in problems relating to the upper abdomen.

The demonstration of a normal or enlarged gallbladder in the presence of jaundice has some importance in that this would be observed in the cases of carcinoma of the head of the pancreas. It would not be demonstrated if the jaundice was caused by cholelithiasis, because of the nonvisualization associated with the attendant chronic cholecystitis.

When performing selective celiac and mesenteric angiography it is important to be aware of the possibility of the variable position or intrahepatic localization of the gallbladder because this may be misinterpreted as a ring shaped metastasis of the liver (Fig. 2*F*).

Concerning the indications for selective celiac and mesenteric angiography in gallbladder and biliary tract disease, in the present state of our knowledge, no clear-cut indications can be offered until more experience is gained. Generally, the indications should be related to cases of suspected malignancy, extrahepatic obstructive jaundice without a clear-cut history of stones, and finally to cases where masses palpable below the liver edge are not clearly defined by routine investigations.

Selective celiac and mesenteric angiography has been performed by us even in elderly patients whose tortuous iliac arteries can invariably be catheterized by a simple modification of the Seldinger guide wire.[2] The single absolute contraindication is sensitivity to the contrast material. We performed the investigation in cases with severe jaundice, uremia and/or thrombocytopenia, without complications. In one case, thrombosis at the puncture site required thrombectomy.

As to technique, selective right hepatic angiography in combination with the use of vasodilator drugs should improve the visualization of the gallbladder wall and we hope to investigate this in a future study.

The intention of this preliminary report is to encourage other investigators to verify our observations. If our findings prove to be consistent in a large number of patients, angiography could assume an important role in the differential diagnosis of gallbladder disorders. In cases of tumors of the gallbladder selective angiography also aids in defining the spread of the tumor and gives valuable information as to the operability and the surgical measures to be adopted.

SUMMARY

The normal gallbladder can be demonstrated by selective celiac and mesenteric angiography.

Examples of pathologic appearances and the differential diagnosis are presented.

While further studies will doubtless serve better to define the specific nosologic entities, our experience has shown that selective celiac and mesenteric angiography has a place in the evaluation of obscure disorders of the biliary tract.

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# THE EFFECT OF NEOLOID ON TELEPAQUE CHOLECYSTOGRAPHY*

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THE procedure of performing 2 or even 3 different roentgen examinations of the gastrointestinal tract on the same day has been accepted by many radiologists. Certainly the saving in patient time is a worthwhile consideration if there is no associated encroachment upon the quality of the roentgen examination.

We have been performing oral cholecystography prior to barium enema examinations on a number of patients for approximately a year and one-half and have found this to be a workable combination. The gallbladder examination is performed and carried to completion prior to the administration of the barium enema. It occurred to us, however, that the routine use of 4 ounces of neoloid* (emulsified castor oil, 36.4% W/W) on the evening prior to the barium enema examination might adversely affect the absorption of telapaque† (iopanoic acid, USP), which, of course, is also administered the evening before the examination.

Castor oil is the triglyceride of ricinoleic acid, an unsaturated fatty acid. In the intestine, the triglyceride is hydrolized by fat-splitting enzymes into glycerol and ricinoleic acid. Ricinoleic acid is a mucosal irritant which stimulates the motor activity of the small intestine by virtue of its irritating effect. Like other fatty acids the ricinoleic acid is largely absorbed prior to reaching the colon, and there is very little direct effect on the colon.[1]

It seemed possible that the irritation of the small intestinal mucosa by the ricinoleic acid and the increased motor activity of the small intestine might adversely influence the absorption of telepaque.

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† Winthrop Laboratories, New York, New York 10016.

## MATERIAL AND METHODS

We reviewed the films of 206 oral cholecystographies: 103 of the patients had had oral cholecystography without having received neoloid; 103 patients had received 4 ounces of neoloid on the afternoon prior to the gallbladder examination. These films were all reviewed by 2 radiologists and a rough quantitation of opacity of the gallbladder was recorded. Although we realized that this is a very subjective quantitation, we used the following guide to determine the degree of opacification:

0 No opacification
1 Faint opacification (nondiagnostic)
2 Fair opacification
3 Good opacification (gallbladder density equal to that of the vertebral bodies)
4 Excellent opacification (gallbladder density greater than that of the vertebral bodies)

The degree of opacification was graded on the original studies only, although repeat examinations were done on most of the patients in whom opacification was of a nondiagnostic quality initially. A total of 11 (10.7 per cent) diseased gallbladders were identified in the cholecystogram-alone group. There were 5 (4.9 per cent) diseased gallbladders in the group in which neoloid was also given. We do not feel that this small percentage of pathologic gallbladders significantly affected our conclusions.

Our routine preparation orders called for 4 ounces of neoloid to be administered at 4:00 P.M. the day prior to a barium enema examination and 6 telepaque tablets to be given at five-minute intervals beginning at 9:00 P.M. the evening prior to the gallbladder examination. From Table 1, it is

* From the Department of Radiology, Duke University Medical Center, Durham, North Carolina.

TABLE I

OPACIFICATION RESULTS

| Telepaque Alone | | | Telepaque and Neoloid | | |
|---|---|---|---|---|---|
| Degree of Opacification | Number of Cases | Per Cent | Degree of Opacification | Number of Cases | Per Cent |
| 0 | 14 | 13.6 | 0 | 2 | 3.7 |
| 1 | 5 | 4.8 | 1 | 9 | 8.7 |
| 2 | 8 | 7.7 | 2 | 15 | 14.6 |
| 3 | 36 | 35.0 } 73.8 | 3 | 29 | 28.1 } 74.8 |
| 4 | 40 | 38.8 } | 4 | 48 | 46.7 } |
| Total | 103 | | | 103 | |

apparent that neoloid administered at this interval prior to the telepaque does not appreciably interfere with the absorption of telepaque.

Actually there is a theoretic advantage to the administration of castor oil prior to gallbladder examination. Castor oil is a fatty substance and may cause contraction and emptying of the gallbladder which may enhance the opacification of the gallbladder by leaving it more free to accept the telepaque-laden bile.

## SUMMARY

A series of 103 neoloid-prepared barium enema patients had cholecystographies compared with 103 cholecystographies done on patients who did not receive neoloid.

Our results indicate that there is no appreciable interference with the absorption of telepaque by neoloid administered several hours prior to the telepaque.

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# HEPATIC ARTERIOVENOUS FISTULA AFTER PERCUTANEOUS LIVER BIOPSY*

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THE following is a brief report of a patient with an iatrogenic intrahepatic arteriovenous fistula after percutaneous liver biopsy. The fistula caused an audible continuous murmur and was demonstrated by selective celiac arteriography.

## REPORT OF A CASE

A 42 year old man with hepatomegaly, a pulmonary mass, and pleural effusion had had roentgenologic studies of the intestinal and urinary tracts. These showed downward displacement of the right kidney and hepatic flexure of the colon. A primary neoplasm was not discovered.

Because of an area of diminished uptake on an $Au^{198}$ liver scan, a clinical diagnosis of hepatoma with pulmonary metastasis was made.

Using a Vim-Silverman needle, two attempts were made to obtain a specimen of the liver at a site corresponding to the abnormal area in the photoscan. On biopsy, no evidence of malignancy was seen in either specimen. A continuous murmur was heard over the liver after the second biopsy.

A celiac arteriogram showed stretched intrahepatic arterial branches suggesting an intrahepatic mass. Evidence of a solitary arteriovenous fistula between branches of the hepatic artery and portal vein and of opacification of the needle tract was also seen (Fig. 1 and 2). No tumor vessels were noted. Selective studies of the superior mesenteric and right renal arteries were also done without any evidence of malignancy being revealed.

On laparotomy it was found that a large, unresectable retroperitoneal tumor replaced the right adrenal gland. It did not invade the liver.

*Necropsy Findings.* The patient died 10 weeks after the operation. Necropsy disclosed that a large adenocarcinoma of the right adrenal gland displaced, elongated, and deformed the liver in such a way as to explain the stretched hepatic artery branches seen on celiac arteriography. No intrahepatic tumor nor hepatic metastasis were found. An area of necrosis was seen on the posterior surface of the liver. Pulmonary metastasis was present.

## DISCUSSION

The complications of needle biopsy of the liver have been reviewed previously,[6,10] but the production of an intrahepatic arteriovenous fistula has not been mentioned. Traumatic fistulas between the portal vein and extrahepatic portion of the hepatic artery have been described.[4,5,8] If the fistulas are in the porta hepatis, they may be enclosed partly by the liver.[7]

The occurrence of renal arteriovenous

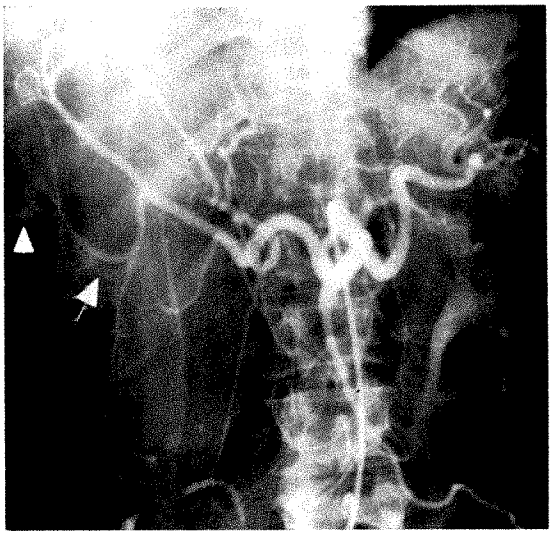


FIG. 1. Selective celiac arteriogram. Early phase shows the contrast medium passing from a branch of the hepatic artery into the needle tract (upper arrow) and the branch of the portal vein (lower arrow). Extrinsic pressure from carcinoma of the right adrenal gland distorts the liver and the branches of the hepatic artery supplying the right lobe, and also displaces the right kidney and gastroduodenal artery.

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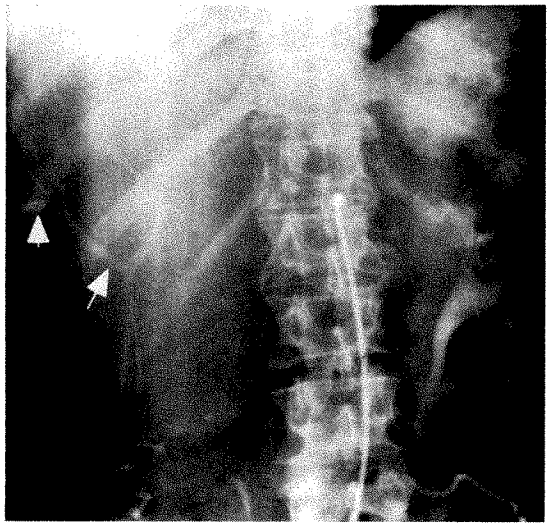


FIG. 2. Selective celiac arteriogram. Later phase. Most of the contrast medium has disappeared from the arteries. Contrast medium remains in the needle tract (upper arrow) and in the intrahepatic branch of the portal vein (lower arrow).

fistula after percutaneous renal biopsy is well known.[1,2,9] It is not unexpected that the same complication should occur after hepatic biopsy. The presence of a murmur over the liver, first heard after the second biopsy in the present patient was perhaps a helpful diagnostic clue. This murmur must be differentiated from the arterial murmur[3] that may be heard in benign or malignant liver tumors and hepatitis, and from the venous hum of collateral circulation.[3] Celiac arteriography helped in this differentiation. The hepatic congestion found at necropsy may have resulted from a pulsating portal hypertension secondary to the hepatic artery-portal vein fistula, with differing levels of pressure in these 2 vessels.

SUMMARY

The visualization by selective celiac arteriography of an arteriovenous fistula within the liver between a branch of the hepatic artery and a branch of the portal vein, caused by percutaneous liver biopsy, is described.

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Dr. John R. Amberg has reviewed the manuscript and has given helpful advice.

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# IPODATE AND THE NONFUNCTIONING GALLBLADDER*

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IN RECENT years, our Department of Radiology, in common with many others, has been looking for ways to streamline many of our studies, as an answer to the constantly increasing volume of work. The classical gallbladder series, with repeat dose, 24 hour cholecystograms for nonvisualization, has consistently delayed our patients in their diagnostic work-up. In an attempt to reduce the time required to examine a gallbladder, this study was undertaken to determine if the addition of ipodate (oragrafin) granules to the routine dose of iopanoic acid (telepaque) would cause a nonvisualized gallbladder to fill. Incidental to this primary intent, a study was also undertaken to determine if a cause for the nonvisualization other than disease could be found in the patients' symptoms or hospital course. It is well known that many gallbladders without apparent disease will not opacify after routine doses of contrast medium.

Many papers have been written about ipodate, both in capsule and granule form, mainly concerned with visualization of the common duct and with the relative density of ipodate and other contrast media.[1–8,11,12] The general feeling appears to be that gallbladder visualization using ipodate alone is comparable with that obtained with other media and that side effects are less with ipodate. There are varying reports concerning the visualization of the ducts, the consensus being that while ipodate is more convenient and safer, the filling of the common duct is, at best, only slightly better than with other oral media and not as good as with methylglucamine iodipamide (cholografin).[1–8, 10–12]

Two studies performed along the lines of this one showed definite improvement in visualization of the gallbladder after ipodate granules in patients whose original cholecystograms showed poor concentration.[9,10] This study will attempt to confirm these findings with more patients and to comment upon common duct filling.

## MATERIALS AND METHOD

The routine gallbladder series in our department has consisted of the administration of telepaque tablets, by body weight, after a low-fat supper, on the evening before the examination. In the morning, posteroanterior and oblique roentgenograms were taken of the right upper quadrant. If the gallbladder did not properly appear, the patient received a second dose of tablets that night and returned the following day for more roentgenograms (24 hour examination).

During the present series, if the gallbladder was not properly visualized on routine roentgenograms, 2 packets (6 gm.) of ipodate granules, dissolved in water, were administered and the patient asked to lie on his right side. Three hours later, repeat roentgenograms were made. If the gallbladder remained unfilled, the usual "24 hour examination" was performed.

A group of 114 consecutive patients was studied in this manner. The hospital charts were then reviewed to determine the patients' symptoms, signs and ultimate course. Nurses' notes were read in an effort to discover reasons for nonvisualization.

## RESULTS

The age and sex distribution were slightly slanted toward the older, male patient, possibly because many examinations were ordered for the patient with chest pain. The actual incidence of roent-

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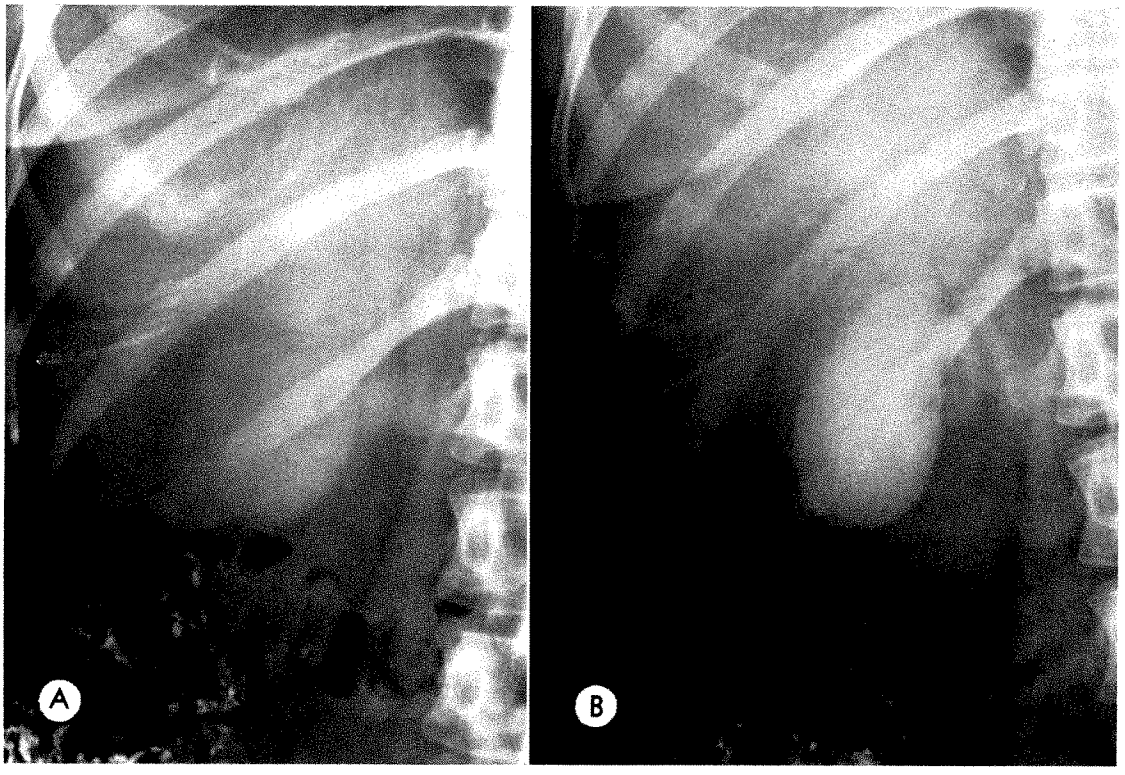



Fig. 1. (*A*) Cholecystogram 12 hours after routine dose of telepaque. (*B*) Cholecystogram 3 hours later, after dose of 6 gm. of ipodate granules. Note filling of the common duct.

genographic disease is about equal in the sexes and is quite consistent in the age groups—about 33 1/3 per cent (Table I and II).

Table I

AGE DISTRIBUTION

| Age (yr.) | Patients | Roentgenographically Diseased |
|---|---|---|
| 0–20 | 2 | 0 |
| 21–30 | 11 | 2 |
| 31–40 | 10 | 2 |
| 41–50 | 22 | 7 |
| 51–60 | 29 | 10 |
| 61–70 | 24 | 10 |
| 71– | 16 | 9 |

Table III shows that by far the largest number of patients demonstrated roentgenographically normal gallbladders after the ingestion of ipodate. Three patients, although roentgenographically normal, were operated and discovered to have "chronic cholecystitis without cholelithiasis." Nine patients were enough improved to definitely demonstrate stones. Of 38 patients who did not sufficiently improve to allow positive diagnosis, 18 were proven diseased at surgery, 6 were roentgeno-

Table II

SEX DISTRIBUTION

| | Patients | Roentgenographically Diseased |
|---|---|---|
| Male | 62 | 19 |
| Female | 52 | 21 |

Table III

RESULTS

| | Surgically Diseased | Normal (No Surgery) | No Follow-Up |
|---|---|---|---|
| Improved with Ipodate | 9 | 67 | 0 |
| Not Improved with Ipodate | 18 | 6 (on later study) | 14 |

graphically normal on later studies and 14 were not operated on or further examined, for various medical reasons (5 had diagnoses of pancreatitis or cirrhosis).

Table IV demonstrates that of 25 patients who showed no visualization after the granules and had received a "24 hour examination," 7 improved enough to allow a definite diagnosis. The granules, then, cannot eliminate entirely the 24 hour study.

Table V illustrates that the common duct was visible enough in 47 examinations (41 per cent) to rule out significant obstruction. The duct was more liable to be visible in a patient with a normal gallbladder than in a patient with a roentgenographically abnormal one. No fatty meal was given and no attempt was made to use ipodate to visualize ducts in cholecystectomized patients.

## COMMENT

It is apparent that many poorly visualized gallbladders can be rendered visible by the ingestion of ipodate granules. In most instances, definite diagnosis can be made within 18 hours of the original administration of opaque medium, rather than waiting 36 hours. A whole day may be saved and other diagnostic studies may be performed. In our series, 66 per cent of patients with poorly visualized gallbladders were definitely diagnosed within this time. Of 38 patients not definitely diagnosed because of continued nonvisualization, 18 were later proven diseased, 6 were roentgenographically normal on later studies and 14 were not followed up, for various reasons. No patients were roentgenographically abnormal and surgically proven to be normal.

Table IV

24 HOUR STUDIES

| | Surgically Diseased | Normal |
|---|---|---|
| Improved for Diagnosis | 3 | 4 |
| Not Improved for Diagnosis | 11 | 7 (no follow-up) |

By far the largest number of examinations consisted of patients with roentgenographically abnormal gallbladders who ultimately showed normal organs after ipodate granules. Careful perusal of charts revealed no consistent reason why these patients should have nonvisualization after iopanoic acid and good filling after ipodate. There were many more patients with absent or very vague upper gastrointestinal symptoms than there were with classical complaints and only 5 patients experienced side effects after telepaque (mild nausea). This problem remains unsolved.

It is to be noted that only 6 patients experienced side effects after the ingestion of ipodate granules, 4 with nausea and 1 each with diarrhea and vomiting.

Although the common duct was partially visible in almost half the patients, most of these patients had normal gallbladders. It is not proven that ipodate granules can

Table V

COMMON DUCT FILLING

| | Well Filled Gallbladder | Poorly Filled Gallbladder |
|---|---|---|
| No Filling | 36 | 31 |
| Partly Filled | 26 | 6 |
| Fully Filled | 14 | 1 |

substitute for cholografin in the examination of the common duct in the patient with a diseased or absent gallbladder.

The greatest value of the ipodate granules appears to be in the elimination of the time-consuming "24 hour study" in the patient with the anatomically normal gallbladder who, for some inexplicable reason, does not fill it with opaque medium on routine examination. The granules do not completely eliminate the "24 hour study," however. There were several patients in this study, who, having had the granules without improvement, went on to visualize normally in 24 hours.

SUMMARY

A group of 114 patients with poorly filled gallbladders after routine administration of iopanoic acid were given 6 gm. of ipodate granules immediately following their routine cholecystography. In 66 per cent of these patients opacification of the gallbladder was obtained within 3 hours, allowing a definite diagnosis. Of the remaining 33 per cent, one-half were ultimately proven diseased. The common duct was incidentally visualized in 40 per cent of all patients, most of whom demonstrated normal gallbladders.

A detailed study of patient records did not reveal any information which could explain the nonvisualization of apparently normal gallbladders on routine study.

The ipodate granules are thought to be an effective means of shortening the time expended on gallbladder diagnosis.

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# AGENESIS OF THE GALLBLADDER*

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CONGENITAL absence of the gallbladder is a rare anomaly. About 150 cases have been reported,[4] of which two thirds were discovered at surgery and the remainder were diagnosed on postmortem examination. The purpose of this report is to focus attention once more on this abnormality.

## ILLUSTRATIVE CASE

E.K., B.C.H. #2049218. This 55 year old Negro female was admitted to the Boston City Hospital on January 8, 1966 with epigastric and periumbilical pain of 2 days' duration. Two months prior to admission she had been hospitalized because of a similar episode consisting of nausea, vomiting, and severe right subcostal pain after meals. Oral cholecystography failed to opacify the gallbladder. Intravenous cholangiography was performed and a normal common bile duct was demonstrated. Neither the gallbladder nor the cystic duct was visualized (Fig. 1). Her symptoms subsided spontaneously and she was discharged. On the present admission she reported right upper quadrant pain aggravated by greasy food and several episodes of vomiting. Past history revealed that she had undergone an appendectomy at the age of 42 and gynecologic surgery at the age of 43 years. These operations were performed in South Carolina and an abstract of the hospital admissions could not be obtained.

On physical examination, she had localized tenderness in the epigastrium and the right upper quadrant. The uterus was palpated and was not abnormal. There was a short right lower quadrant scar and a suprapubic scar. Hematocrit was 36 per cent, white blood cell count 7,500 and amylase 100 Bessy-Lowry units. An upper gastrointestinal series was within normal limits. A laparotomy was performed on January 21 with the preoperative diagnosis of cholecystitis. When the gallbladder was not found, an intraoperative cholangiogram was done. This revealed absence of the cystic duct and the gallbladder (Fig. 2). The common duct was explored from the confluence of the hepatic ducts to the duodenum; it was of the normal size and consistency. No cystic duct could be found. There was no evidence of any prior right upper quadrant surgery. A right renal cyst was noted and biopsied; the liver was biopsied and the abdomen was closed. The postoperative course was complicated by segmental atelectasis of the lung from which the patient recovered uneventfully and she was discharged on February 2. Two months later she again complained of right upper quadrant pain.



FIG. 1. Intravenous cholangiogram demonstrates a normal common bile duct; however, neither the gallbladder nor the cystic duct is visualized.

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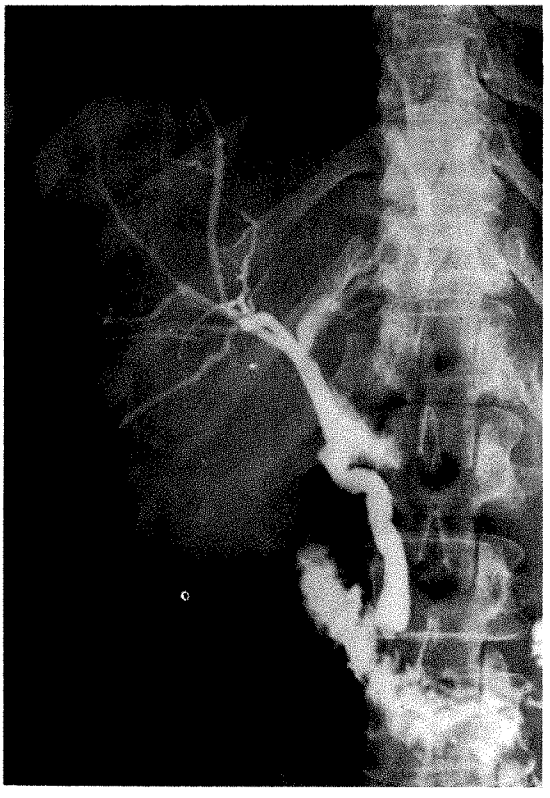


FIG. 2. Intraoperative cholangiogram obtained reveals absence of the cystic duct and the gallbladder.

## DISCUSSION

Agenesis of the gallbladder and the cystic duct is attributed to an abnormality in the embryologic development. This occurs in the 3 mm. stage of the embryo as a result of failure in development of the caudal portion of the hepatic sacculation, which is the forerunner of the gallbladder. Absence of the gallbladder alone may also occur because of failure of the solid stage to re-establish a lumen by the 15 mm. embryonic stage. In this instance the cystic duct would be present.

It is not possible to make a preoperative diagnosis of this condition as it has no characteristic clinical or roentgenologic features. Kobacker[2] suggested that this may be a hereditary condition, but this assumption has not been verified because of the lack of adequate studies.

Unlike many of the cases previously reported, this patient had neither jaundice nor common duct stones at surgery. The reason for her symptoms was not discovered. Dixon and Lichtman[1] collected 50 cases from the literature suitable for analysis and added 10 cases of their own; 35 of these 60 cases had symptoms of cholecystitis, 13 did not, and in 12 they were not described. Twenty-nine cases had jaundice; 4 cases had stones in the common bile duct; the common bile duct was dilated in 19, of normal size in 20, and not mentioned in 21. On the basis of these figures the authors concluded that "congenital absence of the gallbladder is a distinct disadvantage." This impression is drawn from the relatively high frequency of biliary dysfunction in cases of gallbladder agenesis, but is not a conclusion supported by statistical studies. Until there are data comparing the frequency of biliary tract disease in cases of congenital absence of the gallbladder with its frequency in the general population, the morbidity of congenital absence of the gallbladder will probably remain unknown. There is no evident reason why the congenital absence of the gallbladder should be any more difficult to tolerate than its surgical removal.

Speculation as to the mechanisms of biliary function and dysfunction associated with gallbladder agenesis is abundant; thus Rogers *et al.*[3] postulate contraction of the common duct in response to cholecystokinin release. Facts, however, are scarce, and this infrequent congenital anomaly remains of doubtful significance and obscure pathophysiology.

## SUMMARY

A case of congenital absence of the gallbladder and cystic duct is reported. The finding of an absent gallbladder in our case is considered incidental and the cause for symptoms could not be attributed to the

absence of the gallbladder and the cystic duct.

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# PSEUDOCYSTS OF THE PANCREAS

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ALTHOUGH the medical literature is replete with reports of pseudocysts of the pancreas, these deal primarily with the medical and therapeutic aspects.[7,12,21,24,25] The paucity of the radiologic literature on this subject prompted us to present this review of several cases, emphasizing the roentgen findings.

Various classifications of pancreatic cysts are available. A useful and simple classification has been suggested by Thigpen and presented by Brilhart and Priestley[2] as follows: (1) pseudocysts (inflammatory, traumatic); (2) retention cysts; (3) neoplastic cysts (cystadenomas, cystadenocarcinomas, sarcomas, teratomas); (4) congenital cysts; (5) parasitic cysts; and (6) miscellaneous pancreatic cysts

Although we are considering primarily pseudocysts in this discussion, some comments are appropriate regarding other types of pancreatic cysts.

Retention cysts are believed to result from obstruction of one of the major pancreatic ducts and have been described in association with pancreatic lithiasis, chronic pancreatitis, and trauma. They have an epithelial lining and contain exocrine secretions of the pancreas. Retention cysts occasionally may be confused with pseudocysts since the epithelial lining of the former may undergo pressure atrophy. Neoplastic cysts of the pancreas are usually multilocular and are occasionally seen in association with adenocarcinoma of the pancreas. However, the most common neoplastic cyst of the pancreas is the cystadenoma. Sarcomatous and teratomatous cysts are rare, as are congenital pancreatic cysts (dysontogenetic cysts). Parasitic cysts, usually the result of the Echinococcus, are seldom encountered in the United States.

The pancreas is the only organ in the body which reacts to trauma or inflammation by autolysis and the formation of a pseudocyst.[19] The location of the pancreas astride the vertebral column makes it particularly vulnerable to blunt trauma. Some authors report a traumatic etiology in as high as 20 to 50 per cent of cases.[4,9] One half of the approximately 35 reported cases of pancreatic pseudocysts in children have had a traumatic etiology.[6,10,16,20] In adults, however, interstitial or hemorrhagic pancreatitis is the most frequent cause of pseudocyst formation. The initial insult to the pancreatic acini, whether traumatic or inflammatory, results in the escape and activation of trypsinogen and lipase with progressive autolysis and destruction of the parenchyma. The pancreas usually responds to such injury by pouring out large amounts of fluid. The activated enzymes are most commonly released into the lesser omental sac by rupturing through the anterior wall of the pancreas, resulting in a severe chemical peritonitis with the production of profuse inflammatory exudate by the peritoneal and serosal surfaces. This exudate seals off the foramen of Winslow with encystment of the fluid in the lesser omental sac forming a pseudocyst.

The fluid found in pancreatic pseudocysts varies in appearance and consistency. Generally, cysts secondary to chronic pancreatitis contain clear or turbid fluid which may be mucoid, whereas cysts secondary to acute pancreatitis or pancreatic necrosis may contain necrotic tissue particles and hemorrhagic fluid. In one series of pancreatic pseudocysts, the average amount of fluid was 2,686 cc. with a range from 15 cc. to 7 liters.[19] In this same series, 9 of 12 cases contained enzymes in the fluid. The presence of trypsin in the

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fluid of the pseudocyst is proof of a pancreatic origin as is an amylase level markedly elevated over that in the serum.[22] An absence of enzymes in the fluid does not, however, exclude a pancreatic origin.

Pancreatic pseudocysts may rupture spontaneously or secondary to trauma with a fatal outcome in 75 per cent of cases.[11,15] The tear usually occurs in the gastrocolic or gastrohepatic omentum causing the fluid to enter the peritoneal cavity with subsequent peritonitis.[17] Such an occurrence can result in the sudden disappearance of a palpable abdominal mass and can resemble a perforated peptic ulcer clinically. Perforation into the stomach, small bowel and colon can occur.[11] A very unusual case of rupture of a pancreatic pseudocyst into the portal vein with secondary nodular subcutaneous and generalized fat necrosis has been reported.[26] In this case, the pancreatic ferments gained direct entry into the venous system via the portal vein.

Pain is the most frequent symptom of pancreatic pseudocyst, while the most common physical finding is a palpable mass.[2,3,14] Other features observed are nausea, vomiting, fever, diarrhea, abdominal tenderness, hydrothorax and icterus. Alcoholism is an extremely frequent factor. In a recent medical review of patients with pancreatic pseudocysts, 57 per cent showed glycosuria or hyperglycemia or both;[3] 40 per cent had elevated serum amylase levels; 19 per cent had anemia; and 14 per cent revealed serum bilirubin levels greater than 2.9 mg. per 100 ml.

The plain roentgenogram of the abdomen in a patient with a pancreatic pseudocyst may demonstrate a soft tissue mass and/or displaced air filled loops of bowel. Pancreatic calcifications may be present, indicating chronic pancreatitis. Two cases in which a pancreatic pseudocyst was defined by displacement of pre-existing pancreatic calcification have been described.[1] One case in which the pancreatic pseudocyst was densely outlined by calcium in its wall has been reported.[13] In most cases, plain roentgenograms are not contributory and contrast studies of the upper gastrointestinal tract, small bowel, colon and urinary tract are necessary.

The most common sites of pancreatic pseudocysts are: (1) between the stomach and transverse colon, bounded anteriorly by the gastrocolic ligament; (2) between the stomach and liver bounded anteriorly by the gastrohepatic omentum; and (3) between the leaves of the transverse mesocolon. Pseudocysts may present in other locations depending on the site of origin within the gland and by virtue of the fact that they may dissect through the tissue planes, taking the path of least resistance. Four pseudocysts of the mediastinum have been reported indicating the manner in which these lesions may "travel."[5,8,10,18] Three of these cases had pseudocysts in the lesser omental sac and spread to the mediastinum via the esophageal or aortic hiatus. In the fourth case, the mediastinal cyst passed through the aortic opening into the region of the body and tail of the pancreas, but no pseudocyst was found in the lesser sac. Presumably the connection between the mediastinum and pancreas was entirely retroperitoneal. Other unusual locations reported include one pseudocyst found between the layers of the sigmoid mesocolon in the pelvis[14] and one attached to the superior pole of the left kidney.[23]

## MATERIAL

We have reviewed 21 cases in which positive roentgen studies and surgical or autopsy proof are available. Of these patients all but one were alcoholics of long standing, although one of the alcoholic patients had a history of recent abdominal trauma as well. Another alcoholic patient returned 21 months after the pseudocyst was surgically diagnosed with adenocarcinoma of the pancreas, suggesting that the originally diagnosed pseudocyst may have been a cystadenocarcinoma, although review of the original sections again failed to reveal evidence of neoplasm. It is conceivable that a pseudocyst developed

secondary to the release of enzymes produced by the neoplastic destruction of the pancreas. The one patient who was not alcoholic had had a splenectomy which may have traumatized the pancreas with release of enzymes and formation of a pseudocyst. The ages of our patients ranged from 26 to 59 years. All were males, but this may be a reflection of the overwhelmingly male population of this hospital.

Only 2 patients presented without pain as the chief complaint. In some, the pain was generalized while in others it could be localized, usually to the left upper quadrant. The pain varied from mild to so severe that the patient could not stand erect. Five of the patients had associated vomiting and 4 reported weight loss. Only 8 of the patients had a palpable mass on physical examination. When a mass was palpated it was variably described as cystic or solid. About half of the patients described tenderness of the abdomen to palpation which was generalized or localized, but others presented with soft nontender abdomens.

Of our 21 patients, 7 had hyperglycemia, glycosuria or both; 6 patients had a significant anemia on admission; only 1 patient was clinically jaundiced. None of the others had a bilirubin level over 2.9 mg./100 cc. Serum amylase levels were reported on 19 patients but were elevated in only 8 of these.

### ROENTGEN FINDINGS

While the findings on the plain abdominal roentgenogram were not often helpful, there was at least a suggestion of a soft tissue mass in several cases. Only 3 patients had typical pancreatic calcification and in none of these was it very extensive. One patient's abdominal roentgenogram showed a fine arcuate calcification similar to that seen in aneurysms. This was located anteriorly and to the left of the spine (L2) and later proved to be situated within the wall of a pseudocyst.

In most cases the presence of a mass was noted only by displacement of contrast-filled hollow viscera. The most common roentgen finding was anterior displacement of the stomach, present in 15 of our cases either with or without additional findings. This is not surprising in view of the fact that most pseudocysts are found within the lesser sac alone or in combination with extensions of the pseudocysts. The gastric displacement may be secondary to a well defined mass or may be of a more diffuse nature without a localized mass being seen. The stomach may also be displaced either laterally or medially. In 2 cases a localized defect of the fundus was the prime abnormality noted. A clearly demarcated mass most often presents posterior to the body of the stomach. Four of our cases, however, revealed a mass impressing the greater curvature of the stomach. Again, this could be a generalized elevation of the antrum or antrum and body, or pressure outlining a more localized mass. Frequently the first and second portions of the duodenum were displaced anteriorly, but in all cases in which this was noted, the stomach was also displaced anteriorly. The duodenal loop appeared widened in the anteroposterior projection in only 2 of our cases, and this was probably due to elevation of the antrum rather than an increased duodenal sweep. In 1 case the first and second portions of the duodenum were far to the left of their normal location without a demarcated mass being seen. In another, the only change noted was medial displacement of the lateral aspect of the second portion of the duodenum with fixation of the folds.

The colon was depressed in 1 case and elevated in another. In 3 of our cases the splenic flexure showed localized abnormality. In 2 of these it was displaced inferiorly and anteriorly, while in the other it was displaced medially. Two of these cases also revealed inferior displacement of the left kidney.

In several patients there was great concern regarding the presence of malignant neoplasm based on fixation of the wall of

the displaced organ associated with the presence of abnormal mucosal folds. In 3 cases this involved the splenic flexure of the colon, in 1 the descending duodenum and in 1 the duodenal jejunal junction. This was also a problem in the cases involving the gastric fundus. Frequently, an intramural lesion was strongly suggested in the differential diagnosis.

Only 1 patient had selective celiac and superior mesenteric arteriography performed, and this revealed complete obstruction of the splenic vein. A second patient, previously reported,[9] had splenic vein obstruction demonstrated at autopsy examination.

## REPORT OF CASES

Case I. F.F.P. This 59 year old white male alcoholic developed epigastric and left upper quadrant pain 2 weeks prior to admission. The physical examination was normal except for slight epigastric tenderness. All laboratory studies were within normal limits except for a fasting blood sugar of 566 mg./100 cc. An upper gastrointestinal series (Fig. 1, *A* and *B*) revealed a large mass posterior to the stomach with a suggestion of invasion of the wall of the stomach. At laparotomy a large pancreatic pseudocyst was found and the histologic examination revealed intramural acute and chronic inflammation and necrosis from several areas of the gastric wall. Cyst-gastrostomy was performed.

Case II. B.F.D. This 43 year old white male alcoholic was a known diabetic with a history of recurrent pancreatitis. He was admitted because of recurrent abdominal pain and on examination an epigastric mass was palpable. Upper gastrointestinal series (Fig. 2, *A–C*) revealed an arcuate calcification within a mass located posteriorly and superior to the antrum. Some flocculent pancreatic calcification was also noted. At surgery a pseudocyst of the pancreas was found and a cyst-gastrostomy performed.

Case III. B.C. This 39 year old white male alcoholic had had recurrent attacks of acute pancreatitis for about 2 years. A cholecystectomy had been performed 5 months earlier. Four days prior to admission he developed severe abdominal pain and on admission a cystic mass was palpable to the left of the midline under the costal margin. Amylase levels were elevated, but all other laboratory studies were normal. Gastrointestinal series (Fig. 3, *A–C*) revealed a large posterior mass displacing the stomach anteriorly with a second impression on the greater curvature of the body and antrum. At surgery a pancreatic pseudocyst was

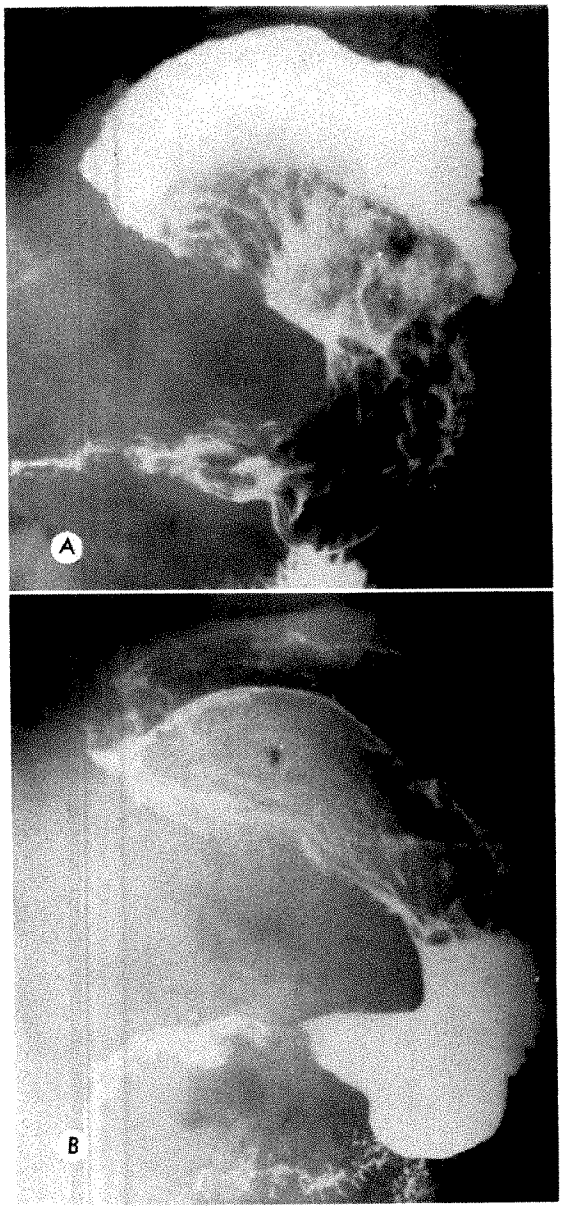



Fig. 1. Case I. (*A* and *B*) Gastrointestinal series revealing a large mass posterior to stomach with fixation to wall and areas suspicious for mucosal invasion. This is the most common location of pancreatic pseudocysts.

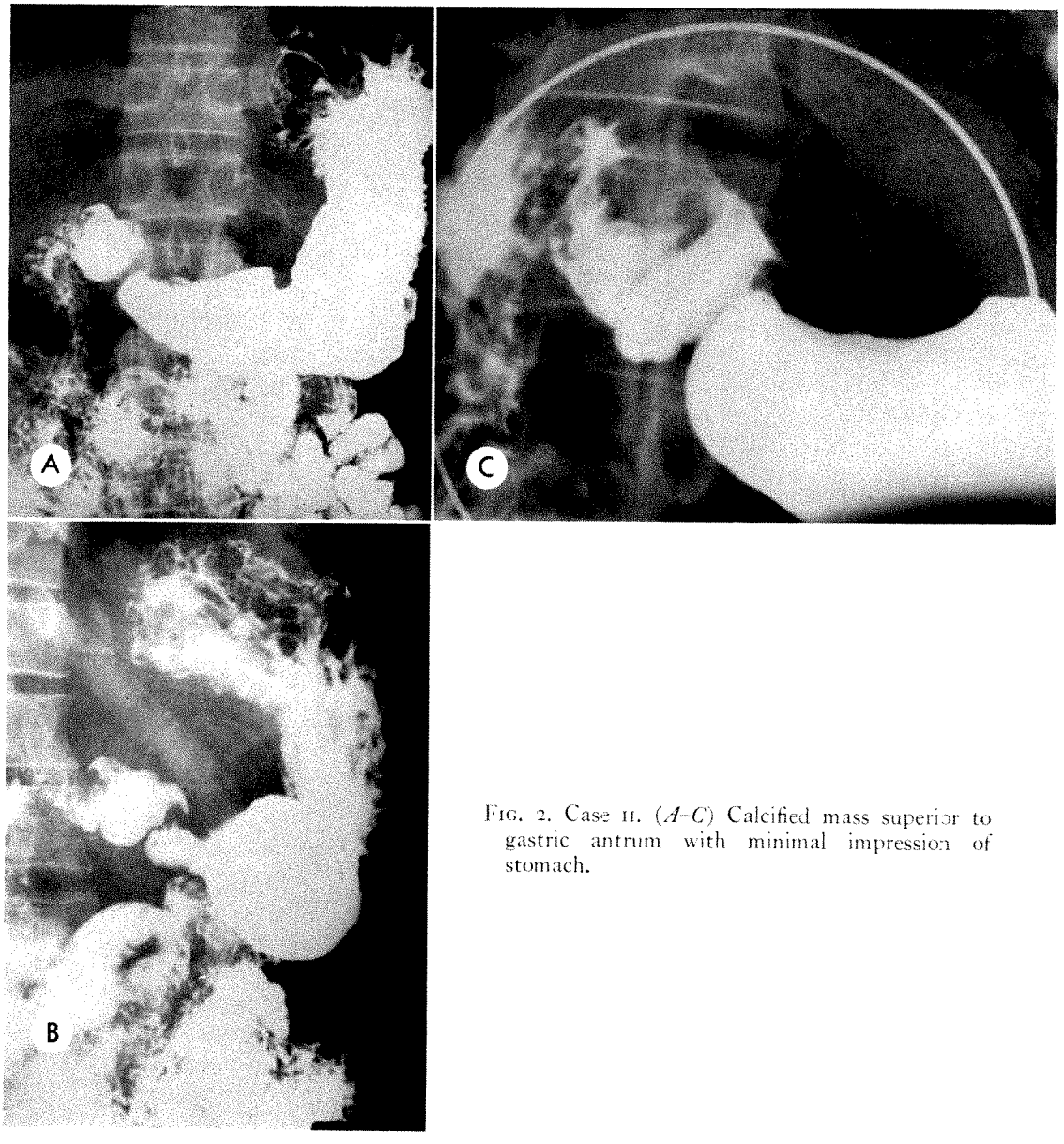



FIG. 2. Case II. (*A–C*) Calcified mass superior to gastric antrum with minimal impression of stomach.

found posterior to the stomach with a second component located along the greater curvature. Cyst-gastrostomy was done.

CASE IV. P.R.R. This 38 year old white male alcoholic presented complaining of intermittent right upper quadrant pain and being "full of fluid." Physical examination revealed ascites and a probable mass in the left upper quadrant. The hemoglobin was 8.9 gm./100 cc. but the remaining laboratory studies were normal. A gastrointestinal series (Fig. 4, *A* and *B*) revealed marked anterior displacement of the stomach and duodenum with separation of the cardia from the diaphragm. The patient died within a few days and an autopsy revealed a large pseudocyst 10 cm. in diameter involving the left lobe of the liver, spleen, stomach and small intestine.

CASE V. V.T. This 51 year old Negro male alcoholic was admitted complaining for two or three days of severe abdominal pain and vomiting. Physical examination revealed tenderness

of the abdomen and an enlarged liver. Serum amylase and lipase levels were normal. The blood sugar was 566 mg. per cent, the white blood cell count was 15,900/cu. mm. with a shift to the left, but the hemoglobin and hematocrit levels were normal. Gastrointestinal series (Fig. 5, *A* and *B*) revealed extrinsic pressure on the greater curvature of the stomach associated with anterior displacement of the stomach and questionable destruction of the mucosa at the duodenal jejunal junction. Laparotomy was performed and revealed a very large pseudocyst of the pancreas extending to the undersurface of the spleen, stomach and liver. It was described as the size of a small basketball.

CASE VI. F.J.M. This 50 year old white male had had previous admissions for chronic alcoholism and gastritis and for Leriche syndrome at which time a bilateral aortofemoral by-pass procedure had been performed successfully. Nine days prior to admission the patient developed nausea and vomiting, following the ingestion of 20 "shots" of whiskey, which persisted despite treatment with meperidine. On admission the blood pressure was 130/90 and pulse 103/min. Abdominal examination revealed slightly hypoactive peristalsis and slight epigastric tenderness but no palpable masses. The patient was treated with intravenous fluids and an upper gastrointestinal series (Fig. 6, *A* and *B*) was performed. This was interpreted

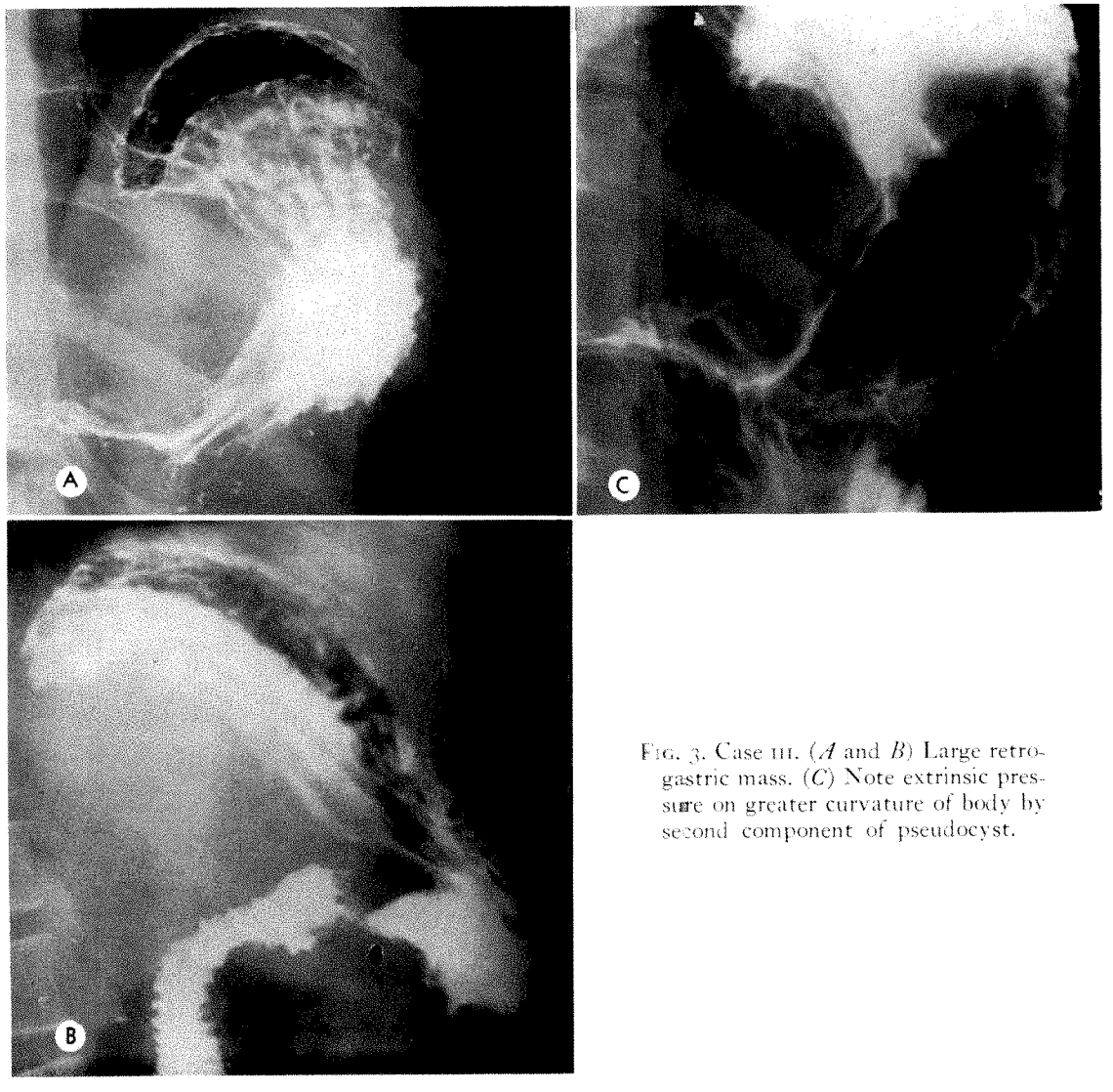



FIG. 3. Case III. (*A* and *B*) Large retrogastric mass. (*C*) Note extrinsic pressure on greater curvature of body by second component of pseudocyst.

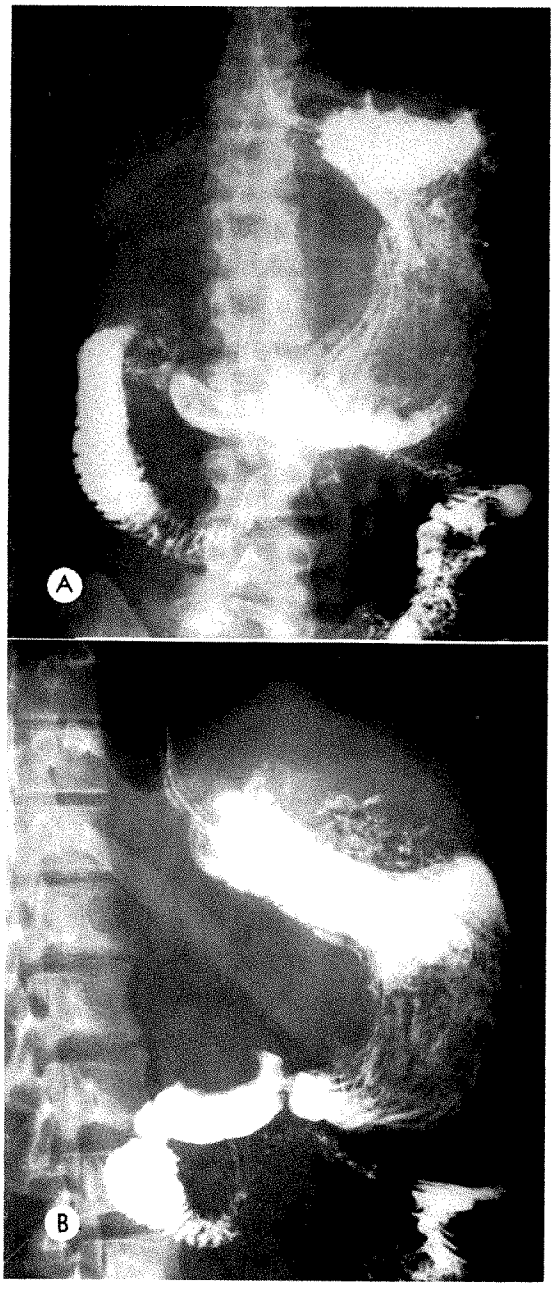



FIG. 4. Case IV. (*A* and *B*) Mass separating gastric fundus and diaphragm. Note also impression on stomach and proximal jejunum with fixation of latter.

as showing partial obstruction of the duodenum by a lesion which had the characteristics of an intramural extramucosal lesion but could also be entirely extrinsic. At surgery the mass was found to represent a large pseudocyst of the pancreas.

CASE VII. R.K. This 49 year old white male alcoholic was admitted with a 4 month history of intermittent left upper quadrant pain. The serum amylase ranged from 200 to 1,000 and

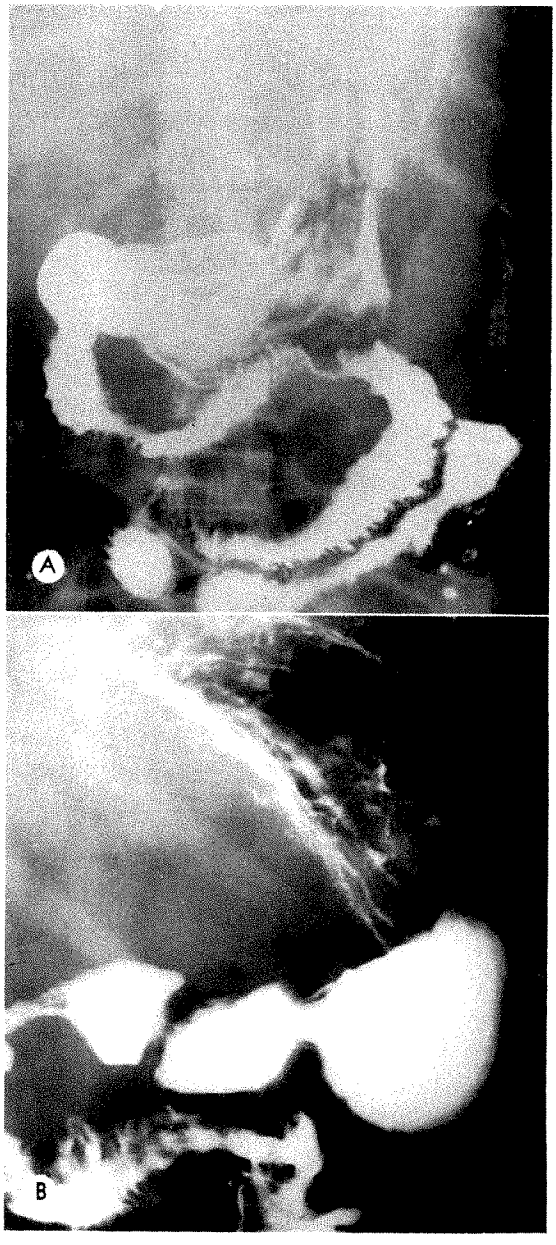



FIG. 5. Case V. (*A* and *B*) Anterior displacement of stomach with abnormality of small intestine most marked at duodenal jejunal junction where mucosal invasion was strongly suspected.

the lipase from 1.5 to 4. Barium enema examination (Fig. 7, *A* and *B*) revealed a mass on the medial aspect of the splenic flexure believed to be either extrinsic or intramural. Gastrointestinal series revealed a subtotal gastrectomy and gastrojejunostomy with some anterior displacement of the gastric remnant. At surgery a

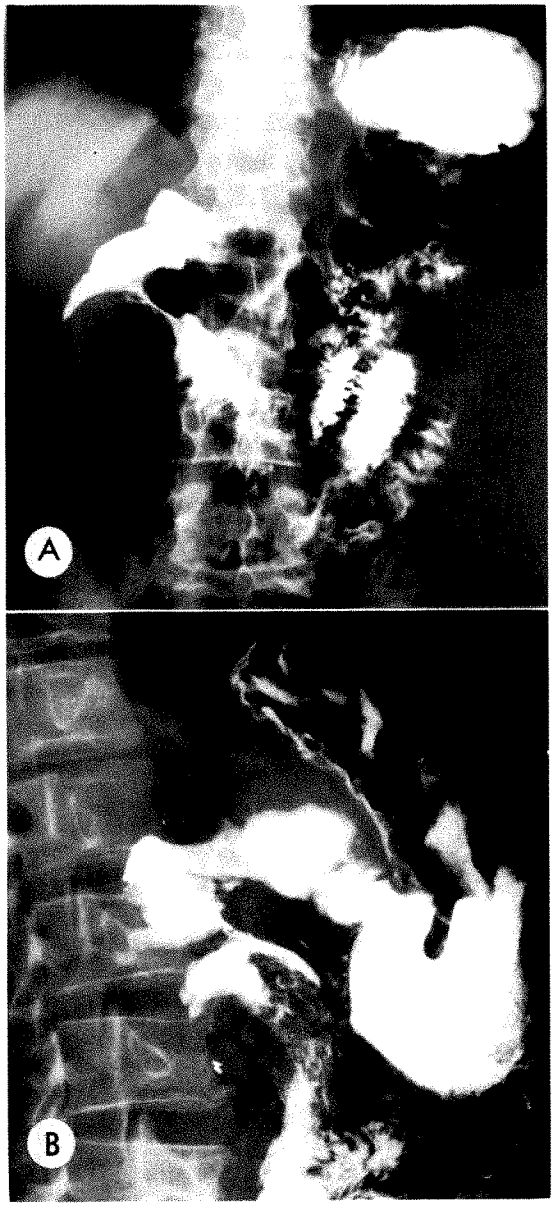



Fig. 6. Case VI. (*A* and *B*) Anteroposterior and lateral views showing mass displacing descending duodenum medially with intramural lesion suggested.

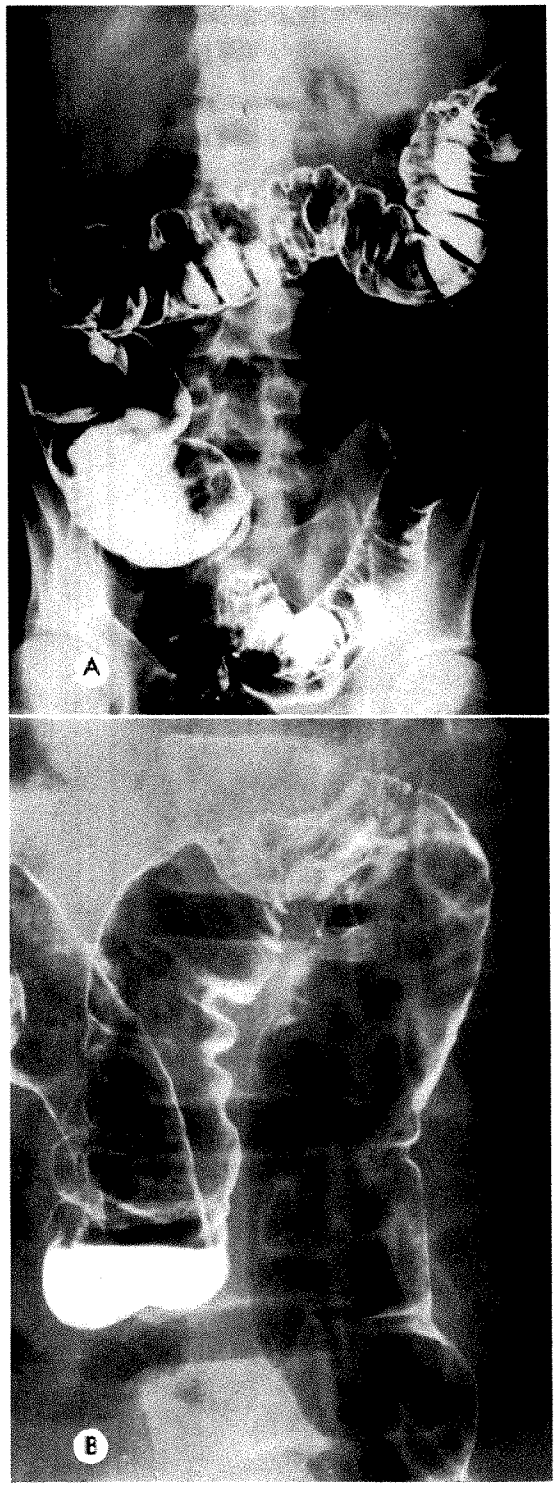



Fig. 7. Case VII. (*A* and *B*) Mass fixed to superior aspect of splenic flexure.

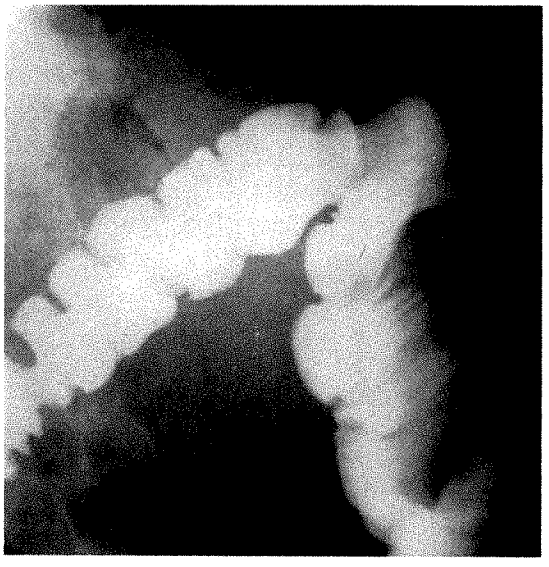


FIG. 8. Case VIII. Spot roentgenogram from barium enema examination revealing extrinsic pressure on posterolateral aspect of splenic flexure.

large pseudocyst was found involving the tail of the pancreas, the spleen and the colon.

CASE VIII. A.O. This 46 year old white male was not an alcoholic. He had had a splenectomy performed 4 years earlier for sideroblastic refractory anemia. Admission at this time was for progressive weight loss, left flank pain and diabetes. On physical examination a left upper quadrant mass was palpated. Amylase and lipase levels were normal. Barium enema examination (Fig. 8) revealed a large mass in the region of the splenic flexure, and slight downward displacement of the left kidney was noted on an intravenous pyelogram (Fig. 9). At surgery a pancreatic pseudocyst was found and resected.

CASE IX. L.B. This 59 year old white male alcoholic was admitted complaining of intermittent lower abdominal pain. On physical examination a left upper quadrant mass was palpable. A barium enema examination (Fig. 10, *A* and *B*) revealed a mass displacing the splenic flexure of the colon and this was found to represent a pseudocyst of the pancreas at surgery. External drainage was performed. The patient returned 21 months later, at which time repeat laparotomy revealed pancreatic carcinoma with multiple peritoneal metastases.

## DISCUSSION

A pseudocyst of the pancreas should be included in the provisional diagnosis of any alcoholic patient presenting with an abdominal mass, particularly if pain is the presenting symptom and if there has been a history of antecedent pancreatitis. The same possibility should be considered in a patient who has been exposed to blunt abdominal trauma and develops a mass in the upper abdomen. Laboratory examinations are not of distinctive value in the diagnosis as these may be entirely normal. However, elevated serum amylase levels, glycosuria, hyperglycemia, and anemia may be present in these patients.

Roentgen examination is the most helpful procedure in suggesting the diagnosis, whether or not an abdominal mass is palpated. Plain roentgenograms may reveal pancreatic calcification, although more often none will be present. Rarely, calcification of the entire cyst wall may define the mass. Intravenous urography may be of benefit in localizing a mass. The most common abnormality revealed is inferior displacement of the left kidney, but this is not a frequent finding. The presence of a mass involving the colon, particularly the

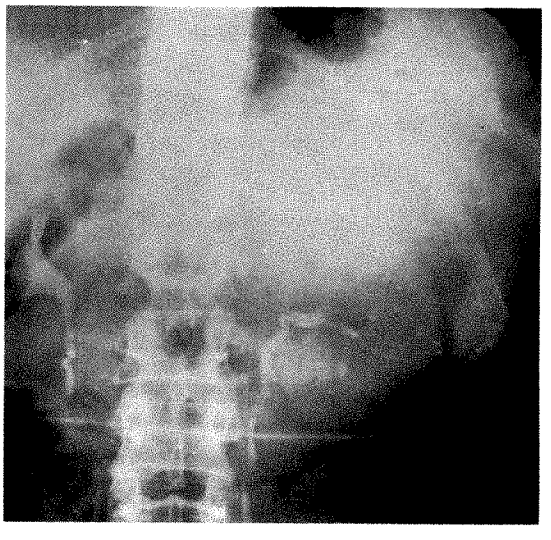


FIG. 9. Case VIII. Note slight downward displacement of left kidney on intravenous pyelogram.

splenic flexure, may be the only positive roentgen finding in a patient with a pancreatic pseudocyst. The appearance of an intramural lesion or an extrinsic mass invading the wall does not permit one to exclude pancreatic pseudocyst from the differential diagnosis. If there is associated inferior displacement of the left kidney and especially anterior displacement of the stomach, the diagnosis should be entertained even more strongly. Displacements of and impressions on the stomach are the most frequent manifestations of pancreatic pseudocysts. While anterior or lateral displacement of the stomach may be present alone or together, they are often found in association with localized impressions which may involve the greater or lesser curvature, fundus, body or antrum. Any portion of the duodenal loop may be displaced and in any direction.

Various authors have stressed that pancreatic pseudocysts produce a smooth contour of the displaced organ with stretching of the mucosal folds in contrast to neoplasms which may produce coarsened or destroyed mucosal folds.[4,8] While many pseudocysts present the roentgen signs of a purely extrinsic mass, the appearance of an intramural lesion or an invasive lesion by no means excludes this diagnosis. Many cases will suggest invasion of the wall of a hollow viscus and, indeed, invasion through the mucosa to the lumen may occur. Certainly, these roentgen changes must be expected when one considers that, although histologically benign, pseudocysts of the pancreas do invade surrounding structures by enzymatic action, pressure necrosis and intense inflammatory change.

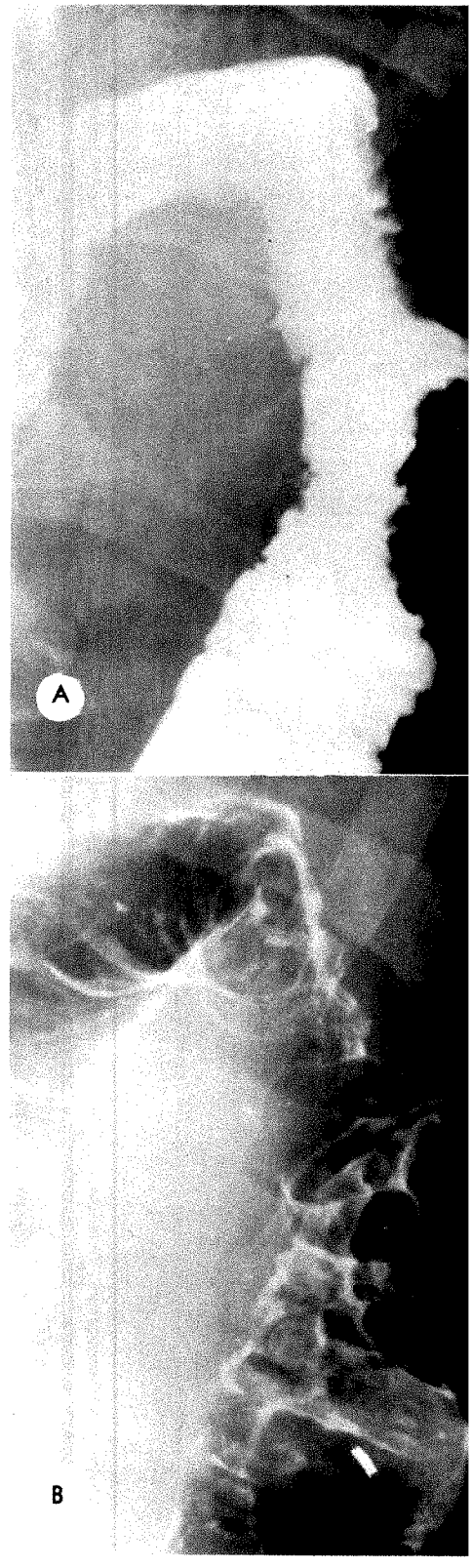



FIG. 10. Case IX. (*A* and *B*) Spot roentgenograms from barium enema and double contrast examination revealing extrinsic pressure with fixation on posterolateral aspect of splenic flexure.

SUMMARY

1. Twenty-one patients with pseudocysts of the pancreas have been reviewed.

2. Most patients will have a history of alcoholism or abdominal trauma.

3. The most common presenting findings are pain and abdominal mass.

4. The roentgen findings are reviewed, stressing the varied locations in which this lesion may present and the not infrequent appearance of an intramural or invasive mass.

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# ROENTGENOGRAPHIC FINDINGS IN LOCALIZED TRAUMA TO THE PANCREAS IN CHILDREN*

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MOST previous reports on traumatic pancreatitis have failed to mention or have not emphasized the roentgenographic findings which, though not pathognomonic, may be highly suggestive of this injury and thus should prompt other corroborative and more specific diagnostic studies. This has acted as the incentive for review of the roentgenographic studies in 5 children with localized pancreatitis secondary to blunt abdominal trauma seen within the last 4 years at the University of Rochester Medical Center. Four of these cases were previously reported[1] at which time the mechanism of injury, clinical features and surgical management were emphasized.

## REPORT OF CASES

CASE I. R.W. (SMH #58-23-41), a 7 year old boy, was riding his bicycle on a sidewalk when he ran into an object and was thrown against the handlebars, striking his abdomen. He quickly recovered from minimal discomfort and continued to ride the bicycle. Three hours later he complained of upper abdominal pain and subsequently vomited. He was brought to Strong Memorial Hospital 5 hours following injury, at which time he had generalized abdominal pain and was unable to assume a position of comfort. His vital signs were normal. There was moderate direct and rebound tenderness in the left upper quadrant of the abdomen but no spasm or guarding. Bowel sounds were hypoactive. The hematocrit was 40 per cent and the white blood cell count was 16,800 per cu. mm.

Plain abdominal roentgenograms showed an increase in left upper quadrant abdominal density with the duodenum dilated, hepatic flexure dilated and sharply "cut-off," and several loops of subjacent air-filled jejunum in the left upper quadrant (Fig. 1). The spacing between the air-filled stomach and the air-filled segments of jejunum was prominent. The findings, those of localized ileus, were highly compatible with traumatic pancreatitis. The serum amylase was 588 Somogyi units per 100 ml.

At laparotomy, 7 hours after injury, that portion of the pancreas overlying the vertebrae was found to be contused and lacerated but the major duct was intact. Fat necrosis was present in the peripancreatic omentum. The laceration was sutured and the area of injury drained. The postoperative course was uncomplicated and when seen in follow-up 14 months post surgery, the patient was completely asymptomatic.

CASE II. J.M. (SMH #58-70-73), a 5 year old boy, was playing on a "jungle gym" when he fell against one of the metal bars, his upper abdomen sustaining most of the blow. He continued to play, but 6 hours later began to com-



FIG. 1. Case I. In the supine anteroposterior roentgenogram the duodenal bulb is dilated (b), there are several loops of dilated jejunum (white arrows) and localized dilatation of the hepatic flexure (black arrows) overlapping the junction of the duodenal bulb with its second portion. (a) stomach.

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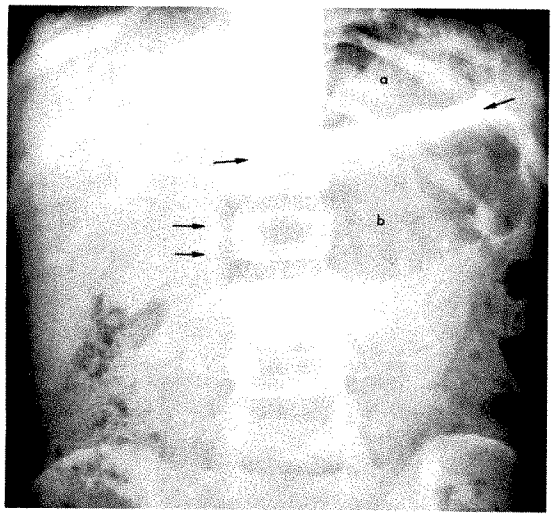



FIG. 2. Case II. In the supine anteroposterior roentgenogram there is localized dilatation of the left transverse colon (b) with prominent spacing and density in the area (arrows). (a) stomach.

plain of abdominal pain and vomited several times. The next day he was brought to the emergency department complaining of generalized abdominal pain. He was very restless but vital signs were normal. There was direct and rebound tenderness throughout the abdomen but no spasm. Bowel sounds were absent. The hematocrit was 40 per cent and the white blood cell count was 16,200 per cu. mm.

Plain abdominal roentgenograms showed a localized slightly dilated air-containing segment of left transverse colon with minimal widening of the space between the stomach and subjacent colon—the changes of localized ileus and peripancreatic infiltration (Fig. 2 and 3). The serum amylase was 253 Somogyi units per 100 ml.

A diagnosis of traumatic pancreatitis was made and he was taken to the operating room 30 hours after injury. At surgery, the pancreas was edematous and ecchymotic in the area overlying the vertebrae but the major duct was intact. Fat necrosis was present in the peripancreatic tissue and there was a small amount of serosanguineous fluid in the peritoneal cavity. Pancreatic drainage, effected with Penrose and rubber sump drains, was persistent for 12 days. Supportive postoperative management carried the patient through this period. When seen in follow-up 11 months after surgery he was asymptomatic.

*Comment.* In Cases I and II the plain roentgenograms of the abdomen were compatible with pancreatic injury and serum amylase determinations were obtained, corroborating this diagnosis. Early diagnosis and prompt surgical intervention resulted in a rapid recovery with little morbidity.

CASE III. S.S. (SMH #26-00-03), a 13 year old boy, was playing soccer when he fell and was struck in the abdomen by the heel of another player. Although he continued to play, 2 hours after injury he complained of severe upper abdominal pain and vomited. He was brought to the emergency department but because of minimal physical findings was sent home. Twenty-four hours later he returned complaining of persistent and intense generalized abdominal pain and more vomiting. Vital signs, hematocrit and white blood cell count were normal. Again, there were minimal abdominal physical findings. He was markedly agitated and restless to the extent that a diagnosis of conversion hysteria was considered.

Plain abdominal roentgenograms showed a localized collection of air in the slightly dilated left portion of the transverse colon (Fig. 4) and roentgenograms the next day showed increased space between the stomach and subjacent colon (Fig. 5), indicating localized ileus and progressive edema of peripancreatic tissues. Four quadrant paracentesis was negative. The serum amylase level at this time was 988 Somogyi units per 100 ml.

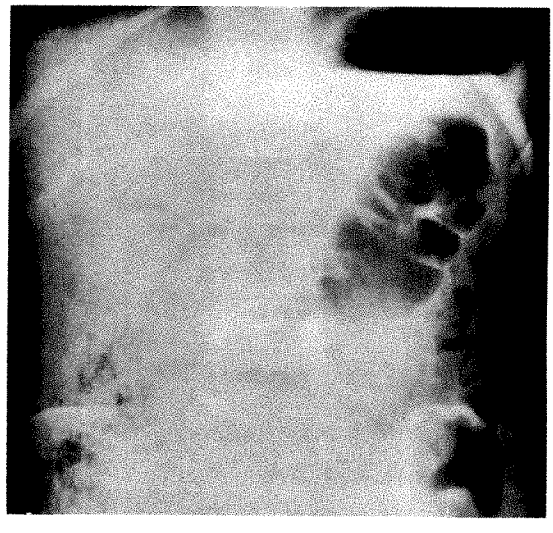


FIG. 3. Case II. In the upright anteroposterior roentgenogram the local dilatation of the left transverse colon (b) is well demonstrated. (a) stomach.

A diagnosis of traumatic pancreatitis was made and he was taken to the operating room for a laparotomy 56 hours after injury. A vertical tear was found in the body of the pancreas just to the left of the mesenteric vessels and in front of the vertebrae and fat necrosis was present. The laceration was sutured, the pancreatic area drained and supportive postoperative measures instituted. His course was prolonged. He developed a severe electrolyte problem because of the profuse drainage of pancreatic fluid and fistula formation. Sump drainage was instituted on the tenth postoperative day and pancreatic juice refed. His recovery was thereafter slow but progressive. The pancreatic fistula closed spontaneously on the sixty-seventh postoperative day. When seen in follow-up 20 months later he was completely recovered.

*Comment.* The pronounced subjective complaints and degree of restlessness in this case were considered to be out of proportion to the early physical findings. This impression caused delay in obtaining appropriate diagnostic studies and contributed to a prolonged and complicated course following surgery.

Case IV. G.D. (SMH #43-53-60), a 15 year old boy, was "roughhousing" with some other boys and was accidentally kicked in the abdomen. Six hours later he complained of upper abdominal pain, became nauseated and vomited. Because of persistence of symptoms he was brought to the emergency department 14 hours later. There was slight left upper abdominal tenderness but no spasm or guarding and normal bowel sounds.

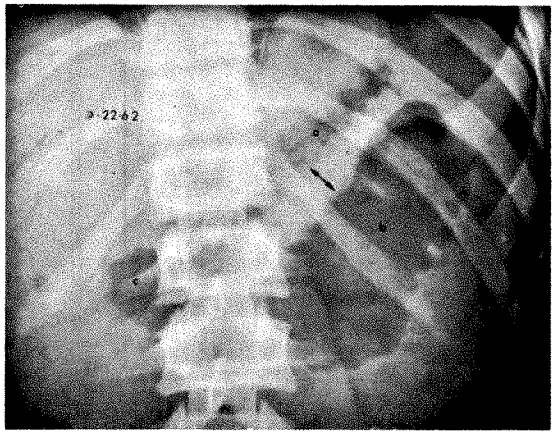


Fig. 5. Case III. In the supine anteroposterior roentgenogram of September 22, 1962 there is increased spacing (arrows) between the stomach (a) and subjacent colon (b). The duodenum (c) is dilated.

Plain abdominal roentgenograms showed a dilated loop of duodenum and cut-off of gas in the colon at the hepatic flexure (Fig. 6). The following day there was persistence of localized dilatation of the hepatic flexure and ascending colon with increased spacing between the transverse colon and stomach (Fig. 7). Radiopaque contrast study showed coarsened duodenal mucosa, a widened duodenal loop, tran-

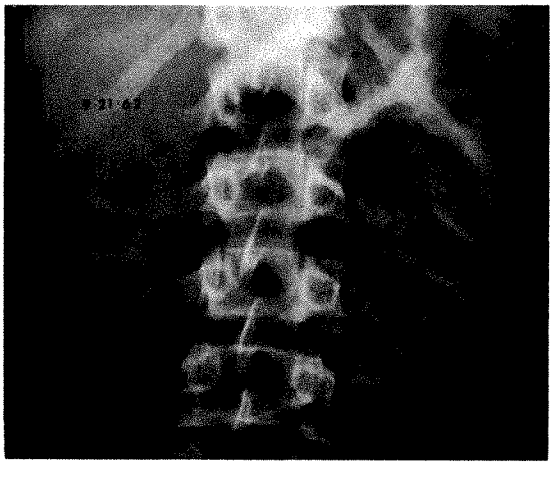


Fig. 4. Case III. In the supine anteroposterior roentgenogram of September 21, 1962 there is dilatation of the left portion of the transverse colon (b). (a) stomach, (d) dilated jejunal loops.



Fig. 6. Case IV. In the supine anteroposterior roentgenogram of October 3, 1963 there is a "colon cut-off" sign (arrows) with air in the dilated hepatic flexure and no transverse colonic air. (a) stomach, (b) hepatic flexure of colon.

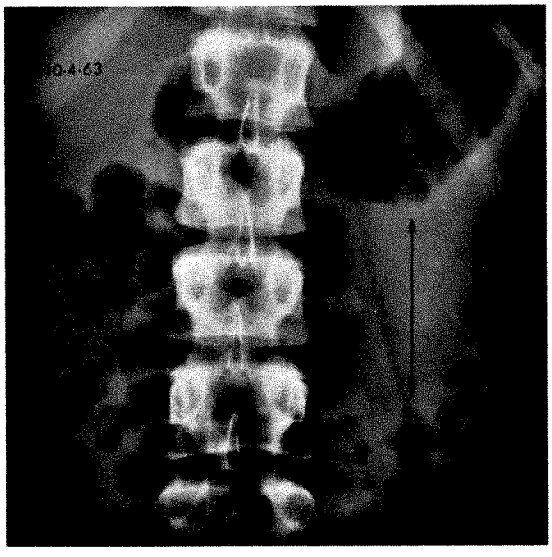



FIG. 7. Case IV. In the supine anteroposterior roentgenogram of October 4, 1963 the ascending colon and hepatic flexure (b) are dilated with disproportionate amount of gas in the transverse colon. There is a marked increase in the space between the stomach and the transverse colon (arrow). (a) stomach.

sient obstruction at the junction of the second and third portions of the duodenum, increased spacing between the stomach and transverse colon and a "reversed 3" sign (Fig. 8). Abdominal paracentesis was negative. The hematocrit was 42 per cent and white blood count 15,500 cells per cu. mm. The serum amylase was 1,200 Somogyi units per 100 ml.

A diagnosis of traumatic pancreatitis was made but because of his apparently good physical condition he was treated conservatively. Fifty hours following injury he was taken to surgery because of progressive worsening of his condition. At laparotomy a large amount of serosanguineous fluid was present in the peritoneal cavity. There was diffuse induration and edema of the entire pancreas with a large mass in the body of the gland. Extensive fat necrosis was present. The mass was incised and drained. His postoperative course was septic and stormy and drainage from the area contained large quantities of pancreatic fluid. Communication of his cutaneous drain with a major pancreatic duct was demonstrated by a fistulogram. He was discharged on the seventieth postoperative day with persistent but markedly decreased drainage. Two months later, the pancreatic drainage ceased but shortly thereafter, he developed recurrent abdominal pain and was readmitted. At that time a mass was palpable in the epigastrium and radiopaque contrast study demonstrated a retrogastric mass which displaced the body of the stomach anteriorly. This finding was consistent with a pseudocyst of the pancreas. The serum amylase was 350 Somogyi units per 100 ml. Re-exploration showed a pseudocyst involving the distal pancreas. A transduodenal pancreatogram was obtained and demonstrated obstruction of the main pancreatic duct at the junction of the body and tail of the pancreas. A distal pancreatectomy including the pseudocyst was performed. He did well postoperatively and when seen 8 months later was asymptomatic with a normal serum amylase and normal fasting blood sugar.

*Comment.* This case emphasizes the need for early surgery once the diagnosis of traumatic pancreatitis has been made. The extent of organ injury cannot be assessed on the basis of clinical signs alone since even apparently minor trauma may result in serious glandular damage.

CASE V. Y.G. (SMH #51-12-06), a 4 year old girl, complained of stomach pain and vomited several times. Later that day the pain subsided but she had a loose bowel movement and was

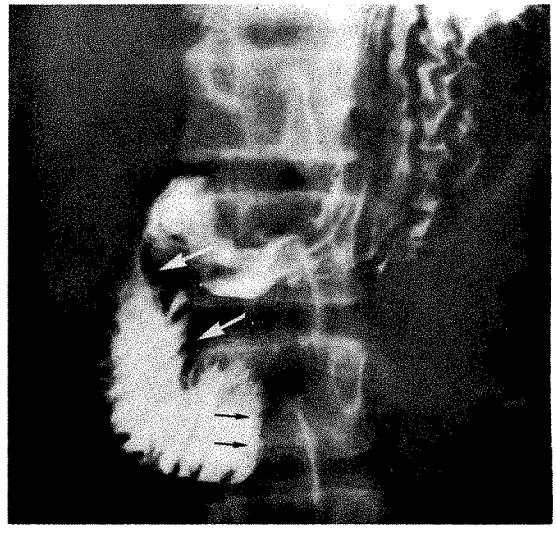


FIG. 8. Case IV. Supine anteroposterior roentgenogram from radiopaque contrast study demonstrating "reversed 3" sign (white arrows) and sharp cut-off at junction of second and third portions of the duodenum (black arrows).

slightly anorexic. Two days later she developed severe abdominal pain, vomited several times and was then brought to the hospital. After careful questioning of the family a history was obtained that the girl's brother had struck her in the abdomen with a broom handle about 20 hours prior to onset of symptoms.

When seen in the emergency department she appeared ill and was restless and irritable. Her temperature was 37.9°C. and her pulse was 120 per minute. There was generalized direct and rebound tenderness but no spasm. Bowel sounds were absent. The white blood cell count was 18,800 cells per cu. mm.

Plain abdominal roentgenograms showed increased soft tissue density especially over the upper abdomen and a dilated second portion of the duodenum with a sharp cut-off of air at its junction with the third portion (Fig. 9). The duodenal loop was widened (Fig. 10) and there were a few adjacent loops of gas-distended jejunum. The findings were indicative of paralytic ileus probably secondary to upper abdominal pathology.

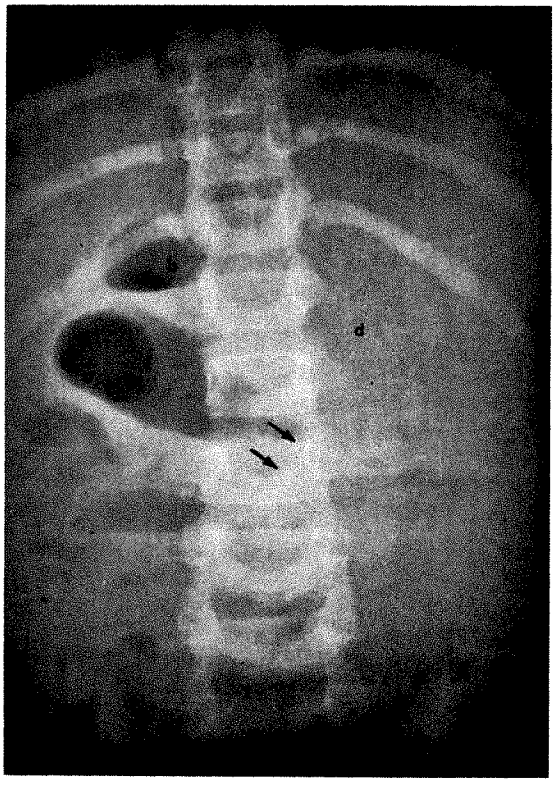



Fig. 9. Case v. Upright anteroposterior roentgenogram demonstrates dilated second portion of the duodenum (c) with a sharp cut-off of air at its junction with the third portion (arrows). (a) stomach, (b) duodenal bulb, (d) increased density in pancreatic area.



Fig. 10 Case v. Upright lateral roentgenogram demonstrates widened duodenal loop (arrows). (a) stomach, (b) duodenal bulb, (c) dilated second portion of duodenum.

A diagnosis of ruptured appendix was made and the child was taken to the operating room. Through a right lower abdominal incision (McBurney's) a large amount of turbid, blood-tinged fluid was found in the peritoneal cavity with areas of fat necrosis involving the omentum. The appendix was not ruptured. An upper mid abdominal incision was then made and a laceration found extending across the junction of the neck and body of the pancreas transecting the major duct. A polyethylene catheter was threaded across both ends of the severed pancreatic duct and brought into the duodenum following ampullary sphincterotomy. A T-tube was inserted into the common bile duct and the area of pancreatic injury and fluid-filled lesser sac were drained. An amylase determination on the pancreatic fluid was 1,600 Somogyi units per 100 ml.

The patient's early postoperative course was

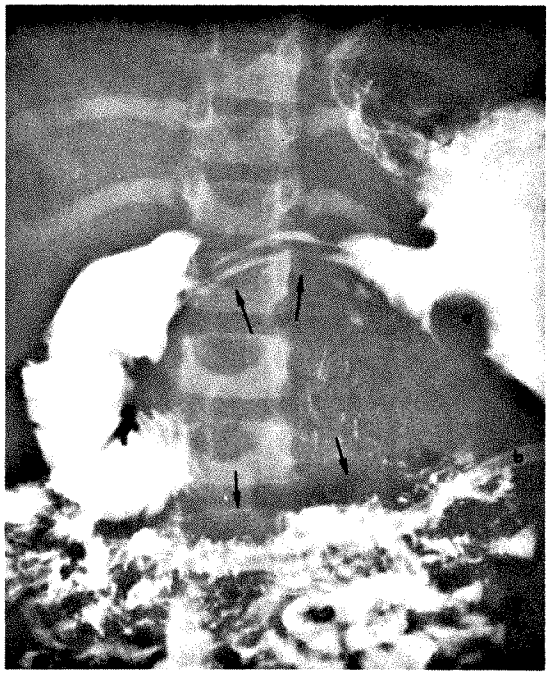


FIG. 11. Case v. Prone posteroanterior roentgenogram demonstrates duodenal "cut-off," widened duodenal loop, antral and jejunal displacement (arrows). (a) gastrostomy catheter bulb in stomach, (b) drainage tube.

stormy. Although her serum amylase was 128 Somogyi units per 100 ml. on the day after surgery, it rose subsequently to 740 Somogyi units per 100 ml. An upper gastrointestinal series showed widening of the duodenal loop and coarsening of the mucosa of the second and third portions of the duodenum (Fig. 11) reflecting peripancreatic edema. A T-tube cholangiogram was normal.

She slowly improved after the third postoperative day and her serum amylase level decreased. External pancreatic fluid drainage persisted for 22 days. A repeat gastrointestinal series showed improvement in duodenal mucosal appearance and return of the duodenal loop to normal size. She was discharged on the thirtieth postoperative day and when seen 6 weeks later her operative wounds were well healed, she was gaining weight and she had no symptoms or abnormal physical findings.

*Comment.* Despite roentgenographic findings indicative of upper abdominal pathology the surgeon did not consider the possibility of pancreatic injury and therefore no preoperative serum amylase determination was obtained. A correct diagnosis would have prevented the initial lower abdominal incision.

## DISCUSSION

### MECHANISM OF INJURY AND CLINICAL FEATURES

Injury to the pancreas occurs in less than 2 per cent of all nonpenetrating abdominal wounds.[3,9] To some extent, the central and deep position of the pancreas in the retroperitoneal space at the level of the first and second lumbar vertebrae affords it protection dorsally by the vertebral column and paraspinal muscles, and ventrally and laterally by the abdominal viscera. When pancreatic injury occurs from blunt trauma, the inflicting blow is usually severe and diffuse and as a result there is associated trauma to other abdominal organs. The rapid onset of symptoms and signs is invariably due to the associated organ injuries and prompts early surgical intervention.

Injury confined to the pancreas is less frequent than injury to multiple organs and occurs in fewer instances than isolated injury to the liver or spleen.[3] In localized trauma to the pancreas, the blow must be specifically directed to that organ area and delivered by an object of relatively small dimension at its striking point. When the pancreas, rich in blood supply but intrinsically weak in structure, sustains blunt trauma, it is forcibly pressed against the vertebrae[18] and by contrecoup sustains additional injury.[17]

Because the trauma in childhood often occurs while at play, a diagnosis of pancreatic injury is seldom initially entertained. In the absence of injury to other viscera, symptoms are typically slow to appear.[5] Moreover, the early physical signs may be mild and the child's subjective complaints not easy to evaluate, as exemplified by the 5 cases presented here. After a latent period of a few hours, the onset of symptoms begins with upper or generalized abdominal pain which usually becomes intense and may radiate to the back and shoulders. Nausea and vomiting follow and at times are severe. Rest-

lessness or agitation appears to be a valuable sign in children, indicative of serious injury.

Vital signs are usually normal. Both direct and rebound abdominal tenderness are variable in intensity and location and may not be present for some hours. Unless there has been massive bleeding the hematocrit is within normal limits while the white blood cell count is only moderately elevated.

### ROENTGENOGRAPHIC FINDINGS

An understanding of the anatomy of the pancreatic area is important in explaining the roentgenographic findings.[14,24] The anatomic bases for the production of roentgen pathology secondary to pancreatitis are: the close relationship of the head of the pancreas to the duodenal loop; the contact of the anterior surface of the right half of the pancreas to the transverse colon; the covering of the lower portion of the pancreas with peritoneum continuous with the inferior layer of the transverse mesocolon; the formation of a shelf with the colon upon which the stomach lies; and the adjacent position of the tail of the pancreas to the left colic flexure.

This is the first report in the American literature emphasizing the roentgenologic aspects of localized traumatic pancreatitis. Ekengren and Söderlund[12] reported in the French literature similar findings to those recorded here. As they indicated, the roentgenographic findings vary depending upon the size and site of pancreatic injury and resulting hemorrhage. The initial roentgenograms may not be unequivocally abnormal but follow-up studies may demonstrate significant progressive findings.

The roentgenographic findings in traumatic pancreatitis may be the same as those of acute pancreatitis[4,6,8,16,25] when the injury entails contusion or laceration without transection of the major duct. There may be additional changes in more severe injury such as complete glandular transection or laceration including severance of the major duct. The plain abdominal roentgenograms may show: (1) increased radiodensity in the left upper abdomen; (2) localized paralytic ileus (small or large bowel "sentinel loops"[15,26] or "colon cut-off" sign[21,22]); (3) increased spacing between the greater curvature of the stomach and subjacent jejunum or transverse colon, particularly emphasized by serial roentgenography; and (4) dilatation of the first and second portion of the duodenum shown by air distention. The occurrence of these signs in the cases presented here is recorded in Table I. Findings 1 and 2 are mainly due to peritoneal and transverse mesocolic irritation from pancreatic enzyme leakage, hemorrhage and overload of lymphatic drainage.[23] Findings 3 and 4 represent even more irritation, inflammatory edema and hemorrhage sometimes distending the lesser peritoneal sac. The fourth finding is especially seen when the head of the pancreas is the specific area of injury. The value of serial roentgenography in showing progressive change is demonstrated by Cases III and IV. Progressive pancreatic hemorrhage and bowel wall edema produce widening of the spaces between bowel loops and stomach as well as increased density. However, widespread retroperitoneal hemorrhage usually does not occur.[12] In only 1 of our cases (Case V) was there enough retroperitoneal hemorrhage, together with intraperitoneal hemorrhage, to obscure the left psoas muscle and renal shadows. There was in addition marked forward displacement of the stomach and duodenum. None of the cases reported here showed pleural reaction or fluid in the left hemithorax.

Radiopaque contrast study of the stomach and proximal small bowel enhances the preoperative diagnosis of this injury[23] and may show: (1) coarsened duodenal mucosa; (2) duodenal atony with transient obstruction at the third portion of the duodenum; (3) widening of the duodenal loop associated with elevation of the pyloric end of the stomach;[7] (4) stretching of the proximal jejunum; (5) the "reversed 3"

TABLE I

FREQUENCY OF SIGNIFICANT ROENTGENOGRAPHIC FINDINGS IN TRAUMATIC PANCREATITIS IN PLAIN ABDOMINAL ROENTGENOGRAMS

| Case | 1. Increased density | 2. Localized ileus | 3. Increased spacing between adjacent organs | 4. Reversed 3 sign | 5. Dilatation of duodenum | 6. Development of pseudocyst |
|---|---|---|---|---|---|---|
| I | x | x | x | | x | |
| II | x | x | x | | | |
| III | x | x | x | | | x |
| IV | x | x | | x | x | x |
| V | x | x | | | x | x |

sign of Frostberg;[18] and (6) mass impingement upon stomach and bowel. (A good cross-table lateral roentgenogram is imperative in assessing forward displacement of the stomach and duodenum.) The occurrence of these findings in the cases presented here is recorded in Table II. As stated previously, the findings are the result of inflammatory irritation from leaking pancreatic enzymes, lymphatic stasis and hemorrhage. The "reversed 3" sign in particular reflects swelling of the head of the pancreas in the presence of a fixed ampulla of Vater. Hematoma formation in the lesser sac and around the transverse mesocolon may cause mass impingement deformities not unlike those produced by pancreatic pseudocyst formation.

Because one of the differential diagnostic possibilities considered from the clinical picture may be ruptured viscus, the use of a water-soluble contrast medium is preferable to barium. However, in younger children and infants a water-soluble medium should be used sparingly and cautiously because of its hyperosmolarity.[20] Dilution to isotonic levels should be effected before instillation or injection.

The roentgenographic findings in traumatic pancreatitis cannot be considered pathognomonic. However, in the presence of minimal but progressive clinical findings they may provide highly suggestive evidence prompting a serum amylase determination. Elevation of the serum amylase following abdominal trauma is considered diagnostic of pancreatic injury.[1,19]

### SURGICAL MANAGEMENT AND COMPLICATIONS

Once the diagnosis of traumatic pancreatitis has been made, laparotomy is indicated. Supportive therapy is important and must be carried out both preoperatively and postoperatively. The operative procedure will depend upon the findings at exploration.[10] In most cases the pancreas will be contused or lacerated without involvement of the major duct. In these, debridement, suture of laceration with nonabsorbable material, and adequate drainage of the area of injury through the lesser peritoneal sac are sufficient. Transection of the major pancreatic duct may be treated by distal pancreatectomy, Roux-en-Y pancreatojejunostomy, or sphincterotomy with catheter cannulation of the main pancreatic duct (Case v).

The complications of pancreatic trauma are frequent and include pancreatic pseudocyst, external pancreatic fistula and pancreatic abscess.[18] Post-traumatic pancreatic pseudocyst arises either as a direct continuation of initial hemorrhage and hematoma as in Case v or by gradual

delayed development, as in Case IV (Table II). In both cases radiopaque contrast study of the stomach and proximal small bowel showed a persistence of, or an increase in size of, a mass with associated edema and adjacent organ displacement including widening of the duodenal loop. Transduodenal pancreatography can be of particular help in the presence of pseudocyst formation, as in Case IV, by demonstrating the condition of the ductal system at surgery. This radiopaque method of ductal visualization has been described by Doubilet *et al.*[11]

External fistulas occur to some degree in all patients following surgical drainage of the pancreas although most close spontaneously within a few weeks.[2] Persistence of the fistula invariably indicates interruption of the major pancreatic duct, often with obstruction of the proximal duct system, as in Case IV. In this patient, radiopaque contrast injection (50 per cent Hypaque) of the fistulous tract was of help in directing appropriate treatment. Pancreatic abscess is relatively infrequent and none of our cases developed this complication.

With early recognition of this injury accompanied by surgical intervention, the mortality from isolated blunt trauma to the pancreas should be no more than 5 per cent.[17]

TABLE II

FREQUENCY OF SIGNIFICANT ROENTGENOGRAPHIC FINDINGS IN TRAUMATIC PANCREATITIS WITH USE OF RADIOPAQUE CONTRAST MATERIAL*

| Case | Coarsened duodenal mucosa | Transient obstruction of third portion of duodenum | Widening of duodenal loop |
|---|---|---|---|
| IV | x | x | x |
| V | x | x | x |

* Cases I, II, and III had no radiopaque contrast studies.

SUMMARY

The roentgenographic features of localized trauma to the pancreas in children are presented from a review of 5 cases. Plain abdominal findings are those of localized ileus in the upper quadrants as shown by an air-distended loop of small or large bowel in combination with inflammation of peripancreatic tissues, and indicated by increased density with increased spacing between adjacent air-containing viscera.

The radiopaque contrast findings are coarsened mucosal markings, duodenal atony with transient mid-duodenal obstruction, widening of the duodenal loop, stretching of the proximal jejunum, the "reversed 3" sign and mass displacement of viscera by hematoma formation. Roentgenology can be further helpful in assessing progress and demonstrating complications.

The innocent source and apparent trivial force of trauma, usually sustained at play, is noted. Delayed onset of symptoms, abdominal pain out of proportion to the early physical findings, vomiting and restlessness are the significant clinical features.

Suspicion of pancreatic trauma from the symptoms and signs in association with highly suggestive roentgenographic findings, should prompt a serum amylase determination which, when elevated, is diagnostic of injury to this organ.

Early surgical intervention with adequate drainage of the area of injury is the mainstay of treatment.

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# THE ARTERIOGRAPHIC APPEARANCE OF CARCINOMA OF THE UNCINATE PROCESS OF THE PANCREAS*

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SINCE there is no current practical means by which the parenchyma of the pancreas can be opacified, the roentgenographic diagnosis of carcinoma of the pancreas depends on the demonstration of changes produced by the tumor in adjacent organs or in the vascular pattern around or within the gland. Tumor "stains" on angiography of pancreatic carcinoma are rare. Lesions of the head of the pancreas lie in close relationship to the duodenal sweep and often produce changes which can be detected on the upper gastrointestinal series. Carcinoma of the body or tail of the pancreas may indent or infiltrate the posterior wall of the fundus of the stomach or the duodenum in the region of the ligament of Treitz. Carcinoma of the uncinate process of the pancreas can involve the transverse portion of the duodenum but this is often difficult to identify on barium studies. A similar difficulty is encountered in the arteriographic diagnosis of lesions of the uncinate process, as they do not involve the larger branches of the celiac artery until they are very extensive. We have recently studied 2 patients with carcinoma of the uncinate process in whom the pattern of vascular involvement and displacement was similar and clearly indicated both the location and the extent of the tumor.

## ANATOMY

The pancreas is an elongated retroperitoneal organ which is grossly divided into three main segments that are not clearly demarcated from each other. The head of the pancreas is the largest of these portions and is cradled within the duodenal sweep. As it crosses the vertebral column, the gland becomes narrow in its superior-inferior dimension marking the transition to the body of the pancreas. The junctional region between the head and the body is termed the neck of the pancreas. The body extends to the left and slightly upward behind the stomach. The tail is the pointed, left-sided end of the pancreas and reaches to the hilus of the spleen. The uncinate process is a hook-like posterior extension of the head of the pancreas lying in front of the vertebral bodies along the upper border of the third portion of the duodenum and tucked in between the aorta posteriorly and the superior mesenteric vessels anteriorly (Fig. 1).

The blood supply to the uncinate process is a part of the vascular complex that also supplies the head and neck and is derived from both the celiac and superior mesenteric arteries. The superior mesenteric artery arises from the aorta behind the neck of the pancreas and courses downward with the superior mesenteric vein in a groove within the pancreas. This groove is formed by the uncinate process behind the vessels and the lower portion of the neck of the pancreas in front and the union of these two parts of the pancreas to the right. The superior mesenteric vessels emerge from the undersurface of the pancreas between the two leaves of the mesentery of the small bowel. The first branch arising from the superior mesenteric artery is usually the inferior pancreaticoduodenal artery which passes to the right behind the superior mesenteric vein and then divides into two branches which form arcades with branches of the gastroduodenal artery of the celiac axis. One branch passes anterior to the head of the pancreas to join the

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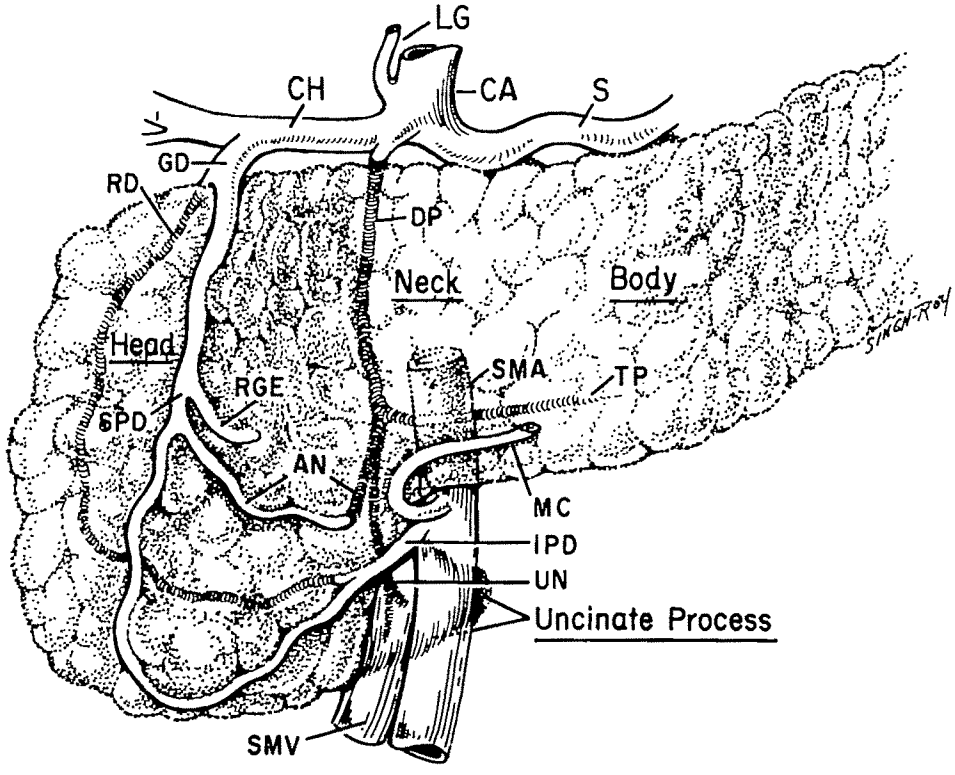



FIG. 1. Diagram of the arterial supply of the head of the pancreas and the uncinate process as seen from in front. The shaded vessels lie within the pancreas or posterior to it. (CA)—celiac artery, (S)—splenic artery, (CH)—common hepatic artery, (GD)—gastroduodenal artery, (RD)—retroduodenal artery, (SPD)—superior pancreaticoduodenal artery, (RGE)—right gastroepiploic artery, (SMA)—superior mesenteric artery, (SMV)—superior mesenteric vein, (MC)—middle colic artery, (IPD)—inferior pancreaticoduodenal artery, (DP)—dorsal pancreatic artery, (TP)—transverse pancreatic artery, (AN)—anastomotic branch between dorsal pancreatic and superior pancreaticoduodenal arteries, (UN)—uncinate branch of the dorsal pancreatic artery, (LG)—left gastric artery.

superior pancreaticoduodenal artery, while the second passes behind the neck of the pancreas and anastomoses with branches of the retroduodenal (posterior pancreaticoduodenal)[1] artery. The middle colic artery most commonly arises from the superior mesenteric artery slightly above or below the inferior pancreaticoduodenal branch and lies within the leaves of the transverse mesocolon as it courses towards the splenic flexure of the large bowel. This vessel also provides a variable number of branches to the right side of the colon.

The other major vessel supplying the region of the uncinate process is the dorsal pancreatic artery. The origin of this vessel is quite variable. Most commonly, it arises from the first part of the splenic artery but it may take origin from the proximal portion of the hepatic, the celiac or the superior mesenteric arteries or directly from the aorta.[5] The dorsal pancreatic artery courses almost directly downward, in its proximal portion, behind the neck of the pancreas and then continues in the groove between the neck and the uncinate process, to the right of the superior mesenteric artery. Thus, the distal portion of the dorsal pancreatic artery lies anterior to the uncinate process. Typically, this vessel gives rise to three main branches: the transverse pancreatic artery which courses horizontally to the left along the posterior surface of the pancreas; a second branch which proceeds to the inferior surface of the neck of the pan-

creas and turns anteriorly around the neck to anastomose with the superior pancreaticoduodenal artery; and a third branch which is distributed posteriorly to the uncinate process and, in some cases, joins the inferior pancreaticoduodenal artery.

The anterior surface of the pancreas is covered by peritoneum except where it is reflected forward in a double layer to form the transverse mesocolon. The line of origin of this portion of the mesocolon runs more or less horizontally across the length of the pancreas. The line of origin of the mesentery of the small bowel begins over the terminal portion of the duodenum and extends downward and to the right along the posterior abdominal wall. The pancreas is in direct continuity with the areolar retroperitoneal tissue that forms the core of the mesentery and the mesocolon providing an easy route of spread for carcinoma involving the uncinate process.

## ROENTGEN FEATURES

In the frontal projection of a selective celiac arteriogram, the normal dorsal pancreatic artery is seen as a small vessel coursing downward near the midline and to the right of the superior mesenteric artery[3,7] (Fig. 2*A*). Because of its central location, the mouth of the dorsal pancreatic artery is occasionally entered fortuitously by a catheter that has been advanced into the celiac artery (Fig. 3). The dorsal pancreatic artery divides into a variable number of branches in the region of the uncinate process and it may not be possible to accurately label each of these vessels. The transverse pancreatic artery, however, can often be identified on a technically satisfactory study.[3] In the right posterior oblique projection, the downward course of the dorsal pancreatic artery parallels the anterior line of the vertebral bodies (Fig. 2*B*). There may be local irregularities in the course of the dorsal pancreatic artery

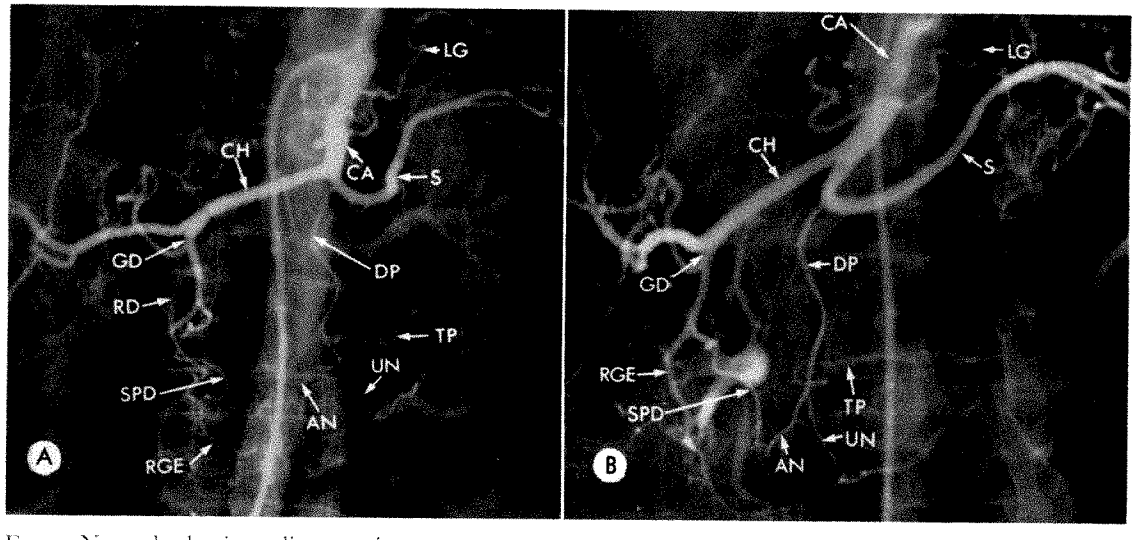



Fig. 2. Normal selective celiac arteriograms.

(*A*) Frontal projection. The catheter tip is in the celiac artery (CA). The dorsal pancreatic artery (DP) arises from the proximal portion of the splenic artery (S) and courses directly downward. Three main branches of this vessel can be seen: (TP)—transverse pancreatic artery, (AN) anastomotic branch joining the superior pancreaticoduodenal artery (SPD), and (UN)—the branch to the uncinate process. The retroduodenal branch (RD) of the gastroduodenal artery (GD) is faintly visualized. (RGE)—right gastroepiploic artery, (CH)—common hepatic artery, (LG)—left gastric artery.

(*B*) Right posterior oblique projection. The dorsal pancreatic artery (DP) courses downward, parallel to the anterior line of the vertebral bodies. The transverse pancreatic artery (TP) is foreshortened in this view. The uncinate branch (UN) of the dorsal pancreatic artery is directed downward and slightly posteriorly. Contrast material is present in the renal pelves and calyces.

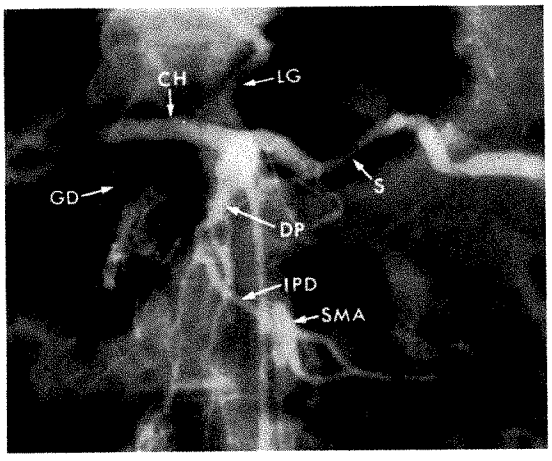



FIG. 3. Selective celiac arteriogram, frontal projection. Carcinoma of the body of the pancreas encasing the proximal portion of the splenic artery (S). The catheter tip has entered the dorsal pancreatic artery (DP) which appears normal. There is a prominent anastomosis between a branch of the dorsal pancreatic artery and the inferior pancreaticoduodenal artery (IPD) and contrast material flows in retrograde fashion into the superior mesenteric artery (SMA). (CH)—common hepatic artery, (LG)—left gastric artery, (GD)—gastroduodenal artery.

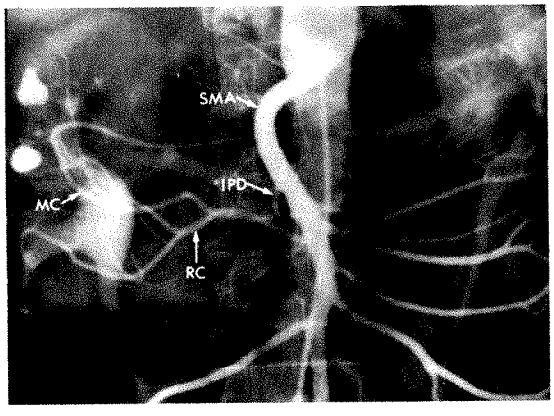



FIG. 4. Normal selective mesenteric arteriogram. Frontal projection. The inferior pancreaticoduodenal artery (IPD) is the first branch arising from the superior mesenteric artery (SMA). Only the proximal portion of this branch is visualized as it courses downward and becomes obscured by the superior mesenteric artery. The middle colic artery (MC) arises as a large branch from the right side of the superior mesenteric artery and, in this patient, supplies a sizeable branch to the right side of the colon (RC) before it reverses its direction and courses in the transverse mesocolon toward the splenic flexure of the colon.

but these are short and sharply angled. An arcuate course or bowing of the dorsal pancreatic artery or its branches is abnormal.

The superior mesenteric artery is a large vessel arising from the anterior surface of the aorta slightly below the level of the celiac artery. The middle colic artery arises most commonly from the right side of the proximal portion of the superior mesenteric artery, courses for a short distance to the right and then reverses its direction and continues to the splenic flexure of the colon[3] (Fig. 4).

The angiographic changes produced by carcinoma of the pancreas[3,4] are those of encasement of the vessels by tumor producing irregular areas of narrowing, displacement of vessels by a mass, amputation of vessels, abnormal tumor vessels and, least commonly, dilatation of supplying vessels and early filling of the veins draining the lesion. Lesions of the head of the pancreas will involve mainly the gastroduodenal artery and its branches (Fig. 5), lesions of the body and tail involve the splenic artery

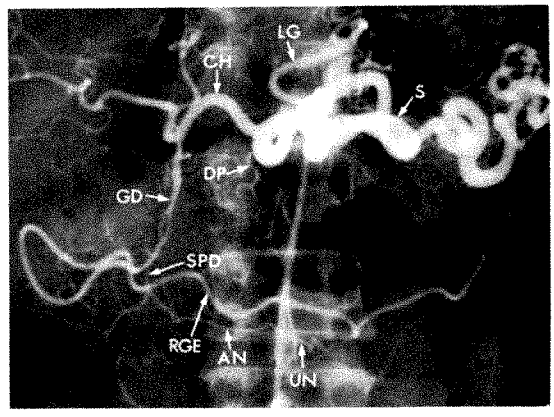



FIG. 5. Selective celiac arteriogram, frontal projection. Carcinoma of the head of the pancreas encasing the gastroduodenal artery (GD). (CH)—common hepatic artery, (S)—splenic artery, (RGE)—right gastroepiploic artery, (LG)—left gastric artery. The dorsal pancreatic artery (DP) is not displaced. (AN)—anastomotic branch of the dorsal pancreatic artery joining the superior pancreaticoduodenal (SPD) artery, (UN)—branch to uncinate process.

(Fig. 3), while those of the uncinate process will first affect the dorsal pancreatic artery.

A carcinoma arising from the uncinate process of the pancreas tends to displace the dorsal pancreatic artery forward and to the right. In the frontal projection of the celiac arteriogram, the dorsal pancreatic artery and its branches, if not occluded, will be deviated towards the right and bowed by the mass (Fig. 6*A*). The anterior displacement of the dorsal pancreatic artery is best appreciated in the right posterior oblique view (Fig. 6*B*). Encasement of this vessel can be identified at times (Fig. 6*B*) but minor variations in the caliber may be difficult to detect because of the small size of the vessel.

As the tumor extends beyond the confines of the pancreas, it will invade the root of the mesentery and may involve the superior mesenteric artery or its branches. The superior mesenteric artery, because of its size and the thickness of its wall shows evidence of encasement later than its branches. The middle colic artery is likely to be the most markedly involved branch (Fig. 6*C*; and 7).

## DISCUSSION

Lesions of the uncinate process of the pancreas are difficult to detect on barium studies because changes in the third portion of the duodenum are often subtle and difficult to visualize clearly. The transverse

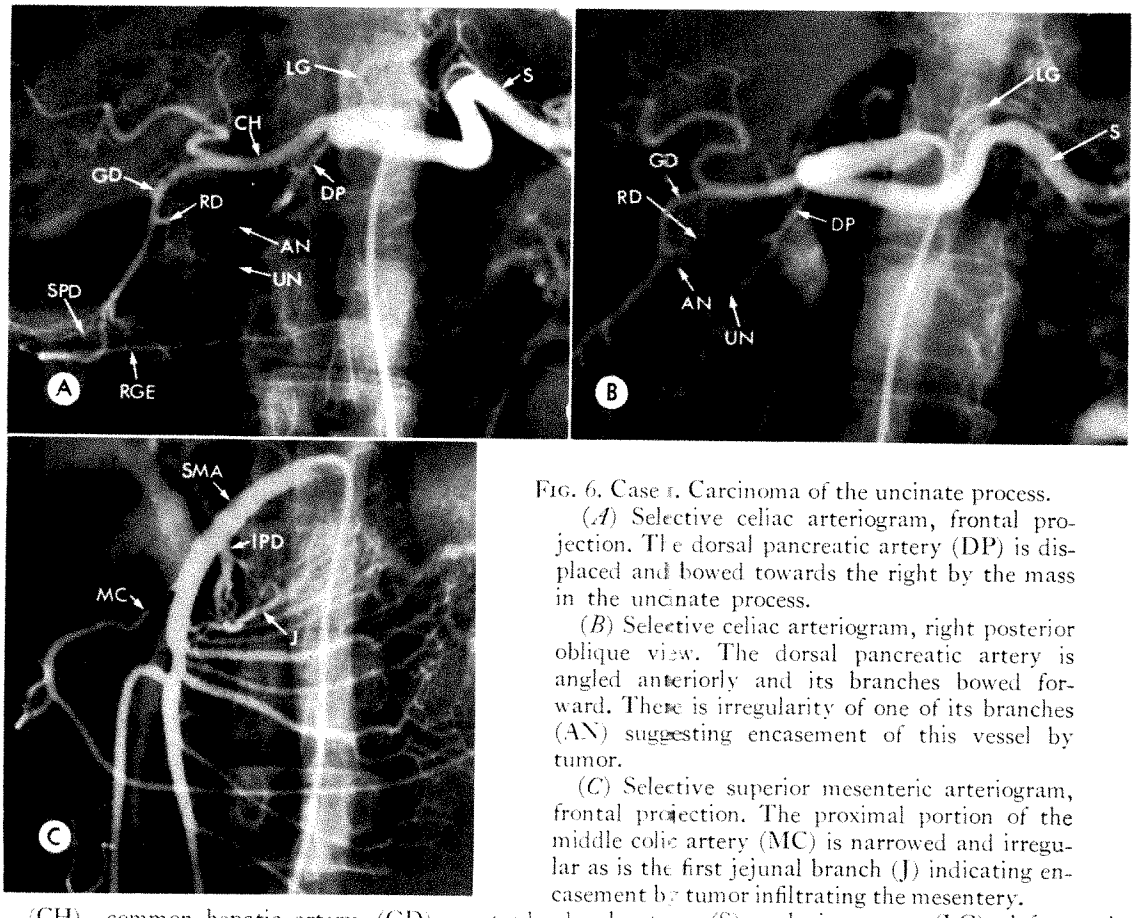



Fig. 6. Case 1. Carcinoma of the uncinate process.

(*A*) Selective celiac arteriogram, frontal projection. The dorsal pancreatic artery (DP) is displaced and bowed towards the right by the mass in the uncinate process.

(*B*) Selective celiac arteriogram, right posterior oblique view. The dorsal pancreatic artery is angled anteriorly and its branches bowed forward. There is irregularity of one of its branches (AN) suggesting encasement of this vessel by tumor.

(*C*) Selective superior mesenteric arteriogram, frontal projection. The proximal portion of the middle colic artery (MC) is narrowed and irregular as is the first jejunal branch (J) indicating encasement by tumor infiltrating the mesentery.

(CH)—common hepatic artery, (GD)—gastroduodenal artery, (S)—splenic artery, (LG)—left gastric artery, (RGE)—right gastroepiploic artery, (SPD)—superior pancreaticoduodenal artery, (RD)—retroduodenal artery, (SMA)—superior mesenteric artery, (IPD)—inferior pancreaticoduodenal artery.

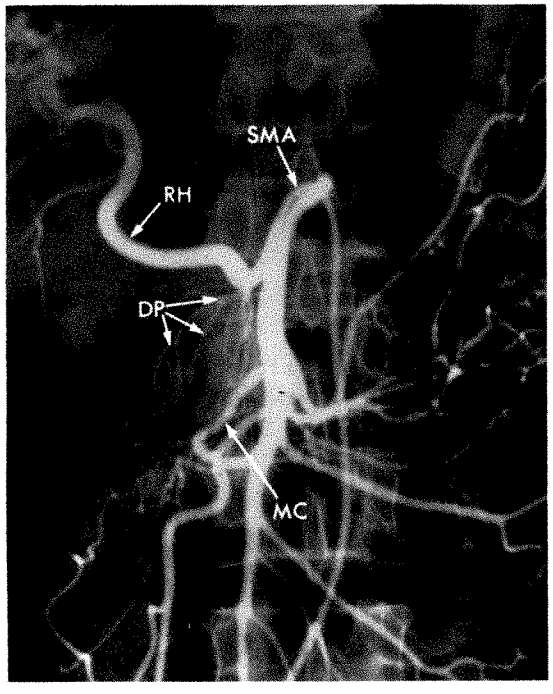



FIG. 7. Case II. Carcinoma of the uncinate process of the pancreas. Selective superior mesenteric arteriogram, frontal projection. The right hepatic artery (RH) arises anomalously from the superior mesenteric artery (SMA). The dorsal pancreatic artery (DP) takes origin from the proximal portion of the right hepatic artery and is displaced and bowed to the right by the mass in the uncinate process. The proximal portion of the middle colic artery (MC) is irregularly narrowed indicating encasement by tumor.

portion of the duodenum is often obscured by the barium filled stomach and indented by the superior mesenteric vessels. In Case I, a barium meal examination performed 1 month prior to the arteriogram demonstrated the entire duodenum which showed no abnormality. At the time of surgery, soon after the arteriogram, the carcinoma had extended into the root of the mesentery and the transverse duodenum was fixed by the mass. In Case II, the transverse portion of the duodenum was never completely visualized on the upper gastrointestinal study. There was a suggestion of fixation of the folds of the descending duodenum. At surgery, the mass was found to be quite large, extending from the uncinate process into the head of the pancreas and the root of the mesentery.

In both cases, displacement of the dorsal pancreatic artery anteriorly and to the right indicated the presence of a mass lesion in the uncinate process. Displacement of vessels alone, however, does not indicate the nature of the mass which could be an inflammatory cyst or benign neoplasm.[8] Encasement of the vessel or other arteriographic signs mentioned previously must be present before a diagnosis of carcinoma can be made. The other branches of the celiac axis were normal. Lesions involving the head of the pancreas (Fig. 5) and the body (Fig. 3) will not affect the dorsal pancreatic artery unless they are quite large. Encasement of the superior mesenteric artery or its branches indicates infiltration of the mesentery so that a curative resection of the lesion is not possible.*

## SUMMARY

1. The anatomy and vascular supply of the uncinate process of the pancreas are reviewed.

2. Carcinoma of the uncinate process tends to involve the dorsal pancreatic artery and its branches in a characteristic fashion. This abnormal pattern can be detected by selective celiac arteriography.

3. Selective superior mesenteric arteriography should also be performed in these cases. Involvement of this vessel or its main branches indicates that the lesion is not surgically curable.

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* Two additional cases of carcinoma of the pancreas involving the uncinate process have been studied. In both, the dorsal pancreatic artery was bowed forward, to the right in one, and to the left in the other. If the mass arises near the root of the uncinate process, it may be to the right of the dorsal pancreatic artery and therefore displace this vessel to the left. The root of the superior mesenteric artery showed evidence of encasement in both cases. Both cases were inoperable.

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# ROLE OF SPLEEN SCANNING IN PLANNING THE TREATMENT AND ASSESSMENT OF SPLENIC RESPONSE IN BLOOD DYSCRASIAS*

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THE property of the spleen to remove partially damaged red blood cells from the blood stream has been utilized by a number of investigators to obtain spleen scans. Johnson *et al.*[6] employed sensitized red blood cells for scanning the spleen. Harris *et al.*[5] later demonstrated sequestration of heat-treated autologous red blood cells in the spleen. Wagner and his colleagues[11] obtained spleen scans of some of their patients by using N-ethyl maleimide (NEM) to damage the red blood cells. However, they found that of all the techniques, the heat treatment method was most satisfactory. More recently, an easier and less cumbersome technique for obtaining spleen scans has been suggested using 1-bromo mercury 2-hydroxypropane (BMHP) labeled with radioactive mercury.[12]

A number of workers have demonstrated the usefulness of spleen scans in diagnosing a variety of diseases.[2,3,4,8,11] They found spleen scans to be helpful in the detection and quantitation of splenomegaly, in the differential diagnosis of obscure abdominal masses, and in the delineation of space-occupying lesions of the spleen. We have also found scanning of the spleen to be of special assistance in locating the best site for external irradiation of the spleen and in assessing the response of the spleen to therapy in some of our cases of chronic myeloid leukemia, Hodgkin's disease and polycythemia vera.

## MATERIAL AND METHODS

Spleen scanning was performed on a series of 28 patients. The series included 7 cases of polycythemia vera, 1 case of secondary polycythemia, 8 cases of acute and chronic myeloid and lymphatic leukemia, and 1 case of Hodgkin's disease. The remaining 11 patients had a variety of disease conditions, *i.e.* hemolytic anemia, obscure abdominal masses, generalized lymphadenopathy, hydatid cyst of the liver and bone metastasis from an unknown primary lesion.

### TECHNIQUE OF LABELING AND HEAT TREATMENT OF RED BLOOD CELLS

A sterile syringe was used to withdraw 15–20 ml. of the patient's blood. The blood was transferred into a screw cap vial containing 4 ml. of modified ACD solution to which 200–300 $\mu$c of $Cr^{51}$ in the form of high specific activity sodium chromate (110 $\mu$c/$\mu$g.) was added. The mixture was incubated at 37° C. for 20 minutes. About 100 mg. of ascorbic acid was then added to stop further labeling of red blood cells. The labeled blood was again incubated at 49°±1° C. in a shaking water bath for 60 minutes.

After a brief cooling the blood was reinjected into the patient.

### TECHNIQUE OF SPLEEN SCANNING

Spleen scanning was performed 2 to 4 hours after the injection of heat-treated $Cr^{51}$ labeled red blood cells. An isotope scanner having a 3 inch×3 inch thallium activated sodium iodide crystal fitted with a 19 hole honeycomb collimator was used. All the patients were scanned in the prone position. The scattered radiation from surrounding tissues was selectively eliminated by using a $\gamma$-ray spectrometer ad-

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justed around the photopeak of $Cr^{51}$ with a window width of 10 volts. The scanning speed employed was 24 cm. per minute. The dot factor for dot scans and the percentage cut-off for photoscans were adjusted according to the count rates over the spleen of individual patients. In a number of patients, ordinary photographic printing paper was used instead of the conventional single emulsion photoscanning film. The use of readily available and inexpensive photographic printing paper for photoscanning has been described previously.[9]

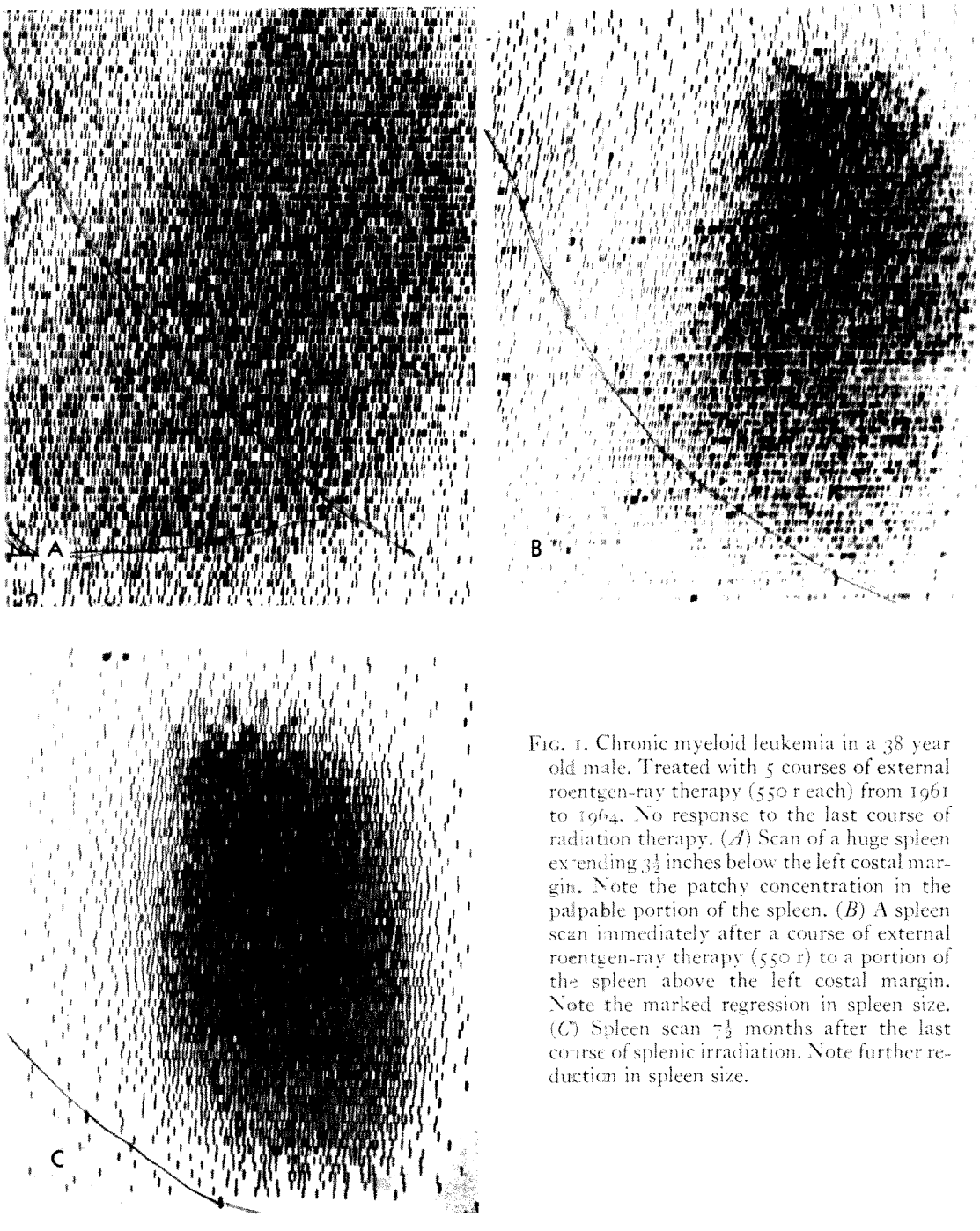



FIG. 1. Chronic myeloid leukemia in a 38 year old male. Treated with 5 courses of external roentgen-ray therapy (550 r each) from 1961 to 1964. No response to the last course of radiation therapy. (*A*) Scan of a huge spleen extending 3½ inches below the left costal margin. Note the patchy concentration in the palpable portion of the spleen. (*B*) A spleen scan immediately after a course of external roentgen-ray therapy (550 r) to a portion of the spleen above the left costal margin. Note the marked regression in spleen size. (*C*) Spleen scan 7½ months after the last course of splenic irradiation. Note further reduction in spleen size.

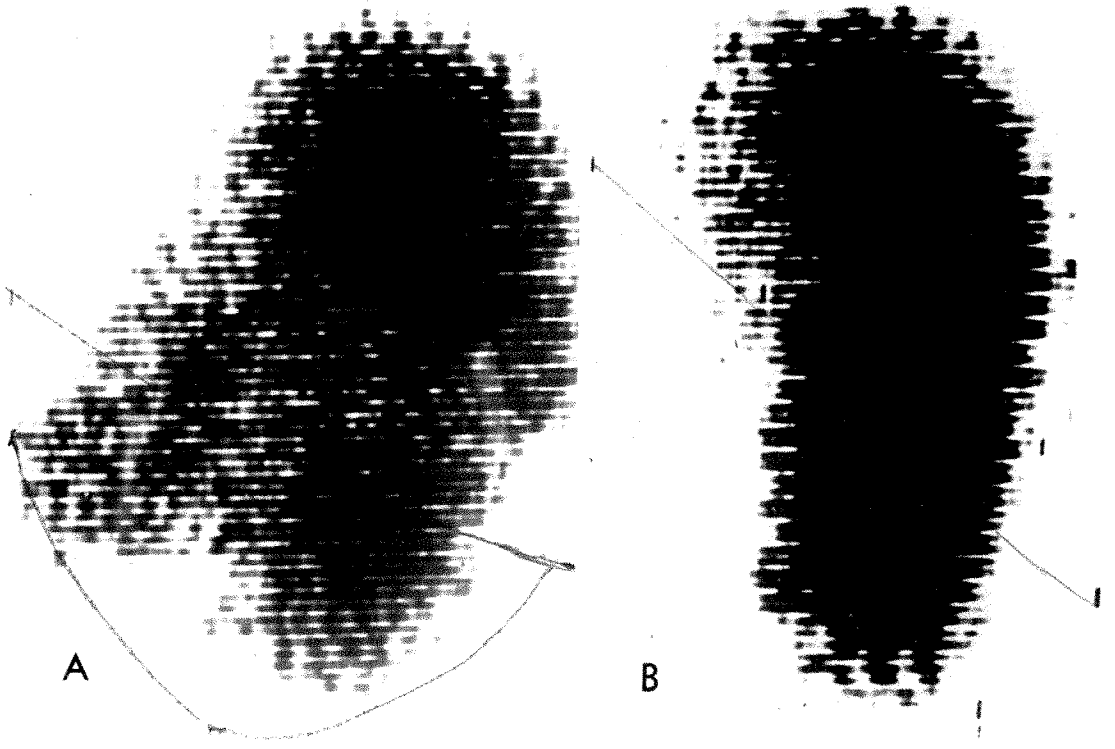



FIG. 2. Hodgkin's disease in a 52 year old male. Previously treated with external roentgen-ray therapy to neck and axillary lymph nodes. (*A*) Scan of an enlarged spleen extending 2 inches below the left costal margin. A well defined comparatively "cold" mass is seen in the region of the hilus of the spleen. (*B*) Repeat scan immediately after a 12 day course of intravenous endoxan. A considerable reduction in the bulk of the spleen is visible. The comparatively "cold" mass in the region of the hilus has completely disappeared.

## RESULTS

The authors have found spleen scanning of considerable aid in the follow-up of patients with various hematologic disorders and in the evaluation of their response to therapy. Four case histories are presented.

CASE I. A 38 year old male was first seen at this hospital 4 years ago with an enlarged spleen, loss of weight, loss of appetite and anemia. No lymphadenopathy was detected. Bone marrow and peripheral blood smear examinations revealed chronic myeloid leukemia. From 1961 until December, 1964 the disease was kept under control by repeated small doses of roentgen irradiation of the spleen (5 courses of 550 r each). The last course of splenic irradiation did not produce a satisfactory remission and it was thought that the disease was becoming resistant to roentgen rays. The scan showed a huge spleen extending $3\frac{1}{2}$ inches below the left costal margin (Fig. 1*A*). Compared to a uniform concentration in the upper 2/3 of the spleen, the lower 1/3 showed a patchy concentration. On the assumption that the patchy concentration in the lower 1/3 of the spleen was due to radiation fibrosis another course of roentgen-ray therapy was given to a portion of the spleen above the left costal margin. A total of 550 r was delivered. Figure 1*B* shows the spleen scan of the patient immediately after the radiation therapy. The scan reveals a definite reduction in the size of the spleen. No extension of the spleen below the left costal margin is demonstrated. The red blood cell count dropped. A spleen scan obtained $7\frac{1}{2}$ months later (Fig. 1*C*) showed further reduction in the size of the spleen. The disease was well under control during this period without any specific treatment.

CASE II. Three years prior to the present examination a diagnosis of Hodgkin's disease had

been made from an inguinal lymph node biopsy in this 52 year old male. He had been previously treated with roentgen-ray therapy of the neck and inguinal lymph nodes. At the present examination he had hard and matted lymph nodes in both axillary and inguinal regions. The spleen and liver were enlarged. The scan demonstrated a large spleen extending 2 inches below the left costal margin (Fig. 2*A*). A well-defined comparatively "cold" mass was seen in the region of the hilus and along the medial border of the lower pole of the spleen. A scan of the patient 12 days after the initial scan is shown in Figure 2*B*. The patient received 200 mg. of endoxan by intravenous route daily during the interim period (for a total dose of 2,200 mg.). A considerable reduction in the size of the spleen ensued although the over-all length of the spleen in the 2 scans remained almost the same. The large "cold" mass noted in the previous scans has completely disappeared.

Case III. A 68 year old female was referred to us with a history of 2 attacks of coronary thrombosis. Clinical examination and laboratory investigations indicated polycythemia vera. The spleen was not palpable. The scan showed an enlarged comma-shaped spleen which extended an inch below the left costal margin, along with a longer projection along the lateral abdominal wall (Fig. 3*A*). A repeat scan (Fig. 3*B*) obtained when the polycythemia was under control after 7 mc of oral radioactive phosphorus revealed a marked reduction in the size of the spleen. No extension below the left costal margin was noted.

Case IV. A 66 year old male patient presented with a history of hypertension. He had multiple ecchymotic pathes over both extremities. Evidence of deep seated thrombosis in both legs was also present. The liver and spleen were not palpable. Laboratory investigations suggested polycythemia vera. The scan showed a very bulky spleen even though it did not extend be-

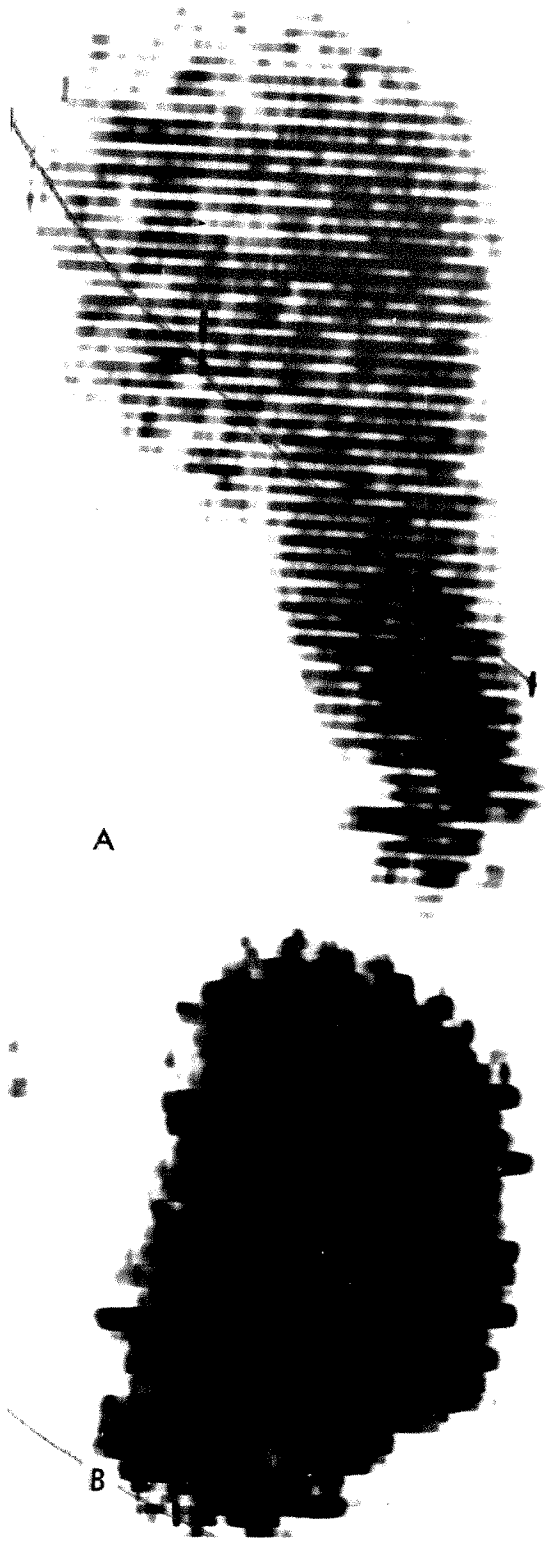



Fig. 3. Polycythemia vera in a 68 year old female. The spleen was not palpable. (*A*) Scan shows an enlarged spleen. A narrow projection along the lateral abdominal wall is noted. (*B*) Repeat scan after an oral 7 mc dose of radiophosphorus. The disease is under control and there is a marked reduction in the spleen size.

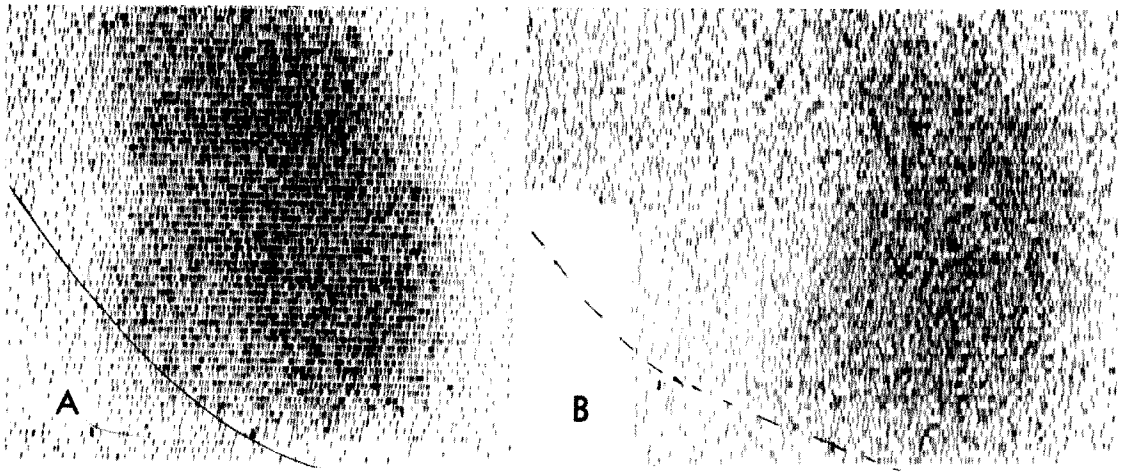



FIG. 4. Polycythemia vera in a 66 year old male. The spleen was not palpable. (*A*) Scan showing a bulky spleen even though no extension below the left costal margin is seen. (*B*) Repeat scan after an oral 7 mc dose of radiophosphorus. With the disease under control there is a considerable reduction in the bulk of the spleen.

low the left costal margin (Fig. 4*A*). A repeat scan obtained 1 year after the first scan is shown in Figure 4*B*. During this period he had received an oral dose of 7 mc of radioactive phosphorus. The scan reveals a regression in the size of the spleen of more than 1 inch in length and its bulk is also considerably reduced.

## DISCUSSION

It is generally recognized that all forms of treatment of leukemia and other allied blood dyscrasias aim at prolonging the life span of the patient with minimum morbidity.[7] It is, therefore, desirable that maximum advantage should be taken of either radiation therapy or chemotherapy as a preferred mode of treatment. The patient with chronic myeloid leukemia (Case 1; Fig. 1, *A–C*) demonstrated the usefulness of spleen scanning in obtaining the maximum benefit from splenic roentgen irradiation. Repeated roentgen-ray treatments to the same area of the spleen during a period of 4 years probably resulted in radiation-induced interstitial fibrosis in the lower $\frac{1}{3}$ of the spleen. The last course of splenic irradiation at the same site, just before spleen scanning, therefore did not produce a remission. The spleen scan proved helpful in detecting an area of the spleen with good concentration; another dose of 550 r over this area produced a remission lasting for $7\frac{1}{2}$ months.

The Hodgkin's mass in the region of the hilus of the spleen (Fig. 2*A*) disappeared after 12 days of intravenous endoxan therapy (Fig. 2*B*). The post-therapy scan in this patient has provided definite evidence of regression of the lesion in the spleen.

An interesting feature of the 2 patients with polycythemia vera was that even though there was no clinical evidence of a palpable spleen, the spleen scans demonstrated an enlarged spleen in both instances. The scan of the first patient (Fig. 3*A*) showed enlargement a little below the left costal margin and a narrow projection along the lateral abdominal wall. The scan of the second patient (Fig. 4*A*) showed bulky enlargement without extension below the left costal margin. Lacking a spleen scan, these 2 patients with polycythemia vera would have been considered as having normal spleens. Calabresi and Meyer,[1] and Szur *et al.*[10] have described splenomegaly in three-fourths of their cases, while Wintrobe[13] has reported a 90 per cent incidence of splenomegaly in his series of polycythemia vera cases. It is possible that a substantial portion of the 10–25 per cent polycythemia vera cases found to be without any palpable spleen might reveal splenic

enlargement on scanning. There is no doubt that spleen scans can provide definite evidence of the presence or absence of an enlarged spleen in polycythemia vera.

SUMMARY

The usefulness of spleen scans in the selection of an area of the spleen for further radiation therapy in a refractory patient with chronic myeloid leukemia is demonstrated.

Estimation of the degree of reduction of spleen size in response to treatment of patients with chronic myeloid leukemia, Hodgkin's disease and polycythemia vera is described as is the disappearance of a Hodgkin's mass following endoxan therapy.

The value of spleen scans in detecting the presence or absence of splenomegaly in cases of polycythemia vera without a palpable spleen is also discussed.

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# RADIONUCLIDE DIAGNOSIS OF MALIGNANT TUMORS OF THE KIDNEY*

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NUMEROUS articles have appeared in the literature evaluating radiochlormerodrin as a renal scanning agent.[2,3,4,8] Although it is eminently suitable in detecting space occupying lesions, the distinction between cyst and malignant neoplasm cannot be made. In a preliminary report[5] the feasibility of visualizing tumor vasculature with technetium 99m pertechnetate and the gamma-ray scintillation camera[6] was advanced. Thirty cases of cysts and malignant neoplasms have been accumulated since the initial report. These have been verified by selective renal arteriography and/or laparotomy.

## TECHNIQUE

One hundred microcuries radiochlormerodrin are injected intravenously and about 2 hours later the patient is placed in the prone position and the kidneys are localized with the gamma-ray scintillation camera (Pho/Gamma, Nuclear-Chicago). Without moving the patient, 10 to 15 millicuries of $Tc^{99m}$ pertechnetate are rapidly injected into an antecubital vein and serial 5 second exposure scintiphotos are obtained.

## RESULTS AND DISCUSSION

The usual sequence of appearance of the bolus of $Tc^{99m}$ in the serial 5 second exposure scintiphotos is the abdominal aorta, both kidneys and spleen almost simultaneously, and finally the liver against an elevated background of abdominal activity. The delayed liver visualization stems from the fact that the portal vein contributes about 80 per cent of the total hepatic blood flow. Figure 1, *A–D* is an example of a normal study.

Figure 2, *A–C* illustrates a patient with a cyst in the right kidney. The radiochlormerodrin scan exhibits nonfunctioning cortex in the upper pole (*A*), and the $Tc^{99m}$ renal series fails to show a detectable blood flow in the area of the defect (*B*). This favors cyst over malignant neoplasm. A comparative selective right renal arteriogram is seen in *C*.

A patient with a long history of uric acid stones and 2 previous nephrotomies is presented in Figure 3, *A* and *B*. The excretory urogram showed poor concentration and some distortion of the superior group of calyces. The radiochlormerodrin scan depicted a large lesion involving the medial aspect of the right kidney (*A*). A $Tc^{99m}$ renal flow series showed a higher concentration of activity in the lesion than in the functioning parenchyma (*B*). A diagnosis of neoplasm was made on the basis of the defect being vascular. Arteriography was not performed, but the kidney was removed and it revealed a large hypernephroma corresponding to the radionuclide examination findings.

A hypernephroma is illustrated in Figure 4, *A–E*. The aortogram (*A*) and selective left arteriogram (*B*) showed that only part of the neoplasm was vascular. The radiochlormerodrin scan (*C*) depicted cortical replacement at the lower pole of the left kidney and the $Tc^{99m}$ renal flow series, which followed, reflected the tumor vasculature (*D* and *E*).

The following example is that of a 65 year old female who was investigated for hematuria. An excretory urogram showed a large mass emanating from the lower pole of the left kidney (Fig. 5*A*). A short rim of calcium was noted at the inferior

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margin of the mass. The selective left renal arteriogram demonstrated tumor vessels and staining in the upper portion of the neoplasm (Fig. 5*B*). Figures 5, *D*, *E* and *F* are taken from the $Tc^{99m}$ flow series and illustrate the tumor "blush."

A 70 year old male with a large right upper quadrant mass and a nonfunctioning right kidney on excretory urography exhibited a poor blood flow with $Tc^{99m}$ which did not conform to a renal configuration (Fig. 6, *A–C*). The preoperative interpretation was malignant neoplasm that was largely necrotic. The pathologic diagnosis was sarcoma that was neither necrotic nor as vascular as previously encountered hypernephromas.

Late scintiphotos often show an increased concentration of activity within the tumor, and these cases can be equally well demonstrated with a conventional rectilinear scanner.[1] However, in those situations where the tumor vasculature is not very prominent, the high background activity of the late scans will obscure the tumor. Similarly, cysts and neoplasms in

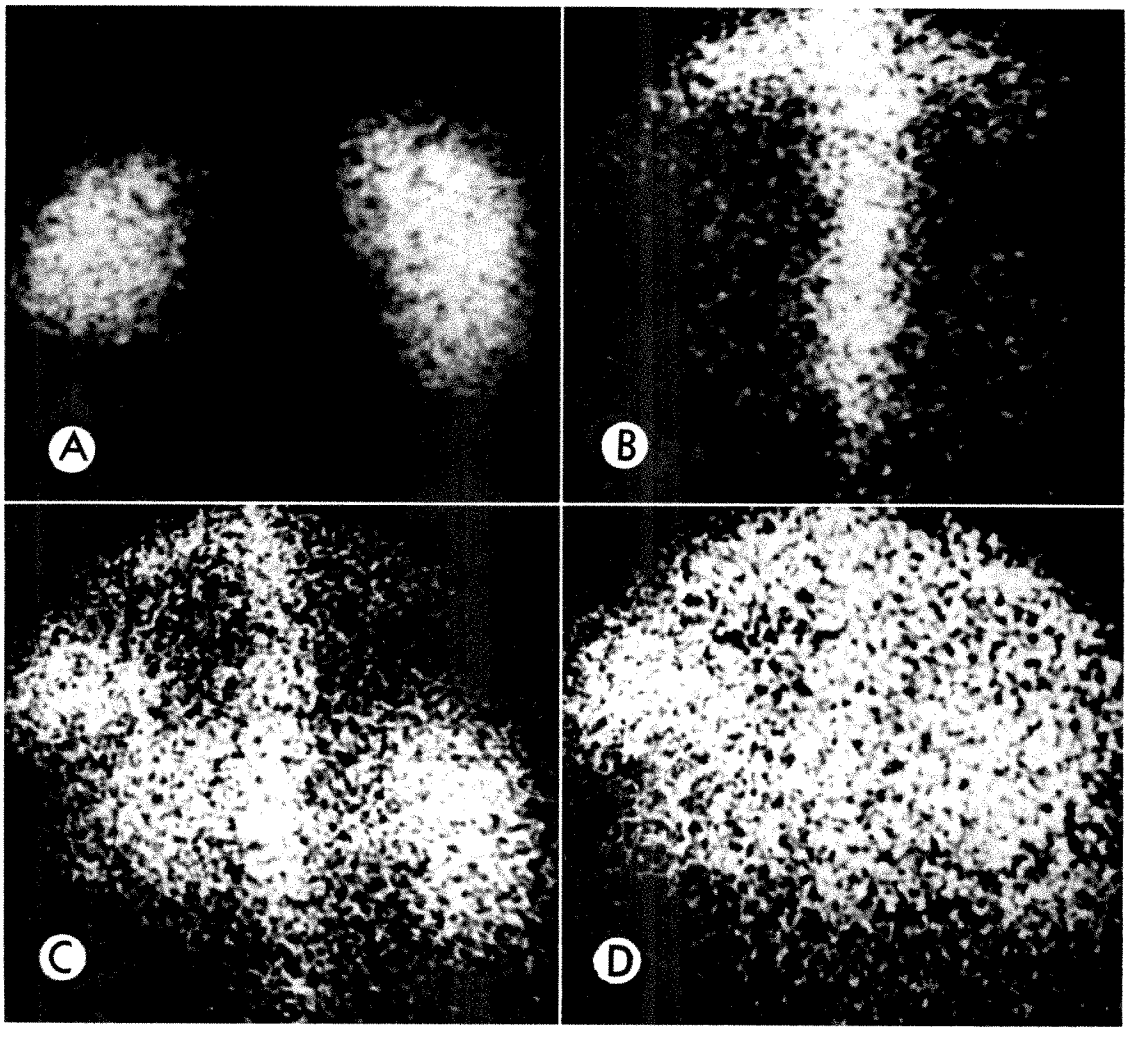



FIG. 1. (*A*) A prone radiochlormerodrin renal scan obtained just prior to the intravenous injection of $Tc^{99m}$ pertechnetate. (*B*) The bolus of $Tc^{99m}$ is seen primarily in the aorta in the 9 to 14 second interval. (*C*) In the 15 to 19 second interval both kidneys, spleen, and aorta are visualized. (*D*) In the 30 to 34 second interval activity is demonstrated in the liver against a generally elevated background.

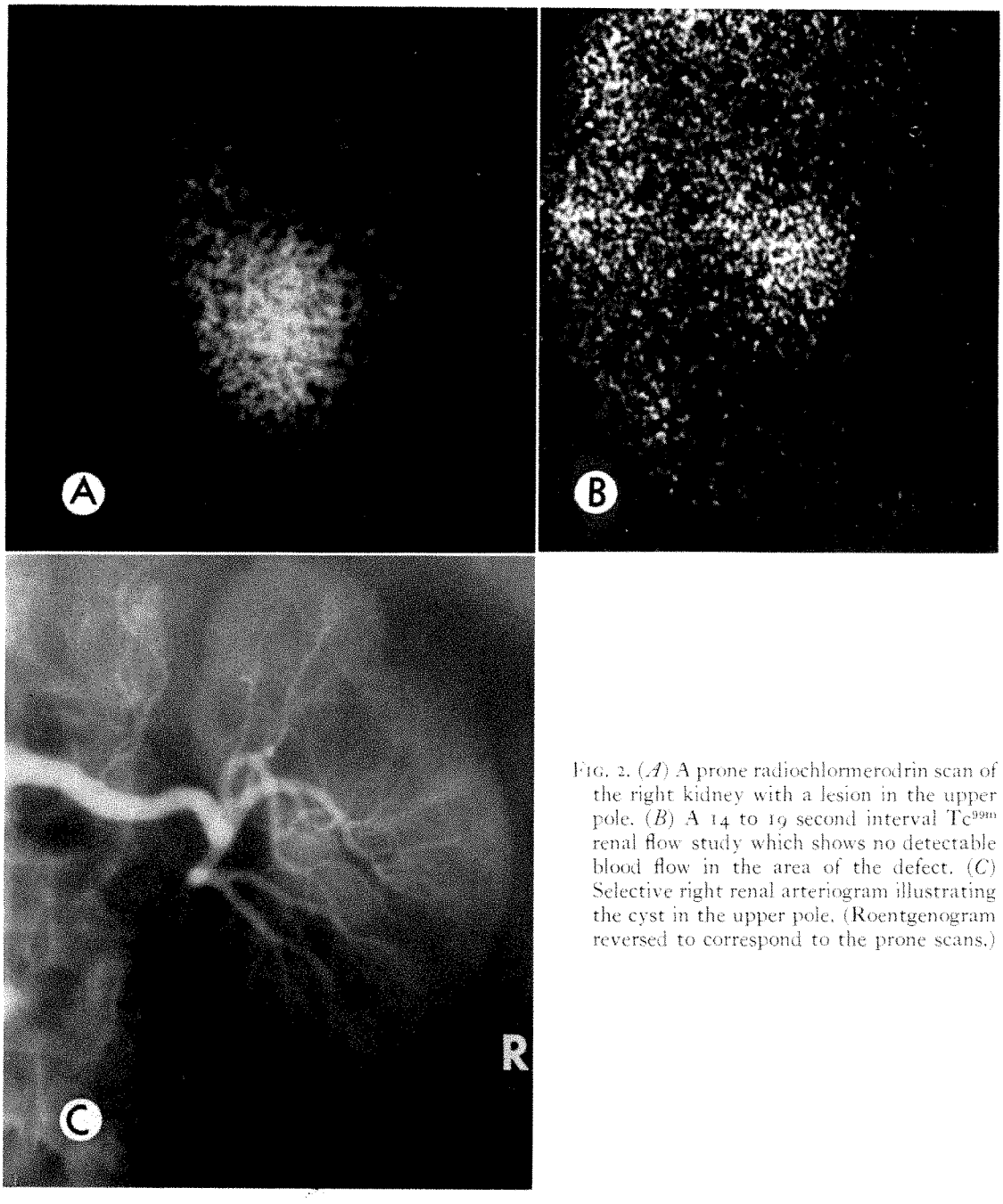



FIG. 2. (*A*) A prone radiochlormerodrin scan of the right kidney with a lesion in the upper pole. (*B*) A 14 to 19 second interval $Tc^{99m}$ renal flow study which shows no detectable blood flow in the area of the defect. (*C*) Selective right renal arteriogram illustrating the cyst in the upper pole. (Roentgenogram reversed to correspond to the prone scans.)

»»→

FIG. 4. (*A*) An aortogram showing a renal neoplasm involving the left kidney. (Roentgenogram reversed to correspond to the prone radionuclide study.) (*B*) Corresponding left selective renal arteriogram exhibiting the tumor vasculature to better advantage. (*C*) Radiochlormerodrin renal scintiphoto obtained prior to injection of $Tc^{99m}$, exhibiting destruction of the lower pole of the left kidney. (*D*) Appearance of $Tc^{99m}$ in the aorta. (*E*) Activity seen in the tumor (arrows).

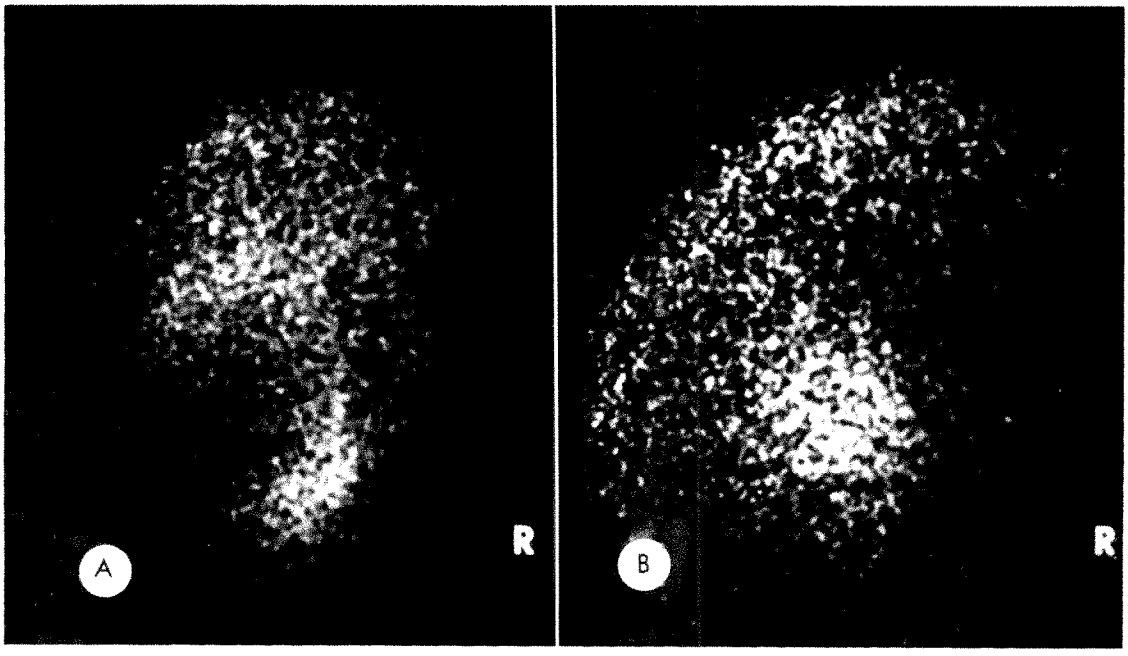
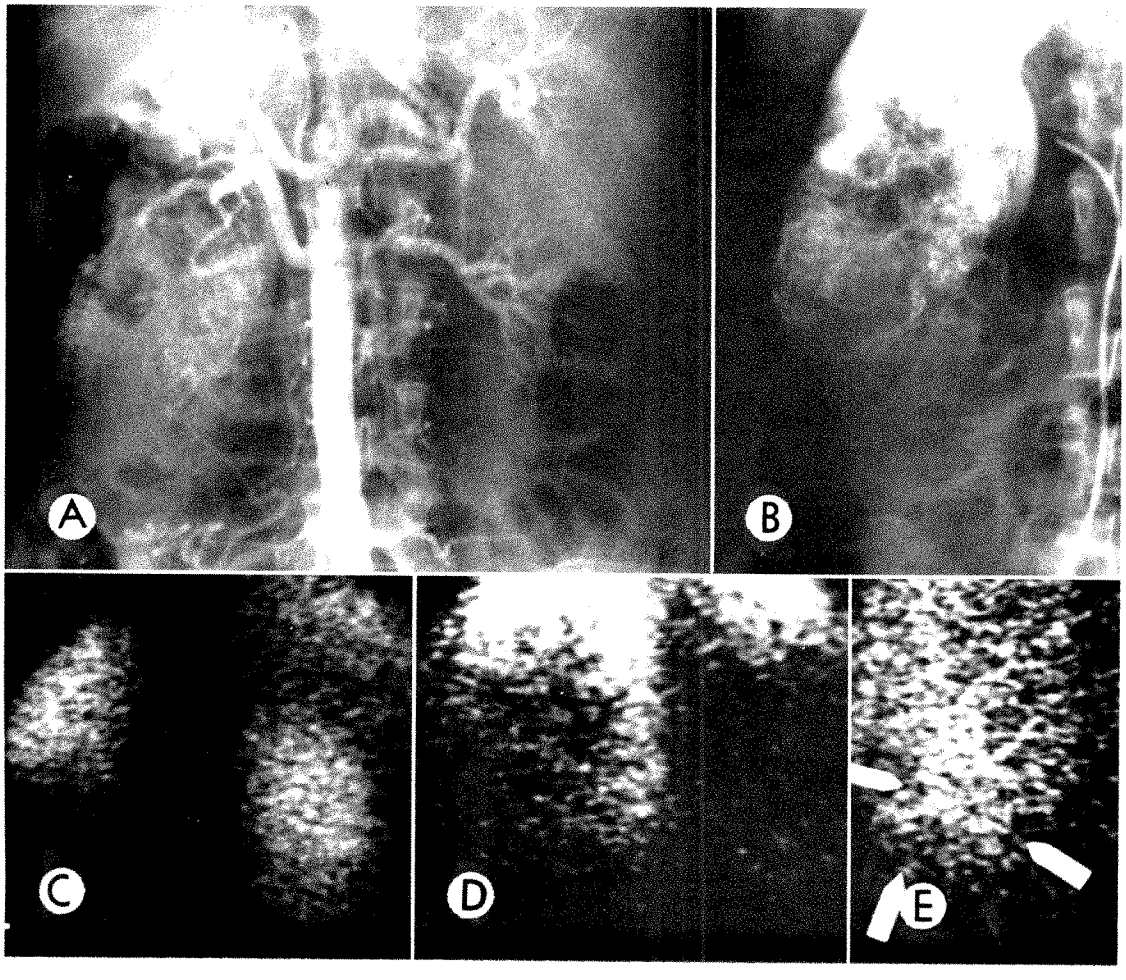



FIG. 3. (*A*) A prone radiochlormerodrin renal scan with a large defect in the medial aspect of the right kidney. (*B*) Corresponding $Tc^{99m}$ flow study exhibiting more activity in the region of the defect than in the upper and lower poles of the kidney. The defect was found to be a large hypernephroma at laparotomy.



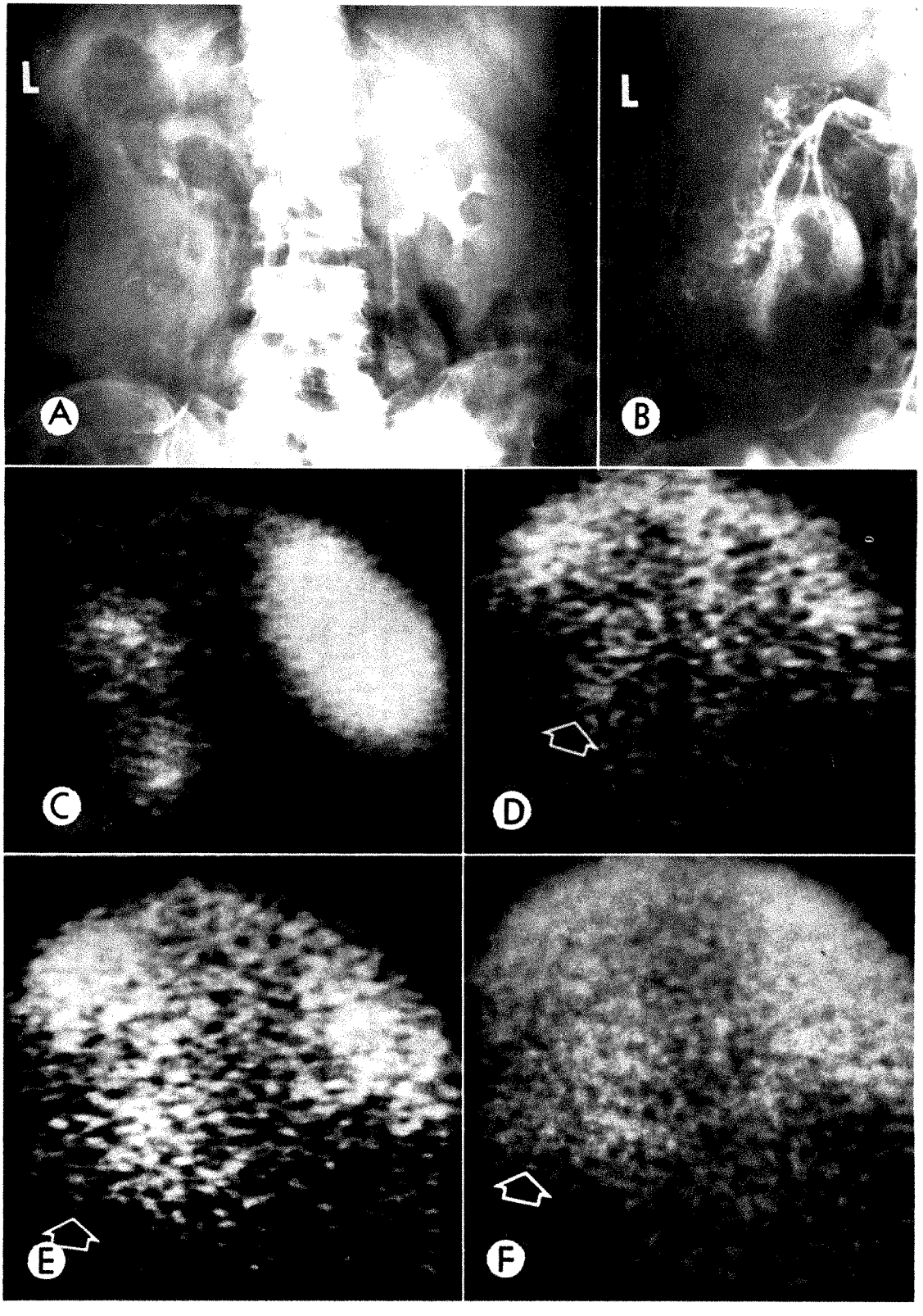

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FIG. 5. (*A*) Intravenous pyelogram showing a large mass emanating from the lower half of the left kidney. A short rim of calcium is seen on the inferior border of this mass. (Roentgenogram reversed to correspond to the prone radionuclide studies.) (*B*) Selective left renal arteriogram depicting tumor vessels in the upper half of this mass. (Roentgenogram reversed to correspond to the prone radionuclide studies.) (*C*) A prone radiochlormerodrin scintiphoto showing a large area of cortical replacement in the left kidney and reduced function in the remaining viable cortex. (*D*) Technetium 99m flow study, 15 to 19 second interval, illustrating early detection of activity in the region of the defect (arrow). (*E*) A 20 to 24 second interval study shows further accumulation of activity in the area of the defect (arrow). (*F*) A late 80,000 count scintiphoto taken 1 minute after injection showing the vascular portion of the hypernephroma (arrow) to somewhat better advantage in this particular case.

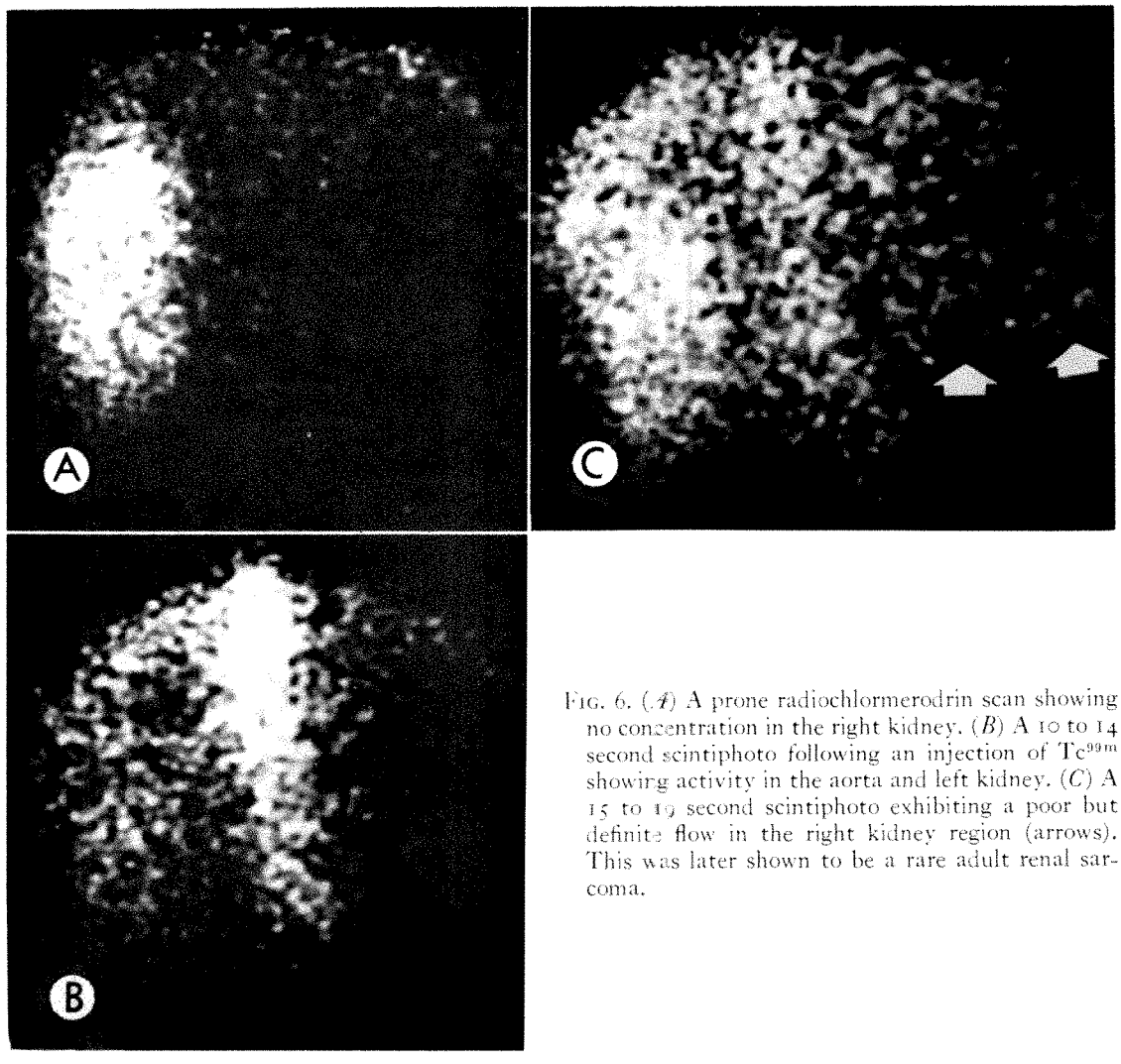



FIG. 6. (*A*) A prone radiochlormerodrin scan showing no concentration in the right kidney. (*B*) A 10 to 14 second scintiphoto following an injection of $Tc^{99m}$ showing activity in the aorta and left kidney. (*C*) A 15 to 19 second scintiphoto exhibiting a poor but definite flow in the right kidney region (arrows). This was later shown to be a rare adult renal sarcoma.

the upper half of the right kidney can also be obscured by activity in the liver.

$Tc^{99m}$ pertechnetate is known to diffuse readily into the tissues, and activity seen in the tumor probably represents interstitial and vascular compartments. We have not performed parallel studies with human serum albumin labelled with $Tc^{99m}$.

The $Tc^{99m}$ flow study is somewhat akin to nephrotomography and serves as another screening procedure. Resolution is inferior to the contrast method, but patient radiation exposure is less,[7] the image is not influenced by gas and fecal material, and there are no known deleterious effects from technetium pertechnetate.

There are other limitations of the technique. It may not be possible to visualize the tumor vasculature in a neoplasm that is largely necrotic, even on careful comparison with the radiochlormerodrin scan. Thus, when $Tc^{99m}$ is seen within a defect it is almost certainly a neoplasm. In the absence of $Tc^{99m}$ the defect is most likely a cyst or an area of ischemia, but tumor is not entirely ruled out.

SUMMARY

Renal tumor vasculature can be detected by monitoring the kidneys with the gamma-ray scintillation camera following a rapid intravenous injection of $Tc^{99m}$ pertechnetate. It is, therefore, possible to distinguish neoplasm from cyst or ischemia.

Some of the limitations of this modality are discussed.

Its advantages as another screening procedure are that it is rapid, innocuous, requires no patient preparation, is free of interference from gas and fecal material, and gives low radiation exposure.

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# ARTERIOGRAPHY FOLLOWING RENAL TRANSPLANTATION*

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A REVIEW of several large series of renal transplantations showed only one communication dealing with vascular studies following this procedure.[1,2,8,10,15,16] Arteriography has been of benefit at this institution in evaluation of the post-transplantation status of 6 patients. The findings in 10 post-transplantation arteriograms obtained in these 6 patients will be described.

## MATERIAL

There were 3 men and 3 women ranging in age from 15 to 40 years. Six transplanted kidneys were each studied once. Repeat examinations were performed on the same transplant in 2 patients. Six transplantations were cadaver kidneys and 2 were from live donors. The indication for arteriographic study was failure to produce urine immediately (within 1 hour) after surgery or a period of oliguria or anuria any time following surgery suggesting the possibility of renal artery thrombosis or transplant rejection. The time between the transplantation and the arteriography ranged from 1 to 125 days. The time between the arteriography and examination of the transplanted kidney at surgery or post mortem ranged from 3 hours to 60 days (Table 1). Sixty per cent methylglucamine diatrizoate (renografin*) was used in 8 examinations and 75 per cent sodium and meglumine diatrizoate (hypaque†) in 2. The volume per examination ranged from 30 to 50 ml.

The clinical and pathologic diagnoses in the recipients prior to transplantation were chronic glomerulonephritis in 3 patients, and arteriolar nephrosclerosis, advanced gouty nephropathy, and bilateral renal vein thrombosis in 1 patient each.

## SURGICAL TECHNIQUE

The donor kidney is transplanted into the same or opposite iliac fossa of the recipient. When transplanted into the opposite iliac fossa the kidney is reversed so that the renal pelvis and ureter are anterior and the renal artery and vein posterior. The renal artery is anastomosed end-to-end to the hypogastric artery and the vein end-to-side to the external iliac vein. The ureter of the donor kidney is anastomosed into the recipient's bladder wall or if an insufficient length of donor ureter is available a uretero-ureteral anastomosis is made.[10,11,14]

## REPORT OF CASES

CASE 1. This 37 year old Negro female was first admitted in August, 1963 with azotemia due to arteriolar nephrosclerosis shown by renal arteriography (Fig. 1)[7] and proven by bilateral renal biopsy.

One month after admission a right cadaver kidney was transplanted into the left iliac fossa. Urinary output following transplantation was 2,000 cc. per day. She was maintained on azathioprine (imuran) and a short course of cyclophosphamide (cytoxan). Fourteen days following transplantation the urine volume decreased and there were other signs of allograft rejection.

Seventeen days following transplantation a renal arteriogram was made by retrograde manual injection of 30 ml. of 60 per cent renografin through a cardiovascular needle inserted into the left common femoral artery (Fig. 2, *A–C*). The anastomosis was patent. The renal artery branches were spread due to edema

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| | Transplantation | | Arteriogram | | Renogram (R) or Renal Scan (RS) | | Physiologic Data at Arteriography | | | | Anatomic Findings of Transplanted Kidney |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Date | Location | Date | Findings | Date | Findings | Blood Pressure | Blood Urea Nitrogen | Urine Output (cc./24 hr.) | Creatinine Clearance (cc./min.) | |
| Case 1 | 9-23-63 | LIF | 10-10-63 | Edema; intact anastomosis; faint nephrogram | 10-11-63 | (RS) Nonvisualization | 140/100 | 114 | 1,440 | — | 10-18-63<br>(N) Recent extraperitoneal hemorrhage; large renal vessels intact; disruption of uretero-vesical anastomosis<br>(H) Mild rejection; focal thrombi in glomerular arterioles |
| Case 2 | 10-11-63 | RIF | 10-12-63 | Extrinsic compression of renal artery; delayed flow; absence of small vessels and nephrogram | — | — | 170/90 | 90 | 100 | — | 10-12-63<br>(S) Numerous clots about graft; stretched, compromised renal artery and vein<br>10-16-63<br>(N) Extensive retroperitoneal hemorrhage; occlusion of upper pole branch renal artery; arteriolar nephrosclerosis |
| Case 3 | 7-27-64 | RIF | 8-27-65 | Marked narrowing of renal artery and branches; absent nephrogram | 8-23-65 | (R) Subnormal vascular and secretory phases | 140/120 | 49 | 1,000 | 8 | 4- 6-65<br>(B) Chronic rejection<br>11-17-65<br>(N) Chronic rejection |
| | 10-16-65* | AB | 11- 8-65 | Occlusion of renal artery | 10-18-65 | (R) Decreased vascular phase | 160/100 | 67 | Anuric | — | 11-17-65<br>(N) Thrombosis of renal artery and vein |
| Case 4 | 2-23-65† | LIF | 6-14-65 | Stenosis at anastomosis | — | — | 180/130 | 40 | — | — | 6-15-65<br>(S) Stenotic segment resected |
| | | | 6-28-65 | Patent anastomosis | 6-29-65 | (R) Normal | 120/80 | 30 | 3,000 | 40 | (H) Intimal hyperplasia<br>(B) Mild rejection |
| Case 5 | 3-25-65 | RIF | 3-25-65 | Small kidney tortuous vessels; delayed flow | 3-25-65 | (R) Major vascular occlusion | 210/110 | 108 | Anuric | — | 3-25-65<br>(S) No thrombosis; small kidney |
| | 8-21-65 | LIF | 8-25-65 | Edema of kidney; embolus of popliteal artery | 8-25-65 | (R) Subnormal vascular and secretory phase | 180/100 | — | 555 | 3 | (H) Cortex thinned to 4 mm.; coagulative necrosis; acute inflammation secondary to prolonged ischemia<br>11-26-65<br>(N) Renal vein thrombosis; gastrointestinal hemorrhage and wide spread cytomegalic inclusion disease |
| Case 6 | 11- 6-65 | LIF | 11-10-65 | Marked delay of flow (17 sec.); absent small peripheral vessels and nephrogram | 11-9-65 | (R) Minimal vascular phase; absent secretory phase | 140/80 | 123 | 300 | — | None to date |
| | | | 12-3-65 | Normal flow; absent small peripheral vessels; faint nephrogram | | | | 900 | | | |

LIF—Left Iliac Fossa; RIF—Right Iliac Fossa; AB—Aortic Bifurcation; N—Necropsy; S—Surgery; B—Biopsy; H—Histology.

of the kidney. The flow of contrast material was normal, the small peripheral vessels were narrow and a poor nephrogram was seen. The findings were interpreted as rejection reaction. A renal scan the next day showed nonvisualization of the graft.

Increased dosage of imuran and steroids and a course of methotrexate therapy did not improve renal function. She died of pulmonary edema 25 days following transplantation and 8 days after arteriography.

At postmortem examination the transplanted kidney was extremely edematous. The vascular anastomoses were intact and patent. The uretero-vesical anastomosis was disrupted. The kidney contained small recent focal infarcts and varying degrees of degeneration of the glomerular tubules together with tortuosity and swelling of the basement membranes consistent with a mild rejection.

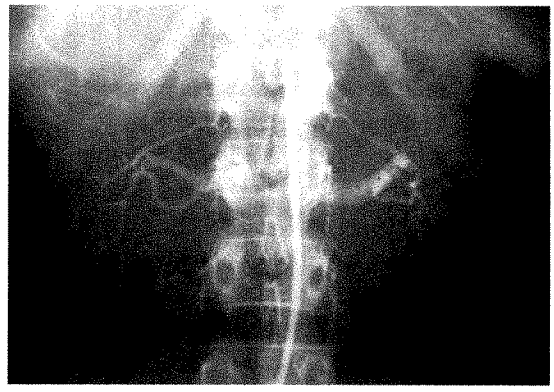


FIG. 1. Case 1. Chronic glomerulonephritis. Arteriogram of recipient's kidneys prior to transplantation. The kidneys are small. The peripheral branches are not visualized. There was no nephrogram. Histologic examination showed chronic glomerulonephritis.

CASE 2. This 19 year old man was seen in May, 1963 with azotemia due to chronic glomerulonephritis.

In October, 1963 the resected left kidney of a 61 year old man with hypertension due to renal artery stenosis was implanted into the patient's right iliac fossa. The donor kidney had been removed because of difficulty in attempting to repair the stenosis. The recipient's ureter was anastomosed to the pelvis of the donor kidney. Bleeding from one portion of the vascular anastomosis required additional sutures. There was tension on both the artery and the vein due to shortness of the vessels. Immediately after transplantation, renal function appeared adequate with an output of 2–3 ml. per minute.

Several hours after surgery the urinary volume decreased. The patient became hypotensive and showed evidence of retroperitoneal hemorrhage. A retrograde pyelogram demonstrated an intact pelvic-ureteral anastomosis (Fig. 3). Arteriography was performed using 50 ml. of 60 per cent renografin injected retrograde manually through a percutaneous cardiovascular needle in the right femoral artery

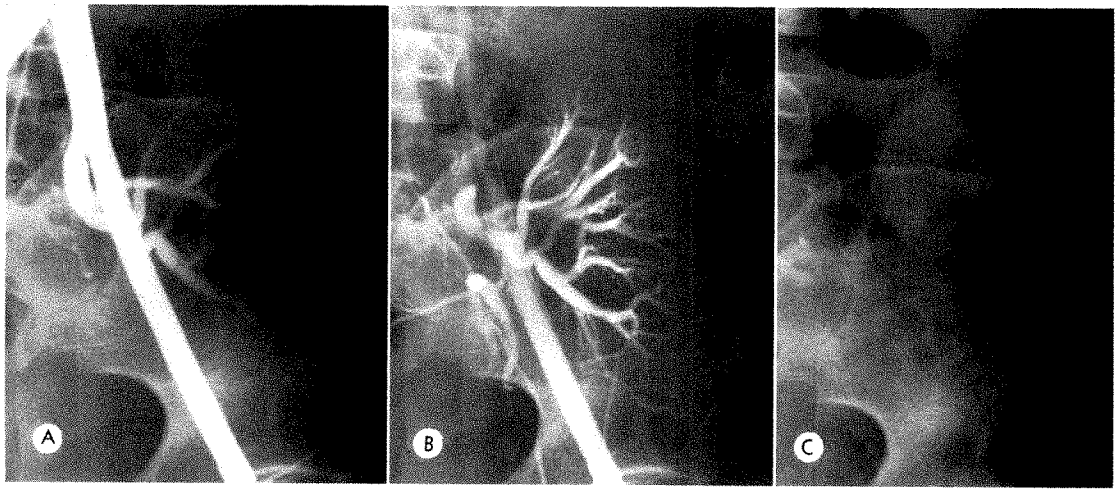



FIG. 2. Case 1. Mild rejection reaction. Arteriography of transplanted kidney by manual retrograde injection through a cardiovascular needle in the left common femoral artery. (*A*) The anastomosis is intact. (*B*) The kidney is swollen and the smaller vessels are well visualized. (*C*) The nephrogram is very faint. Histologic examination showed mild rejection with focal thrombi in the glomerular vessels.

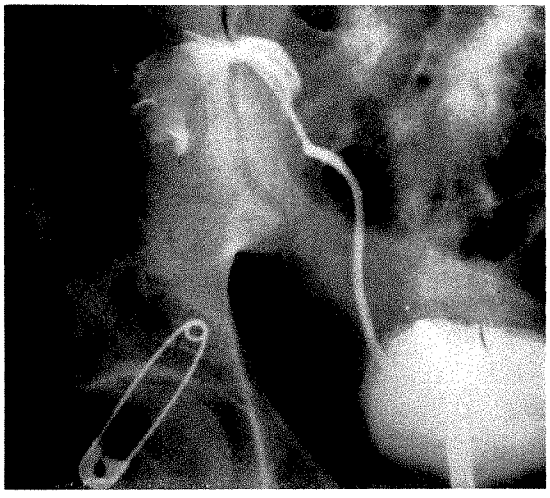


FIG. 3. Case 2. A retrograde pyelogram demonstrates an intact pelvic-ureteral anastomosis. Fluid is present in the right iliac fossa.

(Fig. 4, *A* and *B*). The study demonstrated marked narrowing and stretching of the hypogastric and renal arteries. The artery to the upper pole of the kidney was occluded. A filling defect in the center of the hypogastric artery proximal to the anastomosis appeared to be a thrombus. A slight indentation upon the external iliac artery where the hypogastric artery crossed suggested the possibility of compression of the hypogastric artery by clot and tension on the vessel. The smaller peripheral renal vessels were absent and there was delay in washout of the renal vessels. No nephrogram was seen. This suggested diffuse parenchymal disease due to an acute rejection phenomenon or secondary to arteriolar nephrosclerosis.

Immediately following the arteriography surgical exploration demonstrated a large number of clots about the graft, the right iliac fossa and the right paravertebral gutter. The vascular anastomoses were patent but compressed. General oozing was apparent throughout the graft bed but no bleeding site in the anastomosis was found. After surgery bleeding continued and urinary output was never resumed. The patient died 4 days following the original operation.

At autopsy there was extensive retroperitoneal hemorrhage. The artery to the upper pole of the kidney was thrombosed. The vascular anastomosis was intact and patent. Microscopic examination showed marked arterial and arteriolar nephrosclerosis.

CASE 3. This 33 year old white man was first seen in October, 1963 because of azotemia due to chronic glomerulonephritis. On July 27, 1964 a left cadaver kidney was transplanted into the patient's right iliac fossa. Fourteen days later a mild rejection phenomenon was

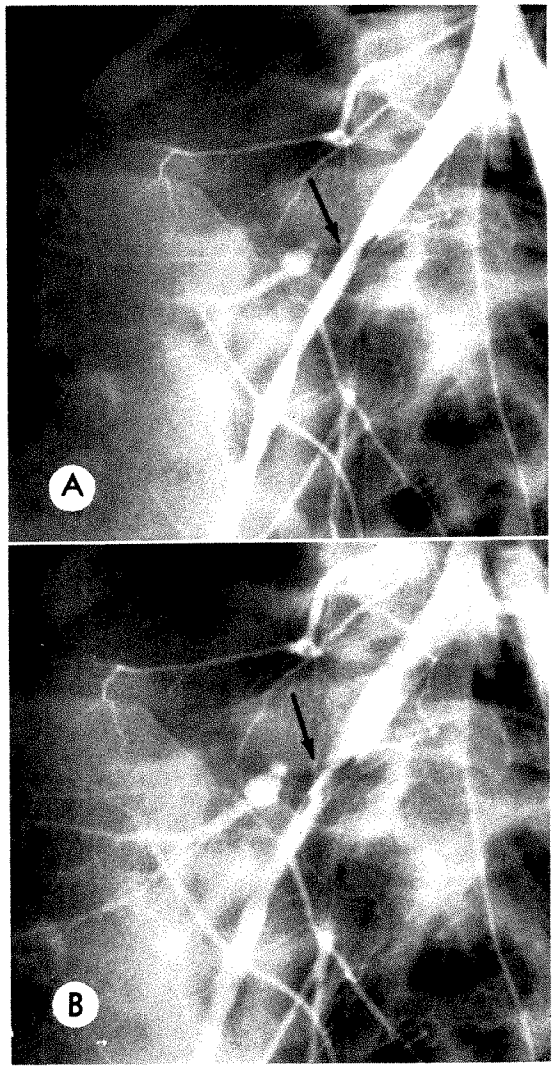



FIG. 4. Case 2. Extrinsic compression of renal artery. Arteriolar nephrosclerosis. (*A*) Renal arteriogram by retrograde injection into the right femoral artery shows an apparent thrombus at the site of anastomosis due to extrinsic compression (arrow). (*B*) The peripheral vessels are markedly attenuated and the upper pole artery is occluded. At surgery the renal artery and vein were found to be stretched and compressed by extrinsic clot. Only the upper pole renal artery was occluded. Autopsy showed arterial and arteriolar nephrosclerosis and no evidence of rejection.

treated with steroids and 6-mercaptopurine. Thereafter the patient had a useful life while being maintained on imuran and methyldopate HCl (aldomet).

An open biopsy of the transplanted kidney on April 6, 1965 because of consistent proteinuria of 1–2 grams per 24 hours for the prior 5 months showed changes compatible with graft rejection. Thrombosis of the right femoral vein occurring suddenly on May 14, 1965 was treated by thrombectomy and saphenous vein ligation.

Following discharge the patient was generally well but the 24 hour urine contained 6–7 grams of protein. Initially the blood urea nitrogen was recorded in the range of 26 mg. per cent and creatinine 2.2 mg. per cent. There was progressive deterioration and 3 days prior to the last admission on August 22, 1965 the blood urea nitrogen had risen to 62 mg. per cent and the creatinine to 4.7 mg. per cent. The blood pressure was 140/70 mm. Hg. The creatinine clearance was 8 ml. per minute.

An $I^{131}$ hippuran renogram was compatible with vascular and parenchymal disease. A renal arteriogram 4 days later demonstrated markedly narrowed vessels involving both the renal artery and its branches (Fig. 5). The larger branches were patent, the finer branches were not visualized and there was no nephrogram. The findings were thought to be compatible with a rejection reaction. The patient's condition continued to deteriorate and an attempt was made to transplant a second kidney to the left iliac fossa but failed because of technical problems. The previously transplanted kidney was resected 10 weeks after arteriography and demonstrated microscopically a second stage rejection reaction with vascular edema, chronic inflammation and tubular atrophy. The renal artery intima was thickened by loose fibroblastic proliferation to the anastomosis. The renal vessels and the site of anastomosis were patent.

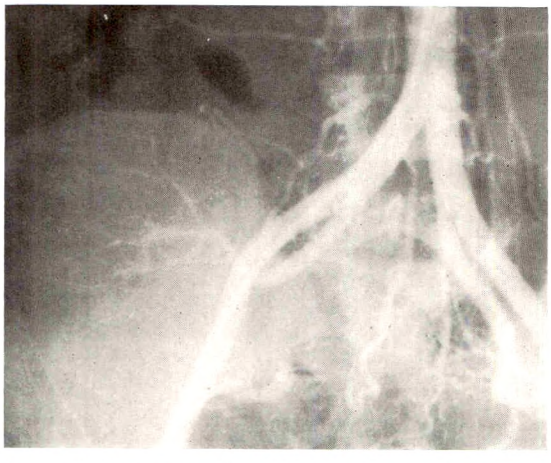


Fig. 5. Case 3. Rejection. First renal transplantation. The smaller branches of the renal artery are obliterated. The larger branches are irregularly narrowed and involve the main renal artery to the site of anastomosis. The kidney is enlarged. No nephrogram was seen.

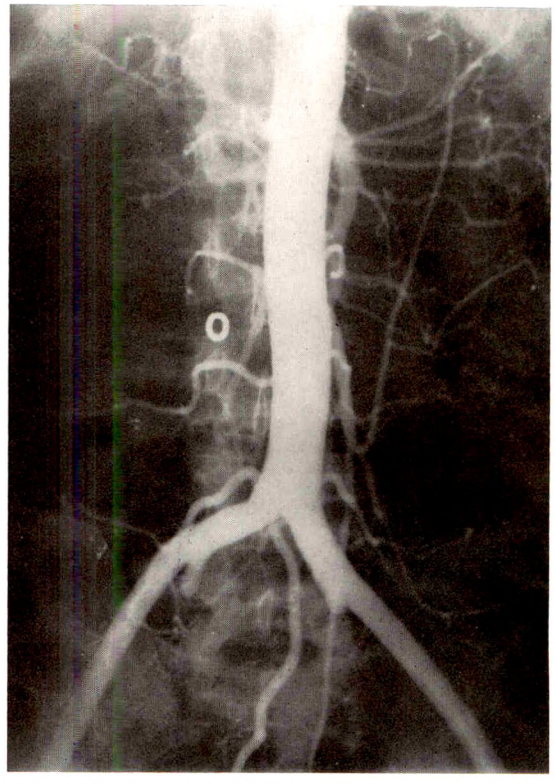


Fig. 6. Case 3. Occlusion of the transplanted renal artery. Second renal transplantation. The right hypogastric artery is resected and the left occluded. The stump of the right renal artery is seen at the level of L1. The middle sacral, inferior mesenteric and right iliolumbar arteries have enlarged to serve as collateral vessels.

Two months later a second renal transplantation was done with anastomosis of the renal artery into the aortic bifurcation and the renal vein into the inferior vena cava at the same level. Following this transplantation the patient was persistently anuric. A second arteriogram showed occlusion of both right and left hypogastric arteries and both renal arteries (Fig. 6). No graft was visualized. The patient died

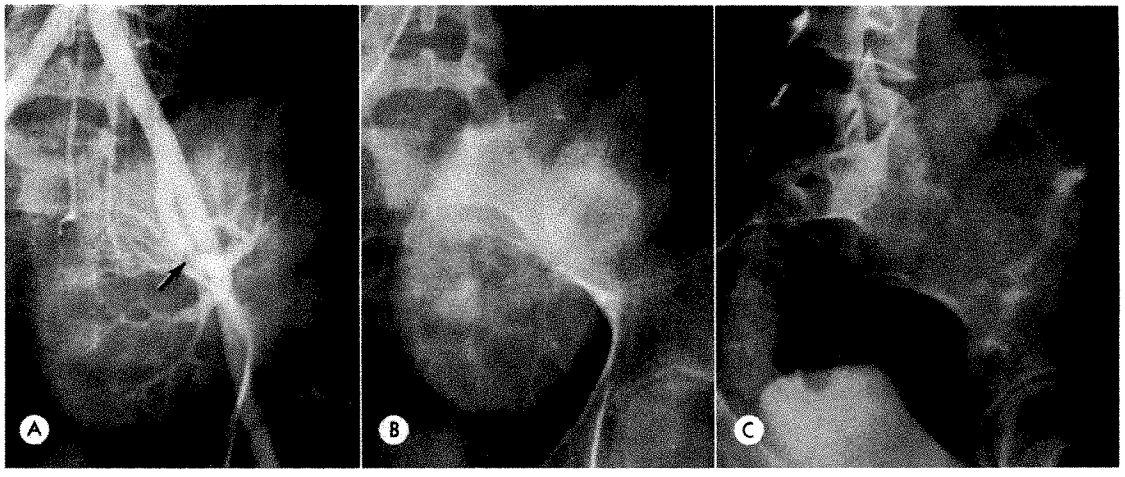



FIG. 7. Case 4. *June 14, 1965*: Anastomotic stenosis. (*A*) Renal arteriogram shows marked stenosis at the anastomotic site (arrow). The kidney is swollen. (*B*) The nephrogram is intense and (*C*) the collecting system is well visualized.

shortly afterwards. Postmortem examination demonstrated total necrosis of the second transplanted kidney with thrombi occluding the renal artery and vein.

CASE 4. This 15 year old white girl was admitted in December, 1964 with azotemia due to chronic glomerulonephritis proven by biopsy of both kidneys. Splenectomy and left nephrectomy were done 1 month after admission. Three weeks later a right nephrectomy and renal transplantation were done, but this graft was rejected several weeks later. No arteriographic study was done.

A second left cadaver kidney was transplanted to the left side. Following surgery the blood pressure, originally 160/120 mm. Hg, gradually decreased to 120/80 mm. Hg within 3 weeks. Three months following the second implantation the patient again developed a hypertension of 170/130 mm. Hg. A bruit was felt over the transplanted kidney. Renal arteriography performed through a catheter in the right femoral artery with the tip at the bifurcation of the aorta demonstrated marked stenosis at the anastomosis. The intrarenal vessels and blood flow were normal. The nephrogram was intense. The kidney was moderately swollen (Fig. 7, *A–C*).

Resection of the stenotic segment was done. The blood pressure decreased to 120/80 mm. Hg. Histologic examination of the anastomosis showed intimal hyperplasia and fibrosis which was not felt to be due to rejection. A biopsy of the transplanted kidney demonstrated a mild rejection. An arteriogram 2 weeks following this surgery demonstrated an intact, widely patent anastomosis with widely spread peripheral renal vessels due to edema and an intense nephrogram (Fig. 8, *A* and *B*). An $I^{131}$ hippuran renogram the following day was normal.

CASE 5. This 40 year old white woman had uremia secondary to gouty nephropathy. On March 25, 1965 she underwent bilateral nephrectomy, splenectomy and renal transplantation. The left kidney of an 8 year old girl, which had been resected during a ventricular-ureteral shunt for hydrocephalus, was transplanted into the patient's right iliac fossa.

The following day an $I^{131}$ hippuran renogram demonstrated no uptake in the kidney compatible with major vascular occlusion. Renal arteriography was performed by manual retrograde injection of 50 cc. of 60 per cent renografin through a percutaneous needle in the right femoral artery (Fig. 9, *A* and *B*). The anastomosis and large renal vessels were intact. However, contrast material still filled the major renal vessels at the end of 14 seconds. The smaller vessels showed a corkscrew pattern suggesting loss of renal parenchyma. The kidney was small. No nephrogram was apparent at any time nor was any collecting system seen. Immediately following arteriography a dark mottled kidney which was thought to be infarcted was removed. Pathologic examination showed no thrombosis in the large renal vessels.

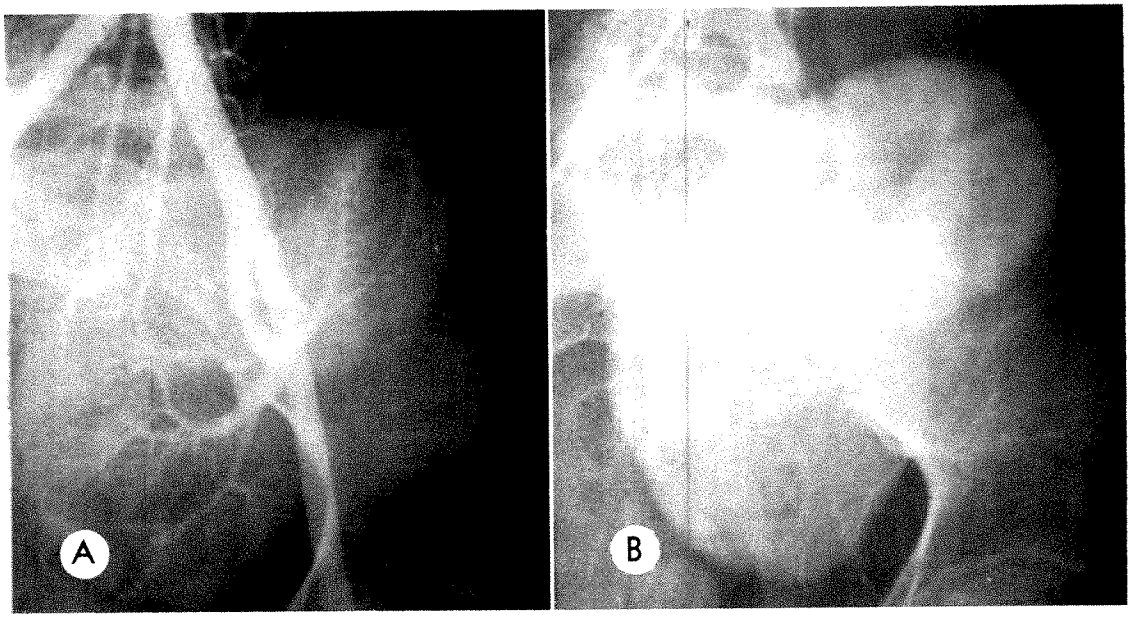



FIG. 8. Case 4. *June 28, 1965*: Postoperative renal arteriogram. (*A*) The anastomosis is patent. (*B*) The kidney is still edematous and the nephrogram is intense.

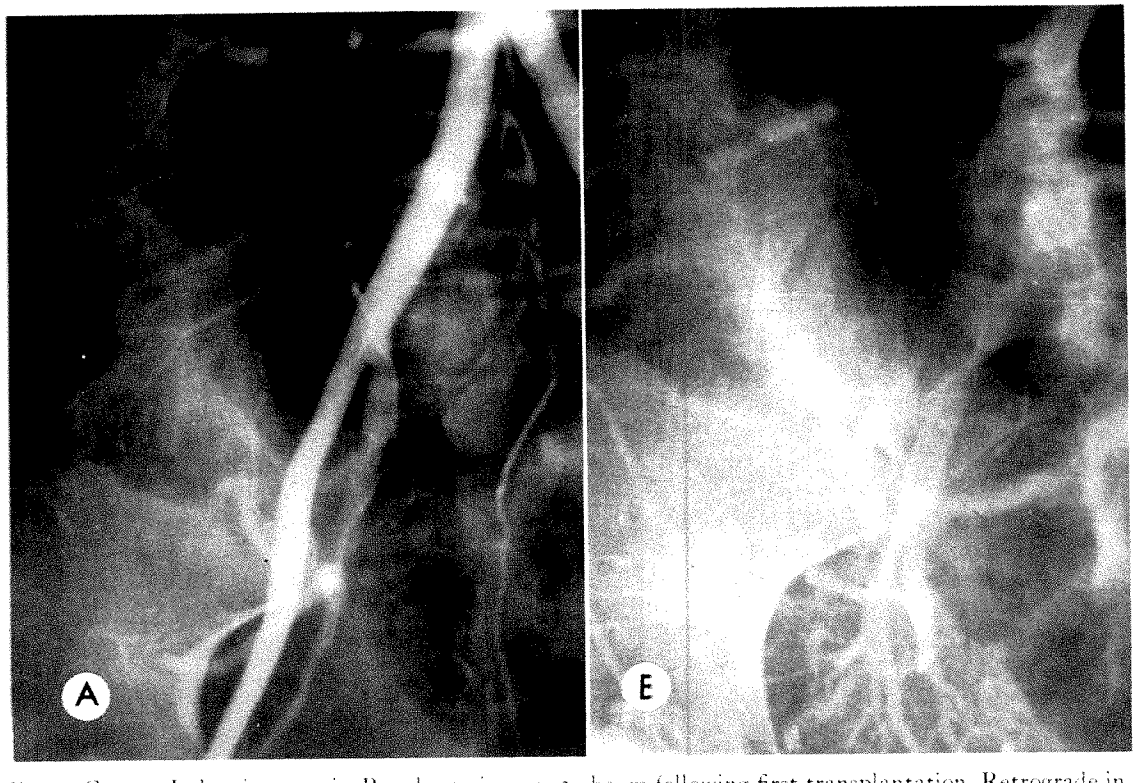



FIG. 9. Case 5. Ischemic necrosis. Renal arteriogram 24 hours following first transplantation. Retrograde injection into the right common femoral artery. (*A*) The anastomosis between the hypogastric and renal arteries is intact. (*B*) After 9 seconds the major renal vessels are still opacified. The smaller renal vessels are tortuous and the kidney is small. No nephrogram or renal collecting system was seen.

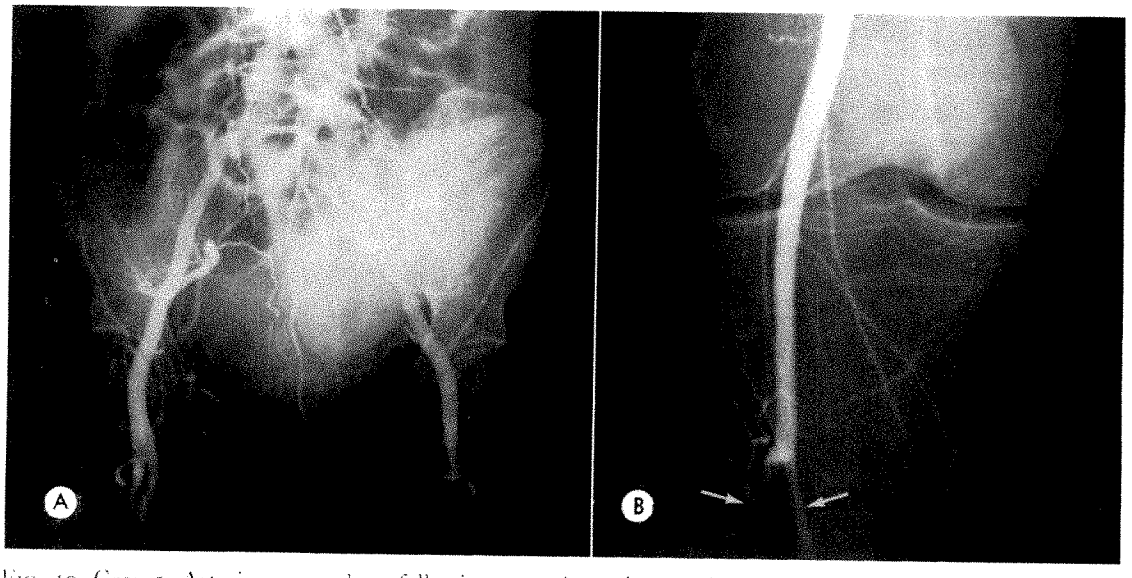



FIG. 10. Case 5. Arteriogram 4 days following second renal transplantation. Pressure injection through a Quinton A-V shunt in the right femoral artery. (*A*) The nephrogram is intense and the kidney is enlarged. (*B*) An embolus straddles the bifurcation of the popliteal artery.

The cortex was markedly decreased in size and microscopically showed a variegated pattern of coagulative necrosis thought to be secondary to prolonged renal ischemia. The pelvis and ureter were unremarkable.

Five months later a second transplantation was done on the left side using a cadaver kidney. The urine output was 2,300 cc. per day initially but decreased to 590 cc. per 24 hours on the fifth postoperative day. A renogram showed a markedly subnormal vascular and secretory phase without an excretory phase suggesting ureteral obstruction or parenchymal disease. Renal arteriography was attempted by injecting contrast material through the arterial arm of a Quinton hemodialysis shunt inserted into the right common femoral artery and vein. It was intended originally to film only the abdomen using a rapid film changer. However, in the process of preparing the shunt a clot was dislodged and the patient noted pain behind the knee suggesting embolism to the popliteal artery. In order to obtain roentgenograms of both the renal transplant and the lower extremities the examination was made with the patient on an RIL arteriographic table. Using a mechanical injector, 50 cc. 60 per cent renografin was injected into the arterial side of the Quinton A-V shunt into the right femoral artery. An error in timing prevented adequate visualization of the anastomosis but a very dense nephrogram was obtained (Fig. 10*A*). Stenosis of the renal artery could not be excluded. Follow up roentgenograms showed no contrast material in the renal pelvis. An embolus in the popliteal artery partially obstructed the posterior and anterior tibial arteries (Fig. 10*B*). The embolus cleared spontaneously over the following week. Attempts at retrograde pyelography were unsuccessful. A cystogram showed a defect at the site of insertion of the donor ureter without extravasation of contrast material (Fig. 11). About 2 months later the renal function deteriorated. Terminally the patient developed generalized cytomegalic virus infection and died of uncontrolled gastrointestinal bleeding.

Autopsy revealed a pale looking swollen transplanted kidney. Organized thrombi were found in the renal vein. The renal artery anastomosis was patent. Microscopically numerous tubular casts, cells and slight mononuclear cell infiltration of the interstitial tissues were seen.

CASE 6. This 35 year old white man had uremia secondary to bilateral renal vein thrombosis.[6] On November 6, 1965 a cadaver kidney was transplanted into the left iliac fossa. Three hours after transplantation there was no urinary excretion and on the first postoperative day this was only minimal. On the third postoperative day an $I^{131}$ hippuran renogram suggested renal artery occlusion or rejection reaction.

Arteriography on the following day demonstrated a patent anastomosis (Fig. 12, *A–C*). There was a considerable delay in the flow and persistence of contrast material in the smaller renal arteries 17 seconds after the beginning of the injection. No nephrogram was apparent and no collecting system was seen. This was interpreted as being compatible with rejection reaction or renal vein thrombosis. Surgical exploration was postponed.

Repeat arteriography 3 weeks later, following increased dosage of immune suppressive drugs, demonstrated interval increase in flow (Fig. 13, *A–C*). The contrast material was completely washed out of the renal vessels by the end of $3\frac{1}{2}$ seconds. A very faint nephrogram was apparent. There were no collateral vessels. The findings were consistent with renal parenchymal disease due to rejection reaction. The urinary output continued to improve and was 1,500 cc. per 24 hours by January 9, 1966.

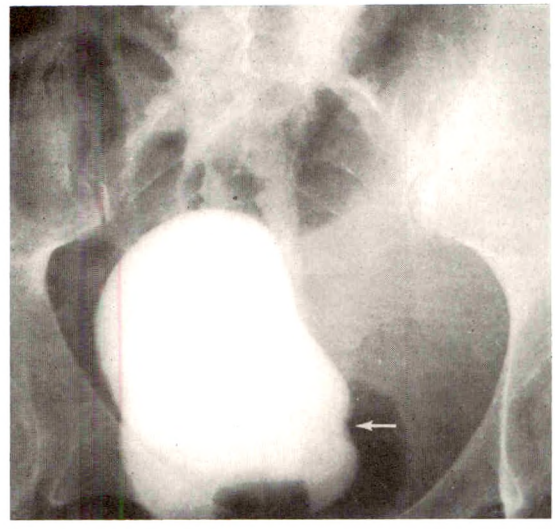


Fig. 11. Case 5. Cystogram showing site of implantation of the donor ureter (arrow). The transplanted kidney impresses on the left side of the bladder.

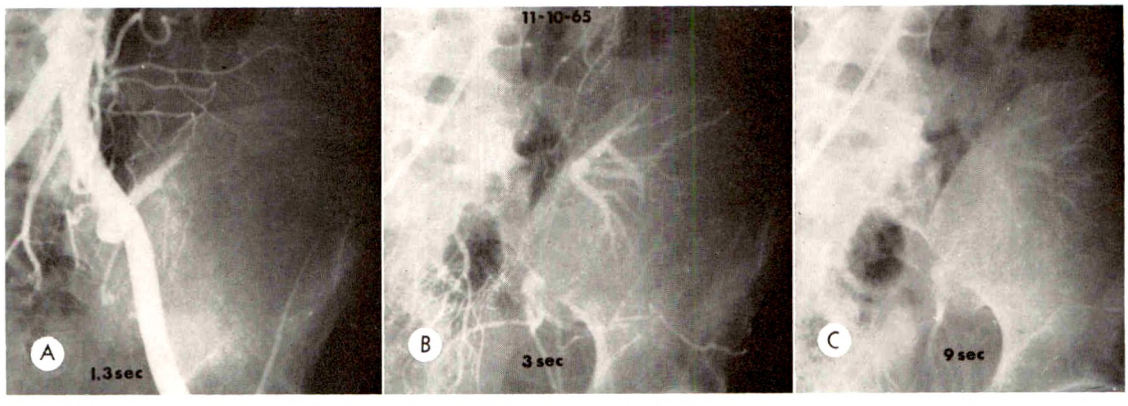



Fig. 12. Case 6. Rejection reversal. (*A–C*) Arteriograms on November 10, 1965, show marked delay in passage of contrast material. The smaller branches are visible but no nephrogram was seen. The kidney is enlarged.

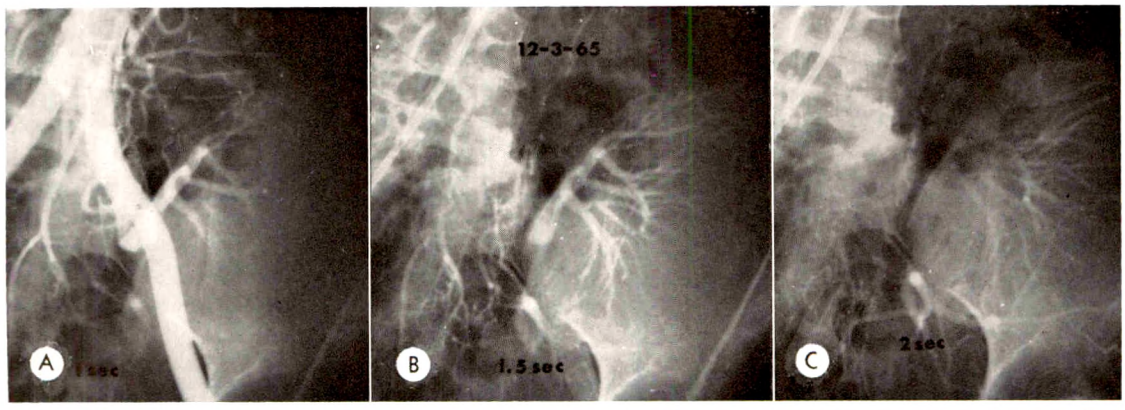



Fig. 13. Case 6. Rejection reversal. (*A–C*) Two weeks later, following immune suppressive therapy, flow has markedly increased. Contrast material is washed out after 3 seconds and a faint nephrogram was apparent.

## RESULTS

The results of the examinations are summarized in Table 1. Of 10 arteriographic studies performed on 6 patients, 6 showed evidence of parenchymal disease consistent with rejection phenomenon and this was verified histologically in 4 patients. A fifth patient (Case 6) probably also had rejection as evidenced by the remarkable improvement in flow following immune suppressive therapy. One patient showed histologic evidence of the effects of prolonged ischemia and another transplanted kidney proved to be involved with arteriolar nephrosclerosis. The 4 instances of vascular occlusion were proven at surgery or postmortem examination. One patient who had an intense nephrogram had organized thrombosis of the renal vein at autopsy 3 months later. The other "normal" examination was made following repair of stenosis of the anastomotic site of transplantation.

## DISCUSSION

Angiography following renal transplantation within the limits of this brief experience has its greatest value in assessing the status of the larger renal vessels, particularly to establish the presence of stenosis or occlusion as the cause of hypertension or oliguria. When stenosis of the renal artery is due to postsurgical fibrosis it is usually localized to a small segment at the anastomotic site (Fig. 7*A*). Narrowing of the renal artery due to the rejection phenomenon is the end result of decrease in size of the parenchymal vessels and progression of this process into the renal artery. This process stops at the anastomotic site where the recipient's hypogastric artery assumes its normal size (Fig. 5).[5,12]

Occlusion of the renal artery or larger branches can be easily diagnosed roentgenographically. Occlusion of a smaller branch artery may not be readily identified in those patients undergoing rejection reaction when there is a poor nephrogram. The avascular parenchyma of the infarcted area would not be visible during the nephrographic phase. Comparison with an arteriogram of the donor kidney prior to transplantation, if available, can be made to determine the number and positions of the vessels within the transplanted kidney. If some of these vessels are missing, the diagnosis of segmental arterial stenosis can be made. If the kidney is reversed during the transplantation it is important to reverse the original study when comparing these two examinations.

The $I^{131}$ renogram cannot accurately differentiate between vascular stenosis and the rejection phenomenon, because severe renal disease will cause marked delay in flow simulating occlusion. The anatomic delineation of the renal vessels by arteriography will readily differentiate these two findings. Others have also commented upon the difficulty of differentiating between renal parenchymal disease (rejection and acute tubular necrosis) and arterial stenosis or occlusion by indirect means. If vascular occlusion is excluded the status of the kidney may then be followed by radioisotope and biochemical studies.[4]

Differential diagnosis of disease processes which affect the parenchyma of the transplanted kidney is much more difficult. Transplant rejection may be seen in varying degrees of severity. Initially the kidney is edematous. This may be followed by loss of the nephrogram and decrease in the rate of flow through the renal parenchyma. With further increase in severity the smaller renal arterial branches are obliterated and the larger branches become narrow and irregular due to subintimal proliferation. Reversibility of the less severe changes may have been demonstrated in one of our patients and has been demonstrated clinically in other series.[2,11,15,16]

The arteriographic patterns in arteriolar nephrosclerosis and acute glomerulonephritis appear to be similar to those of rejection.[5] The transplanted kidney in one of our patients was affected by arteriolar nephrosclerosis without rejection reaction and this could not be differentiated from the rejection reaction on roentgenographic

grounds alone. In both instances the nephrogram is poor and the branch vessels and renal artery show diffuse narrowing and irregularity. Acute glomerulonephritis has been reported to occur in the transplanted kidney and must also be considered when there is decrease in vascularity and in renal flow. The criteria of Friedenberg *et al.*[7] cannot be applied to differentiate pyelonephritis, glomerulonephritis, and arteriolar nephrosclerosis from graft rejection since the findings are dependent upon bilaterality of involvement and the nephrogram. The presence of one kidney and a poor or absent nephrogram in the patient with moderate to severe graft rejection decreases the possibility of accurate diagnosis on the basis of arteriography alone. Attenuation of vessels may be seen in all three diseases and the graft rejection. Size of the kidney cannot be used as a criterion since a small contracted diseased kidney would not be transplanted and a less severely involved kidney would increase in size under most circumstances following transplantation. In the patient with hydronephrosis of long standing the vessels may decrease in size and be widely spread as in a rejection phenomenon.[13] History and retrograde pyelography can readily differentiate these findings.

Prolonged ischemia of the cadaver kidney before implantation leads to marked cortical necrosis within the first 24 hours after surgery. The angiographic appearance may therefore show a very small kidney with corkscrew like parenchymal vessels and a severe delay in washout due to necrosis of the smaller vessels.

Renal vein thrombosis may be difficult to differentiate from rejection and other parenchymal diseases arteriographically. Demonstration of collateral venous drainage establishes the diagnosis but the transplanted kidney may not develop any collateral veins especially in the early postoperative period if at all. The patient in Case 5 had renal vein thrombosis but it cannot be stated with certainty that this existed at the time of the arteriography. In Case 6 the marked delay in flow may have been due to renal vein thrombosis or rejection. Failure to demonstrate any collateral venous drainage and the marked improvement following immune suppressive drug therapy suggests that the rejection phenomenon was most likely the cause of this patient's renal angiographic changes.

Transmission of disease through the donor kidney has been reported. Metastatic carcinoma of the breast from a donor patient has been seen in the donor kidney after implantation into the recipient.[8] Tumor in the liver and transplanted kidney in a recipient who received a renal homograft from a patient who died of squamous cell carcinoma of the pyriform sinus has been reported.[9] Premortem discovery of these lesions may theoretically be possible and should be looked for.

The arteriography can be performed in a variety of ways. A retrograde injection through a needle in the femoral artery on the side of implantation or the opposite side may be made. A catheter placed at the aortic bifurcation has produced equally adequate results. In one patient a Quinton A-V shunt was used as the mode of delivery of the contrast material. There have been no serious complications following the procedures in the patients studied to date, although in one patient a clot was dislodged from a Quinton A-V shunt in preparation for the procedure.

Selective catheterization of the transplanted renal artery and vein has been shown to be feasible. The smaller renal vessels are more readily visualized and less contrast material is used. Catheterization of the renal vein is the only means of adequately determining the patency of this vessel. Examination of the renal artery by selective catheterization is in our opinion the method of choice. If persistent arterial opacification is noted then a selective renal vein study should also be made.[1]

Arteriography is of value to determine the intactness of the vascular anastomosis and the patency of the involved vessels. Parenchymal changes due to rejection

phenomenon, severe necrosis, arteriolar nephrosclerosis and acute glomerulonephritis may be evaluated, although difficult to differentiate. The effectiveness of immune therapy may be followed and occasionally the presence of transmitted disease may be discovered. We have found the renal arteriography to be a readily performable useful procedure without serious complication following renal transplantation.

SUMMARY

Ten arteriographic studies were carried out on 6 patients following renal transplantations. These studies demonstrated varying types of post-transplantation complications including anastomotic site stenosis, rejection phenomenon, arteriolar nephrosclerosis and compression secondary to hemorrhage. Arteriography has its greatest value in evaluating the intactness of the renal vascular system and in establishing the presence of renal parenchymal disease, although not necessarily differentiating the various types.

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# EXPERIMENTAL RENAL GLOMERULOGRAPHY*

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IN PREVIOUS studies, techniques were developed for the angiographic demonstration of the renal interlobular arteries and veins in the dog.[10,11] The toxicity of the methods used was also assessed. On some of the angiograms obtained in these studies at 2× direct roentgenographic magnification, myriad tiny, round, discrete, cortically-distributed shadows were noted early in the so-called "nephrographic phase" of renal opacification. It was postulated that these might represent renal glomeruli. When more refined radiographic equipment became available, which made 3× magnification practical, it was decided to explore this theory. After considerable trial and error, the following technique was evolved.

## METHOD

Mongrel dogs, between 12 and 25 kg. in body weight were anesthetized with the intravenous administration of pentobarbital sodium, 25 mg. per kg. of body weight. The trachea was intubated. The animal was fastened in the supine position onto a radiolucent moveable X-ray table top. Both femoral arteries were exposed below the inguinal ligament. One femoral artery was encannulated with a red Ödman* catheter, the curved tip of which was passed into a renal artery under fluoroscopic guidance, taking care not to wedge the tip in the main or an interlobar artery. This catheter was attached to a Statham strain gauge pressure transducer, and also to a pressure injector† by means of T-connection, to allow continuous monitoring of pressures, even during injection of contrast medium. Through the opposite femoral artery, a catheter equipped with an inflatable balloon was passed to the level of the catheterized renal artery, so that when the balloon was inflated, the renal artery would be occluded, except for the red catheter lodged in its lumen (Fig. 1).

The table-top bearing the animal was moved over the roentgen tube (a Siemens Biangulix‡) which was mounted on a low platform on the floor, and, by means of adjustable supports at either end of the table-top, the distance from the anode to the animal's kidneys was fixed at 13 inches, and from the anode to the X-ray film cassette, held in an over-head support, at 40 inches. This gave 3× direct roentgenographic magnification. The general arrangement is shown in Figure 2. Radelin ultra-detail intensifying screens,§ and fine-grain Ilford X-ray film were found to give the best results, but many studies were also done using Ansco par-speed films, and par-speed screens. Test films were first exposed for position and technique, using the 0.3 mm. focal spot of the Siemens roentgen tube, at 100 ma., approximately 64 to 76 kv., and 0.12 to 0.24 sec. During each exposure, the animals' lungs were held inflated with oxygen at +20 cm. $H_2O$ pressure to forestall movement of the kidneys caused by breathing. If the test films were satisfactory, the angiogram was made during inflation of the balloon catheter in the aorta (with 4 ml. of air). An injection of 5 ml. sodium iothalamate 66.8 (conray-400)‖ was made through the red Ödman catheter, directly into the renal artery, at a pressure of 20 lb. per square inch, using the mechanical injector. The exposure was usually made 2 seconds following the injection. Renal arterial

* Odman-Ledin (I.D. 1.2 mm.; O.D. 2.2 mm.).
† Zimmerman injector (Pemco Corp., Brecksville, Ohio).
‡ Siemens Medical of America, Inc., Union, New Jersey.
§ United States Radium Corp., Morristown, New Jersey.
‖ Mallinckrodt Pharmaceuticals, St. Louis, Missouri.

* From the Surgical, Research, and Radiology Services, Veterans Administration Hospital, Oteen, North Carolina.
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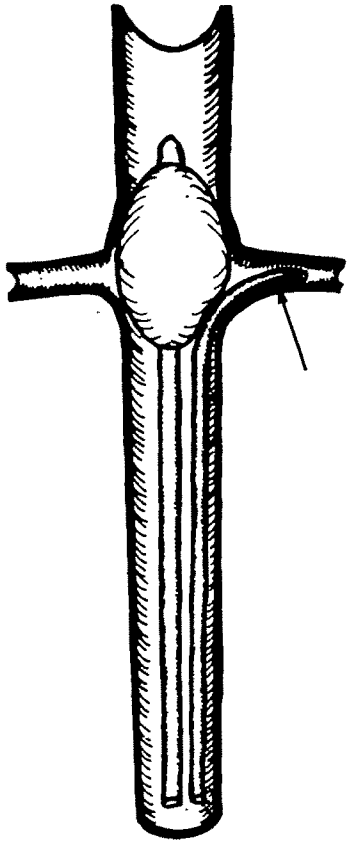


FIG. 1. Diagram showing arrangement of catheters for renal glomerulography: curved-tip polyethylene catheter in renal artery (arrow) through which contrast medium is injected during momentary occlusion of the aorta at renal arterial orifice by balloon catheter.

pressure, and the time interval from the beginning of the injection of contrast medium to the moment of exposure were recorded on an Electronics for Medicine recorder, with recording begun before inflation of the intra-aortic balloon, and continued after deflation of the balloon following the exposure.

Blood for urea nitrogen determination was drawn before angiography, the day after angiography, and once a week for 4 weeks thereafter. In 9 animals, $I^{131}$ renograms were obtained by a method previously described, immediately after, and 24–48 hours after angiography.[10] The animals were sacrificed 4 to 6 weeks after angiography. The kidneys were removed, sectioned longitudinally in the coronal plane, and fixed in formalin. Paraffin sections were prepared and stained with hematoxylin and eosin, and examined.

In 5 animals, in addition to the procedures above, 5 ml. of ethiodol* was injected into the renal artery, and a second exposure was made 30 to 60 seconds after the injection. These animals were sacrificed immediately after the study and the kidneys were removed and sectioned in the same manner. Frozen sections were prepared for fat stains with Sudan III, and also with Oil Red O and silver nitrate. Microangiograms of the injected kidney halves were prepared on industrial X-ray film using a portable industrial X-ray unit, the tube head of which was equipped with a beryllium window. The technique described by Rubin was followed.[8] The resulting roentgenograms were enlarged photographically to the same size as the angiograms obtained *in vivo*.

## RESULTS

Studies using the technique described were carried out in 15 animals. Angiograms obtained in the above manner showed, at this magnification, multiple tiny, discrete, round shadows approximately 1 mm. in diameter, arranged preferentially in the

* Brand of Ethiodized Oil, E. Fougera & Co., Inc., Hicksville, N. Y.

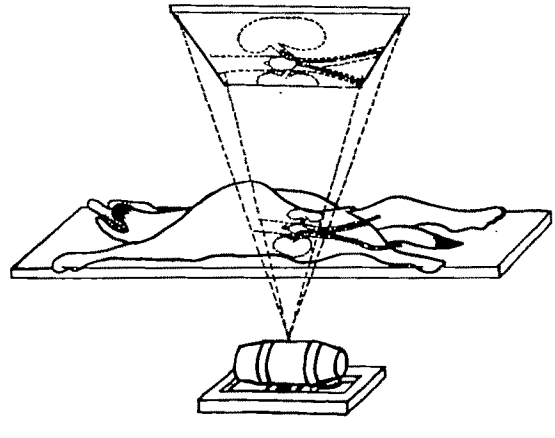


FIG. 2. Diagram of general arrangement of roentgen tube, subject, and cassette.

cortex, and roughly parallel to the interlobular arteries (Fig. 3*B*). On angiograms exposed earlier than 2 seconds following beginning of the injection, these shadows were usually not seen (Fig. 3*A*), while on angiograms exposed later (2.5–3 sec.) the rounded, opacified granules occupied the entire kidney shadow (Fig. 3*C*).

On the ethiodol angiograms, similar-sized and similarly arranged shadows could be seen, which, however, were less radiopaque than those on angiograms performed using sodium iothalamate. However, on microradiograms, these discrete structures were well visualized (Fig. 4, *A* and *B*) and arranged radially in the cortex as glomeruli. On fat stains of ethiodol-injected kidneys, many glomeruli were found to be stuffed with fat-staining material, which was not seen in other blood vessels (Fig. 5); fat-staining of the loops of Henle was prominent in some kidneys, but this was observed also in kidneys which had not been injected with ethiodol, and was easily distinguishable from the iodized oil by silver nitrate stains.[2] It was concluded that the opacified structures seen on renal angiograms were glomeruli, and renal angiograms obtained in this particular phase of renal vascular opacification were termed renal glomerulograms, to distinguish them from renal arteriograms, phlebograms, and "nephrograms."

No animals succumbed as a result of this study. The results of blood urea nitrogen determinations are summarized in

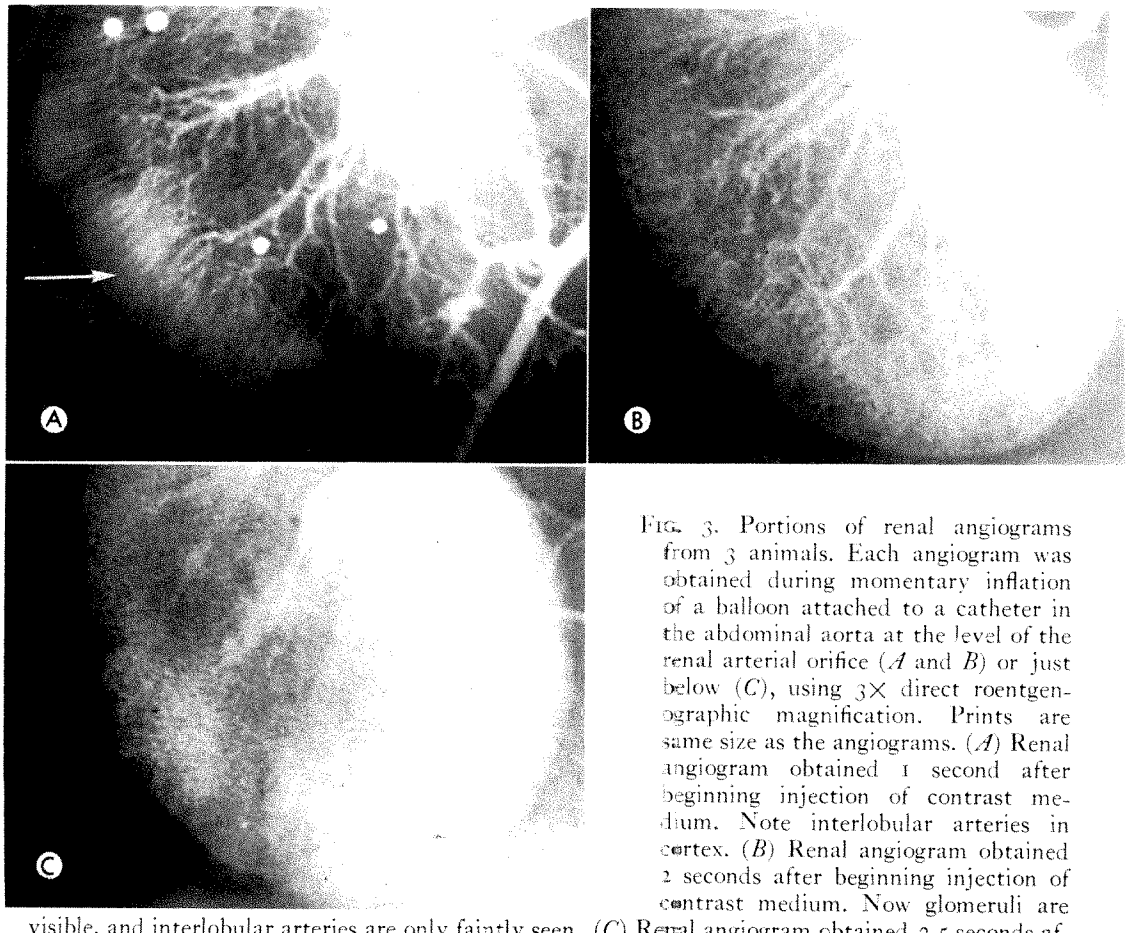



Fig. 3. Portions of renal angiograms from 3 animals. Each angiogram was obtained during momentary inflation of a balloon attached to a catheter in the abdominal aorta at the level of the renal arterial orifice (*A* and *B*) or just below (*C*), using 3× direct roentgenographic magnification. Prints are same size as the angiograms. (*A*) Renal angiogram obtained 1 second after beginning injection of contrast medium. Note interlobular arteries in cortex. (*B*) Renal angiogram obtained 2 seconds after beginning injection of contrast medium. Now glomeruli are visible, and interlobular arteries are only faintly seen. (*C*) Renal angiogram obtained 2.5 seconds after beginning injection of contrast medium. Glomerular shadows more intense and confluent.

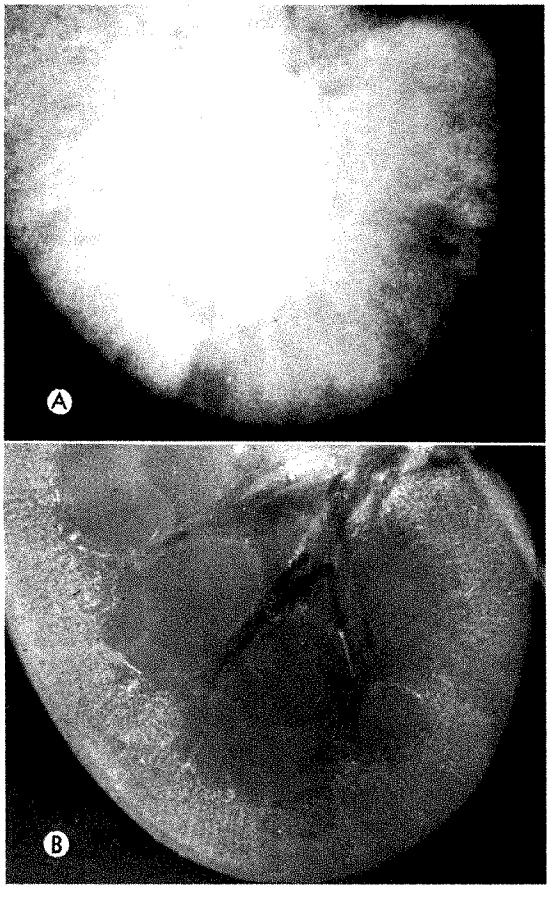



FIG. 4. (*A*) Microradiogram of ethiodol-injected half-kidney slice, photographically enlarged to approximately same size as kidneys in Figure 3. Note myriad minute radiopaque granules, much more sharply delineated because specimen is in direct contact with the film. (*B*) Microradiogram of 3 mm. slice of kidney in *A*. Note arrangement of radiopaque granules in cortex.

Figure 6. In no case did the level of blood urea nitrogen rise above 25 mg. per cent. This includes 6 animals in which several glomerulograms were obtained bilaterally. $I^{131}$ renograms in 9 animals showed slight evidence of altered renal function immediately after angiography, and none 24 hours later (Fig. 7).

Histologic sections using routine stains showed cloudy swelling and mild hydropic degeneration of the proximal and distal convoluted tubules in some animals. It was concluded that the method was reasonably nontoxic to the kidney in spite of repeated injections of radiopaque material (up to 4 per kidney).

## DISCUSSION

Kaneko,[4] working in Takahashi's laboratory, first succeeded in visualizing renal glomeruli in both animals and man, using a special X-ray tube with an autobiased focal spot 0.05 mm. in diameter, and 4× magnification. Confirmation was sought by injecting barium after removing the kidneys, and exposing roentgen films *in vitro*. Interlobular arteries, with accompanying glomeruli, as well as arteriolae rectae were demonstrated clearly *in vitro*, but not so convincingly on the reproductions of the angiograms obtained *in vivo*.

Since we were limited to a single magnified film by the equipment we used, timing of the exposure to coincide with the glomerular phase of opacification was of utmost importance. This limitation led us to use a balloon catheter in the aorta to arrest renal arterial circulation for 4 to 5 seconds while the contrast medium was injected and the exposure made. This brief circulatory stasis produced a temporarily high concentration of contrast medium in the glomeruli, which allowed these tiny structures to be seen quite clearly. The pressure of the injection may also have caused a momentary increase in

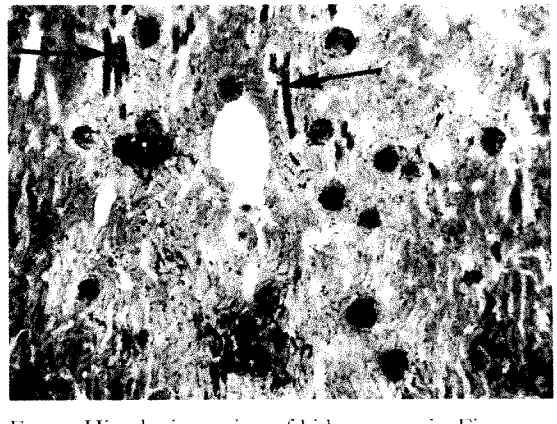


FIG. 5. Histologic section of kidney seen in Figure 4. Note black-staining glomeruli filled with ethiodized oil, in contrast to neutral fat in tubules nearby (arrows). Oil red O and silver nitrate stain, ×25.

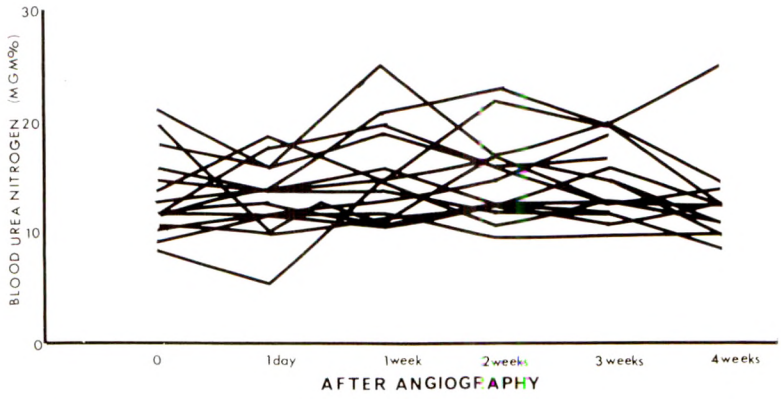



Fig. 6. Graphic representation of blood urea nitrogen levels in 15 animals before angiography (0) and from 1 day to 4 weeks after angiography.

the diameter of the glomeruli, as a result of momentary loss of tone of the afferent arteriole during renal arterial occlusion. In any event, this brief arterial stasis did not seem to alter seriously the crude measures of renal function we employed, or the histologic appearance of the renal parenchyma as observed following sacrifice 4 weeks later. However, more refined studies of the toxicity of the method must be carried out before it can be deemed to be completely or even reasonably safe. A study to this end is in progress.

Ethiodol was selected to confirm the identity of the tiny, discrete spherules seen on angiography, because of the knowledge that iodized vegetable oils such as lipiodol and ethiodol are arrested in capillaries, at least for a time.[4] This gave us the opportunity to obtain an angiogram *in vivo*, following the injection of ethiodol, as well as a microradiogram and histologic sections stained with fat to identify the ethiodized oil in the glomeruli. These 3 together provided firm evidence of glomerular opacification *in vivo*.

To the 3 phases of renal vascular opacification thus far recognized might therefore be added a fourth: the glomerulogram. Until now, this has been submerged as one of three subphases of the nephrographic phase of the renal angiogram.[6] Since it is clearly separable by the technique described, and precedes the opacification due to accumulation of contrast medium in the cortical tubular epithelium, and the tubular spaces, it should not be considered as part of the nephrogram. Judging from the work of Daniel *et al.*[1] using thorotrast, a contrast medium not excreted by the kidney, and from serial magnified angiograms of our own, performed without renal arterial occlusion, the duration of glomerular opacification in the dog must be 2 seconds or less.

Canine glomeruli ordinarily measure approximately 200 to 300 microns in diameter after death of the animal, fixation

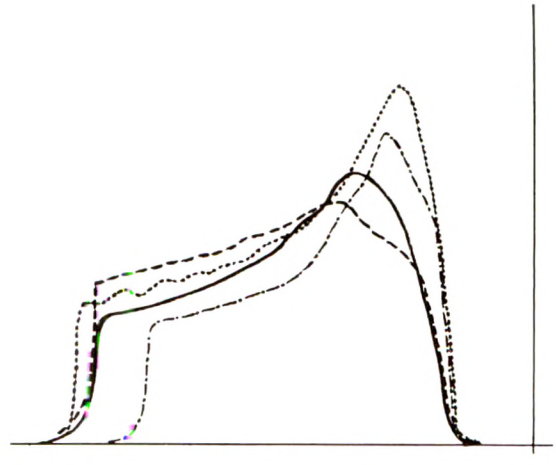


Fig. 7. $I^{131}$ renogram obtained following renal glomerulography. *Solid line*: control renogram (prior to angiography). *Dashed line*: immediately after 2 angiograms. *Dotted line*: 24 hours after angiography. *Alternating dots and dashes*: 7 days after angiography.

of the tissues, and staining. It is possible that during life, or, as mentioned above, during momentary cessation of renal flow and injection of contrast medium under pressure, renal glomeruli assume diameters larger than those seen in the fixed state. This might account for the relatively good delineation of these structures, together with their penumbrae, with a focal spot rated at 0.3 mm. Under these circumstances, when the penumbra is added to the umbra of glomeruli, roentgen images up to 1 mm. in diameter might be expected, at 3× magnification. Such is the approximate size of the spherical images noted on angiograms obtained by the technique described. Variations in size may be due to overlapping of the images of several glomeruli to produce clusters. It is also possible that variations may be caused by the "loop rhythm" of glomerular capillary activity, postulated by Heggie,[3] whereby some capillary loops in each glomerulus are "resting" while others are conducting blood. Since afferent and efferent arterioles measure only 30 microns or less in diameter, it is not surprising that these structures were not visualized.[5] The interlobular arteries on the other hand are twice to three times as large, and some can be delineated, as we have shown.

The renal vascular architecture has been very thoroughly studied *in vitro* and many attempts to correlate morphologic changes with altered function have been made in the past. Except for pioneering work by Prichard and Daniel[7] on the exteriorized kidney, however, direct evidence of the behavior of the fine renal vessels *in vivo* is still meager and has had to be inferred for the most part, from studies of renal vascular resistance, glomerular filtration rate, etc., or from injections of indicator materials into the renal artery just before sacrifice of the animal. Thus, there are many questions regarding the cortical and medullary arterial circulation and the glomerular portion of the cortical circulation which remain unanswered. It is hoped that the addition of renal glomerulography to other methods of study will help resolve some of these questions. The prospects of using this method clinically as an aid in providing more accurate morphologic diagnostic information concerning the fine intrarenal vasculature are also good, and are being explored.

SUMMARY

An angiographic technique is described whereby the glomeruli of the canine kidney in the intact animal can be visualized consistently. The method involves the use of momentary renal circulatory arrest produced by inflating a balloon catheter in the abdominal aorta at the renal arterial orifice, with injection of radiopaque medium directly into the renal artery distal to the balloon through a second catheter. Fine grain film, ultra-detail intensifying screens, and direct roentgenographic magnification to 3 times actual size are also employed. Confirmation of the nature of the tiny round discrete shadows distributed radially and preferentially in the cortex was obtained by comparing the angiograms with those obtained using ethiodol as the contrast medium. The distribution of the ethiodol both by microradiography and histology using fat stains was almost entirely in the glomeruli. The toxicity of the method was cursorily assessed by blood urea nitrogen determinations, $I^{131}$ renograms and histology, and was found to be low.

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# SPECIFIC NEPHROCALCINOSIS OF CHRONIC GLOMERULONEPHRITIS

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PARENCHYMAL calcification is detectable in most, if not all, kidneys whether normal or pathologic. The majority of these are microscopic but in some patients the calcifications are roentgenographically demonstrable. Renal tubular calcification of such magnitude (excluding dystrophic renal calcification of neoplasms and tuberculosis) is termed nephrocalcinosis. The name was coined by Albright *et al.*[1] in 1934 and at that time the condition was thought to be diagnostic of hyperparathyroidism. One year later Lightwood[10] provided autopsy evidence of renal parenchymal calcifications in a patient with normal parathyroid glands, indicating that hyperparathyroidism was not the sole cause of nephrocalcinosis. Since then the following disorders have also been implicated: hyperchloremic acidosis, medullary sponge kidney, chronic pyelonephritis, vitamin D intoxication, sarcoidosis, idiopathic hypercalciuria, chronic glomerulonephritis and carcinomatosis.

Of the causes enumerated above, chronic glomerulonephritis is one of the least frequent.

## HISTORY AND INCIDENCE

A single case was reported in 1947 by Vaughan *et al.*[15] Geraci and his associates[7] described an essentially identical patient in 1950. Mortensen *et al.*[12] found 6 instances of nephrocalcinosis of which 1 was associated with chronic glomerulonephritis. Arons *et al.*[2] added 4 cases to the literature in 1955.

It has been estimated that chronic glomerulonephritis accounts for 3 per cent of cases of nephrocalcinosis.[14] However, the true incidence is quite uncertain. The paucity of reported cases is probably not a valid reflection of its true incidence. Undoubtedly there are many cases not reported and even more that are not appreciated as examples of this entity.

The total available data on nephrocalcinosis, in general, have never been suitably collected or classified.

The current report adds 2 cases to the 7 cited above.

## REPORT OF CASES

CASE 1. H.C., white male, age 32. Admitted on January 24, 1964 with clinical and laboratory evidence of uremia. Blood urea nitrogen on admission was 226 mg. per cent and the blood pressure ranged from 190/140 to 190/130.

Pertinent previous history revealed scarlet fever at 10 years of age.

Clinical diagnosis of chronic glomeruloneph-

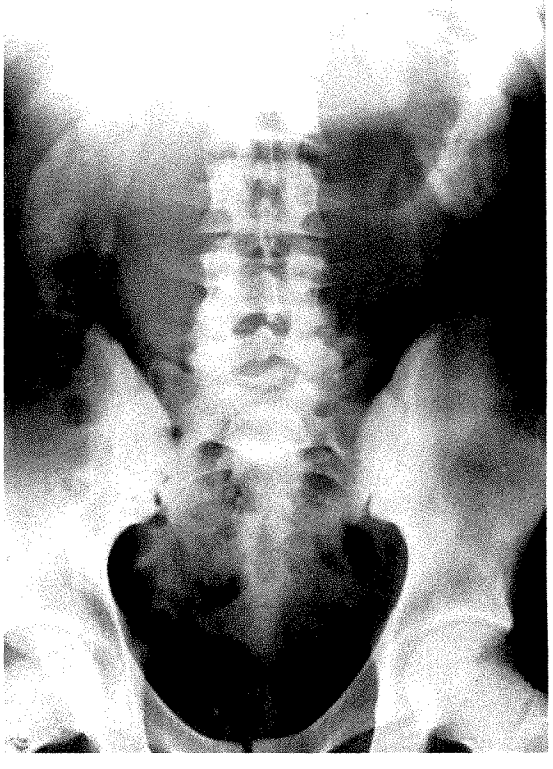


FIG. 1. Case 1. There is an increase in renal density caused by the granular calcifications in the parenchyma. Note small regular renal outlines.

ritis was made in 1958. A plain roentgenogram of the abdomen showed small kidneys containing many granular calcifications in cortical areas (Fig. 1). Retrograde pyelography revealed a normal appearance of the collecting tracts (Fig. 2).

There were 3 short term admissions for peritoneal dialysis early in 1964. In March 1964 adhesions prevented further dialysis from being accomplished. A Barry plastic cannula was inserted and dialysis re-established.

Three days later he was sent to New York City where, with his mother acting as donor, a renal transplantation was performed. There was no urinary output during the first 24 hours; this was attributed to rejection phenomenon. Despite many heroic measures, including 6-mercaptopurine administration, irradiation, capsulotomy and corticosteroids, the patient succumbed. The postmortem study confirmed the diagnosis of chronic glomerulonephritis (Fig. 3).

Case II. M.C.T., Negro male, age 47. Entered with complaint of recurrent headaches that had become progressively more severe during the past 3 years.

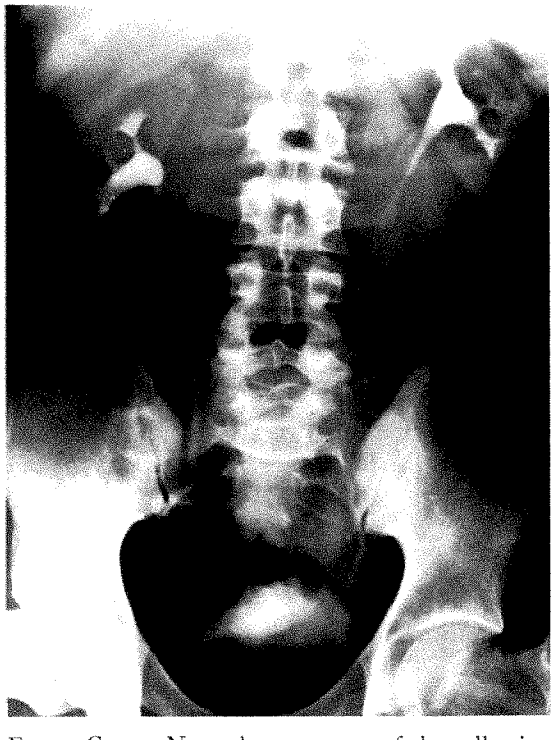


Fig. 2. Case I. Normal appearance of the collecting systems demonstrated by retrograde pyelography.

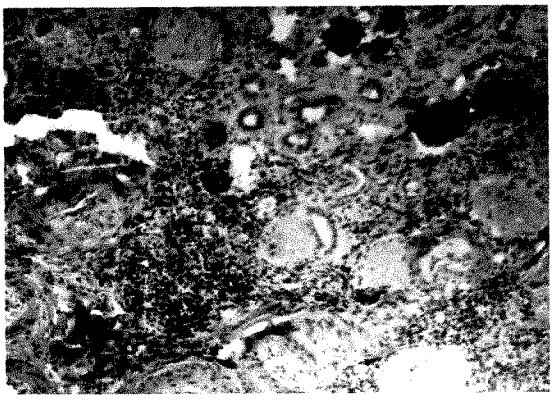


Fig. 3 Case I. Hyalinization of glomeruli. Note extensive calcification in tubules.

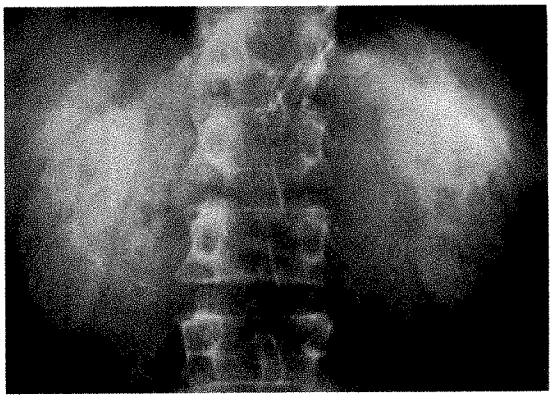


Fig. 4. Case II. Findings essentially identical to those in Case I. There are innumerable granular calcifications that are primarily cortical in location.

Albuminuria was first detected in 1953 during a hospitalization for pneumonia.

In 1963 he first developed severe parietal and occipital headaches. The patient was hypertensive and a "chronic renal disease" was diagnosed.

During admission in August 1966 the blood pressure was 204/130, urine specific gravity 1.005, blood urea nitrogen 100 mg. per cent, 24 hour urine for proteins 4 gm. and creatinine 11.2 mg. per cent.

A plain roentgenogram of the abdomen revealed innumerable tiny punctate calcifications diffusely scattered throughout the renal parenchyma (Fig. 4). A roentgenologic diagnosis of chronic glomerulonephritis was offered.

The patient was placed on a 500 mg. sodium diet but the blood urea nitrogen rose to 141 mg. per cent. At this time he was placed on essential

amino acid diet containing 200 gm. protein and 800 mg. of sodium. The blood urea nitrogen then decreased and was 63 mg. per cent at the time of the discharge.

He was re-admitted in December 1966 complaining of headache and epistaxis. He expired on December 26, 1966 of uremia and acute pulmonary edema.

Chronic glomerulonephritis was found at autopsy.

## DISCUSSION

Ordinarily one is unable to establish an etiologic basis for renal lithiasis by roentgen methods. On the other hand, in the majority of instances, the radiologist can ascertain the cause of nephrocalcinosis when the roentgenologic and clinical features are correlated.

Hyperparathyroidism, hyperchloremic acidosis, chronic pyelonephritis, and medullary sponge kidneys account for approximately 80 per cent of all cases. Less common causes are idiopathic hypercalciuria, chronic glomerulonephritis, sarcoidosis, carcinomatosis and vitamin D intoxication.

Detailed roentgen features of the common causes of nephrocalcinosis have been presented elsewhere, but a brief review follows. In hyperparathyroidism renal lithiasis is much more common than renal calcinosis. However, when nephrocalcinosis occurs, it is manifest by millet seed densities confined to the medulla. Occasionally, larger pyramidal calcifications are present. Fine streaks along the ducts of Bellini are seen infrequently. Hellström[8] reviewed 70 cases of primary hyperparathyroidism; he feels that tubular calcification is a prerequisite to the formation of renal calculi, believing that the latter arise from extruded tubular debris. Similar calcifications are found in hyperchloremic acidosis. They may coalesce to form deposits of considerable size. As with hyperparathyroidism, the calcinosis may give rise to renal lithiasis. The calcifications of medullary sponge kidneys are small round or oval deposits in the papillary portions of the pyramids. The involvement may be unilateral or bilateral. Chronic pyelonephritis is associated with larger deposits and a more unequal distribution than is seen in the above conditions.

## FEATURES OF CHRONIC GLOMERULONEPHRITIS

Chronic glomerulonephritis is rarely accompanied by visible calcification. However, when present, its characteristics enable the radiologist to make a histologic diagnosis on a plain roentgenogram of the abdomen. In Case 11 the diagnosis was first suggested by the radiologist.

The kidneys retain a normal shape but are uniformly small, the longitudinal axis rarely exceeding 10 cm. The calcifications are granular, innumerable and diffusely scattered throughout the cortices. It is the cortical location that distinguishes this type of nephrocalcinosis from the others; no other condition presents such a predominantly cortical localization.

Microscopically, most of the calcium is in the lumina of the tubules. Some is in the epithelial lining cells of the loop of Henle, the distal convoluted tubules and the collecting tubules. Occasionally, calcification is present in cortical interstitial tissues and medullary collecting tubules. The glomeruli may be hyalinized but they do not contain calcium.

## SUMMARY

Nephrocalcinosis associated with chronic glomerulonephritis is rare. Only 7 documented cases have been reported in the English language literature. The detection of 2 cases in 2 years in a relatively small radiology practice indicates that it is considerably more common than the literature suggests.

The roentgen features are described and 2 additional cases are presented.

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# RENAL LYMPHOMA*

## ARTERIOGRAPHIC STUDIES

*By* RONALD A. SELTZER, M.D.,† *and* DALE E. WENLUND, M.D.‡
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ALTHOUGH the arteriographic appearance of most renal tumors is well documented, the findings in malignant lymphoma have not been described. Recently we had the opportunity to perform renal arteriography in a case of renal lymphoma.

### REPORT OF A CASE

A 42 year old housewife (MGH#1346659) with multiple sclerosis was admitted to the Massachusetts General Hospital because of fever, weakness, and anorexia of 7 months' duration. There had been a weight loss of 18 pounds. She had been treated with propylthiouracil for well documented hyperthyroidism for 7 months without relief of symptoms. Six weeks prior to admission she was found to have a blood hemoglobin of 8 gm. per 100 ml. for which she was transfused with 1,500 ml. of whole blood.

Physical examination revealed a thin woman with a few spider angiomata over the thorax. The temperature was 101° F. The blood pressure was 140/60. The liver was large and soft, and a mass was palpated in the left flank.

The hemoglobin was now 9.4 gm. per 100 ml. The white blood cell count was 10,000 cells per cu. mm. The urinalysis was normal except for an occasional red blood cell per high power field. The sedimentation rate was elevated. The serum protein was 6.5 gm. per 100 ml. Electrophoresis showed albumin 26 per cent, $\alpha_1$ globulin 10 per cent, $\alpha_2$ globulin 15 per cent, $\beta$ globulin 9 per cent, and $\gamma$ globulin 40 per cent. The blood urea nitrogen was 10 mg. per 100 ml. The serum alkaline phosphatase and transaminase were normal.

A chest roentgenogram was normal except for a small left pleural effusion. The intravenous urogram (Fig. 1) showed a large liver and a mass in the lower pole of the left kidney, which deformed the calyces without distinct invasion and which deviated the left ureter medially. A selective left renal arteriogram showed the major arteries to the lower pole of the left kidney to be displaced about a large mass (Fig. 2*A*). There was early filling of two small collections of large, irregular tumor vessels at the periphery of the mass from branches of the renal artery. Later in the arterial phase (Fig. 2*B*), a rich collection of small arteries was shown to originate at the proximal margin of the mass and course linearly toward its center. Upon close inspection, these vessels had irregular walls and were clearly tumor vessels, but they were remarkable because of their straight course in a palisade-like arrangement, ending gradually in a brush-like border. The capillary phase (Fig. 2*C*) showed irregular staining of the thick-walled, richly vascular periphery of the mass, an avascular central area, and an indistinct junction of the mass with normal renal

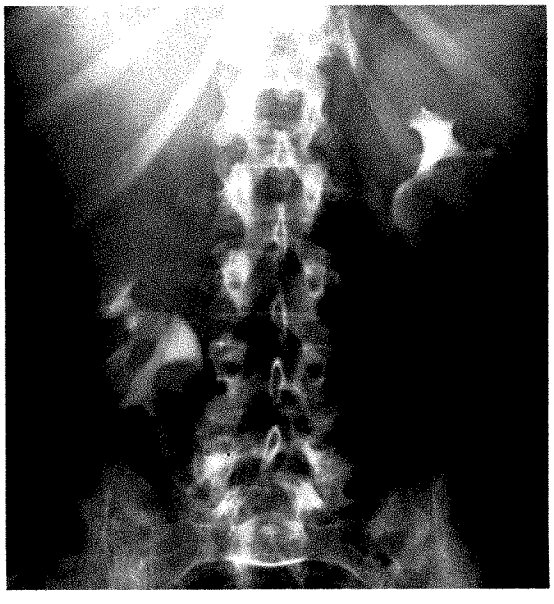


FIG. 1. Intravenous urogram. A mass in the lower pole of the left kidney elevates and distorts the calyces. The left ureter is deviated medially.

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parenchyma. A selective celiac arteriogram (Fig. 3) showed the hepatic arteries to be elongated and straightened within an enlarged liver, but neither tumor circulation nor displaced vessels were present. The capillary phase was normal.

At operation, a large mass was found to occupy the lower pole of the left kidney. The liver was enlarged but no nodules were seen. A left nephrectomy and liver biopsy were performed. The gross specimen (Fig. 4) showed a large, yellow to gray mass in the lower pole of

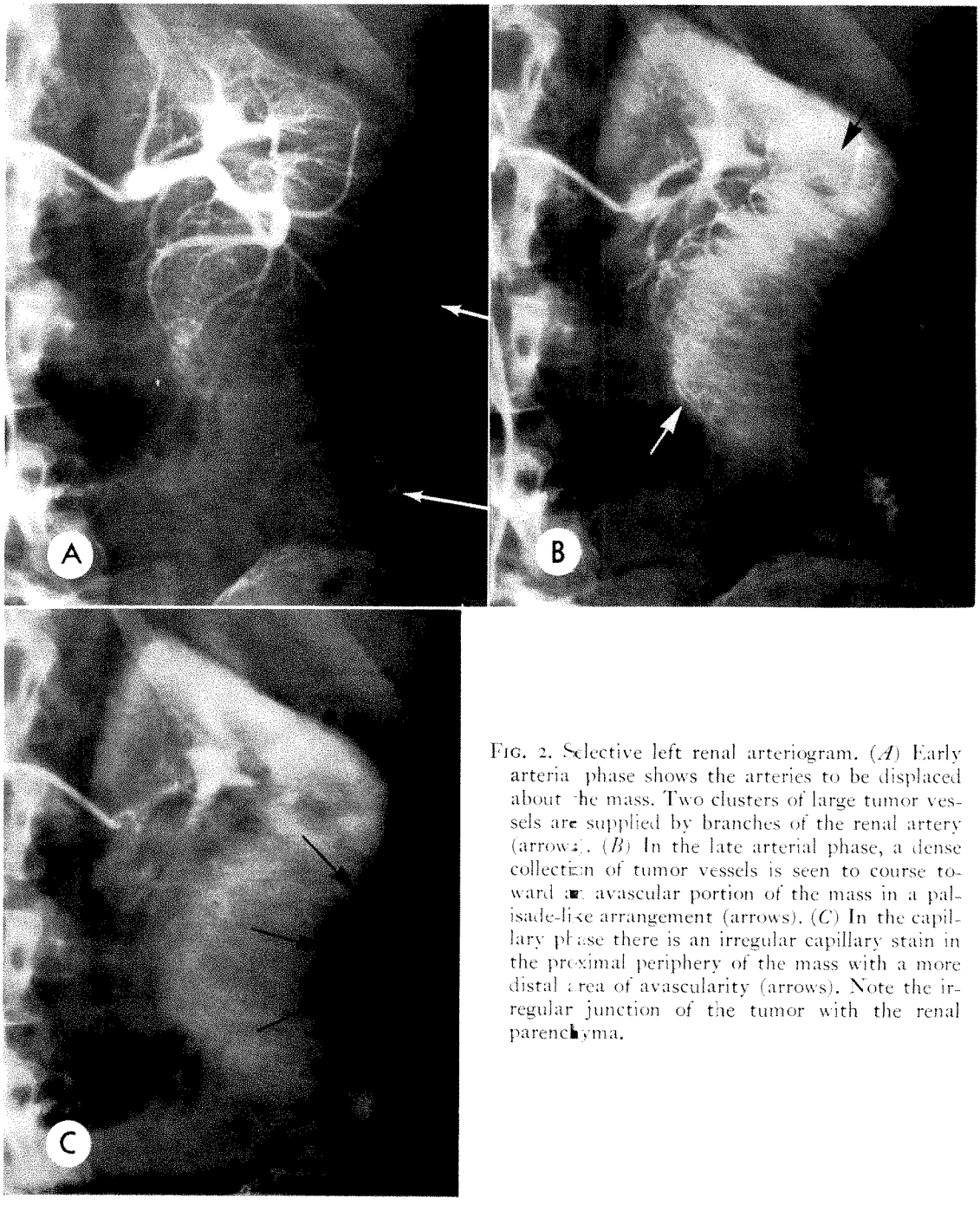



FIG. 2. Selective left renal arteriogram. (*A*) Early arterial phase shows the arteries to be displaced about the mass. Two clusters of large tumor vessels are supplied by branches of the renal artery (arrows). (*B*) In the late arterial phase, a dense collection of tumor vessels is seen to course toward an avascular portion of the mass in a palisade-like arrangement (arrows). (*C*) In the capillary phase there is an irregular capillary stain in the proximal periphery of the mass with a more distal area of avascularity (arrows). Note the irregular junction of the tumor with the renal parenchyma.

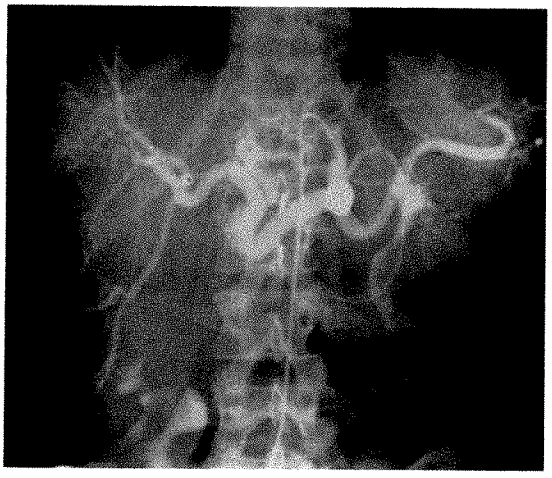


FIG. 3. A celiac arteriogram shows the hepatic arteries to be lengthened and stretched within an enlarged liver, but no tumor vessels or localized masses are apparent.

the left kidney without any area of necrosis. Histologically, it proved to be a malignant lymphoma of the lymphocytic type. Several abdominal lymph nodes were also involved in the process. A liver biopsy at the time of surgery revealed diffuse infiltration of the hepatic parenchyma by lymphoma cells. A cell block from the sediment of the centrifuged pleural fluid contained malignant cells.

## DISCUSSION

In a large series of autopsied cases of malignant lymphoma, Richmond *et al.*[12] found the kidneys to be involved in 33.5 per cent: 13 per cent of Hodgkin's disease; 63 per cent of lymphosarcoma with bone marrow involvement; 38.5 per cent of lymphosarcoma without bone marrow involvement; and 46 per cent of reticulum cell sarcoma. In the entire group, both kidneys were involved three times as often as one. Multiple nodules were the most frequent form of renal involvement in each category accounting for 61 per cent of all cases. Next in frequency was invasion from perirenal disease, present in 11 per cent of the cases. A single bulky tumor was found in only 7 per cent of all cases, being most frequent in reticulum cell sarcoma, and least common in lymphosarcoma. Other types of renal lesions included small solitary nodules in 7 per cent, diffuse infiltration in 6 per cent, and disease evident only on microscopy in 7 per cent.

In spite of the great frequency of renal lesions pathologically (most common organ involved with the exception of the lung in Hodgkin's disease), renal involvement was suspected ante mortem in only 14 per cent of patients. The most frequent clinical findings were a palpable mass in the renal area (9 per cent), hypertension (8.5 per cent), and azotemia (12 per cent). Even in those cases with clinical renal lesions, however, 75 per cent could best be explained by other abnormalities such as dehydration, ureteral obstruction, hyperuricemia, etc. Widely disseminated disease was found to be more common in patients with renal lymphoma than in others.

There are only a few accounts in which malignant lymphoma was believed to arise primarily in the kidney, or in which renal involvement was the predominant feature in an otherwise disseminated disease.[2,4,6–10] It is interesting that Evans *et al.*[5] in reporting on the roentgenographic diagnosis of 100 consecutive cases of renal cancer, had 97 cases of hypernephroma, 1 of a peculiar

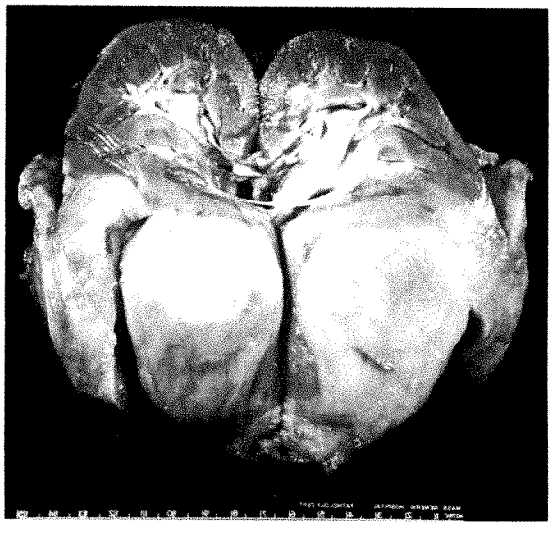


FIG. 4. Gross specimen of the left kidney. A large mass occupies the lower pole. It is somewhat heterogeneous in texture, but no areas of necrosis are present.

mucous adenocarcinoma, and 2 of lymphosarcoma. It is not stated, however, whether the latter were primary or secondary. Our case was probably one of disseminated lymphoma in which the renal involvement was particularly predominant.

The findings on conventional roentgenographic studies of renal lymphoma include diffuse enlargement of both kidneys, localized mass or masses, and decreased or absent renal function. Extrarenal disease may be manifest by renal or ureteral displacement, perirenal mass, ureteral obstruction and hydronephrosis.[12,13]

The arteriographic appearance of renal lymphoma as revealed by the present case has many features in common with other malignant renal tumors (*e.g.*, hypernephroma).[11] There were: (1) displacement of vessels around a mass; (2) typical tumor vessels; (3) an irregular capillary stain of a portion of the tumor; and (4) an irregular junction of the tumor with the normal kidney. In addition, there was an avascular region similar to that found in hypernephromas with areas of necrosis[11] despite the absence of necrosis pathologically. Notable, however, in this case was the peculiarly rich palisade of tumor vessels which occupied the proximal portion of the periphery of this mass. Begg[1] has shown a case of lymphosarcoma involving the acromion process in which there was hypertrophy of a feeding artery but no tumor vessels were mentioned. Strickland[14] showed arteriograms in 2 cases of reticulum cell sarcoma of bone: in one the appearance was normal; in the second, numerous large tumor vessels were seen throughout the mass which were absent in a study after radiation therapy. The pattern in this second case was not different from that of many other malignant bone tumors. It is doubtful that the peculiar appearance of a portion of the tumor vessels in the current case will prove to be characteristic for lymphoma. More likely it is an expression of a highly vascular but densely cellular tumor.

SUMMARY

A case of malignant lymphoma is described which presented as a renal mass and in which renal arteriography was performed. The arteriographic findings were very helpful in this unusual entity.

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# TRAUMATIC RUPTURE OF RENAL CORTICAL CYST INTO THE CALYCEAL SYSTEM*

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THE abnormal kidney is much more easily traumatized than the normal.[6,7] In all reviews of renal trauma, a significantly high percentage of the cases are seen to have pre-existing renal disease, and many of these are cortical cysts. While intraperitoneal, retroperitoneal, and subcapsular rupture of renal cortical cysts have all been described, a search of the literature reveals no documented case of post-traumatic rupture of a cyst into the pelvo-calyceal system.[5,6] This report concerns a patient with multiple cortical cysts of the kidneys and with post-traumatic rupture of one of these cysts into a calyx.

## REPORT OF A CASE

T.V., 53 year old white male. On November 5, 1965, patient ran into a wall while playing tennis and experienced pain and swelling of the right side of his chest and flank. There were no urinary tract symptoms until November 11, 1965 when he had pain with gross hematuria. As a result of intravenous urography done as an outpatient, he was subsequently admitted to Sinai Hospital on November 12, 1965 (Fig. 2). A prior intravenous urogram from 1960 was available for comparison (Fig. 1).

On November 15, 1965 intravenous nephrotomography and urography revealed rupture of a lateral cortical cyst of the right kidney into an upper pole calyx, with filling of the cyst from the collecting system in a retrograde fashion (Fig. 3, *A–C*). A second cortical cyst was seen in the anteromedial portion of the upper pole. A very large posterior cyst of the left kidney was displacing the lower pole superiorly and anteriorly (Fig. 4*A*). A calyceal "diverticulum" was seen in the upper pole of the left kidney (Fig. 4*B*). Several non-displaced lower rib fractures were also noted.

During the one week under observation in the hospital, the patient's urine gradually cleared, and he was discharged on November 21, 1965. He has been asymptomatic to the present.

On May 10, 1966, a repeat nephrotomogram and urogram revealed obliteration of the cyst that had previously ruptured with a residual cortical scar at that site (Fig. 3*D*). The other cysts were unchanged, as was the upper pole calyceal "diverticulum" of the left kidney.

## DISCUSSION

In addition to being more vulnerable to renal trauma, renal cortical cysts have

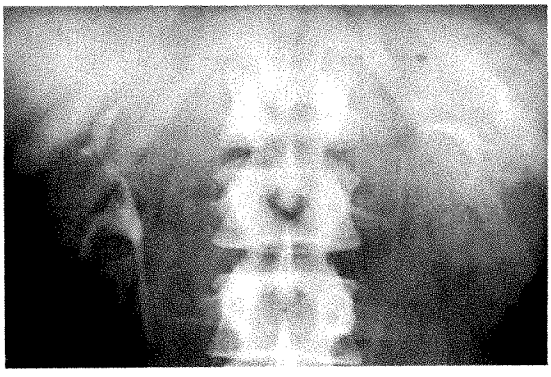


FIG. 1. No apparent abnormality (1960).



FIG. 2. Communication of upper pole cyst with upper pole calyx (arrow). The irregularities seen at the lower border of the cyst probably represent admixture with blood. Also note large mass in the lower pole of the left kidney which was asymptomatic.

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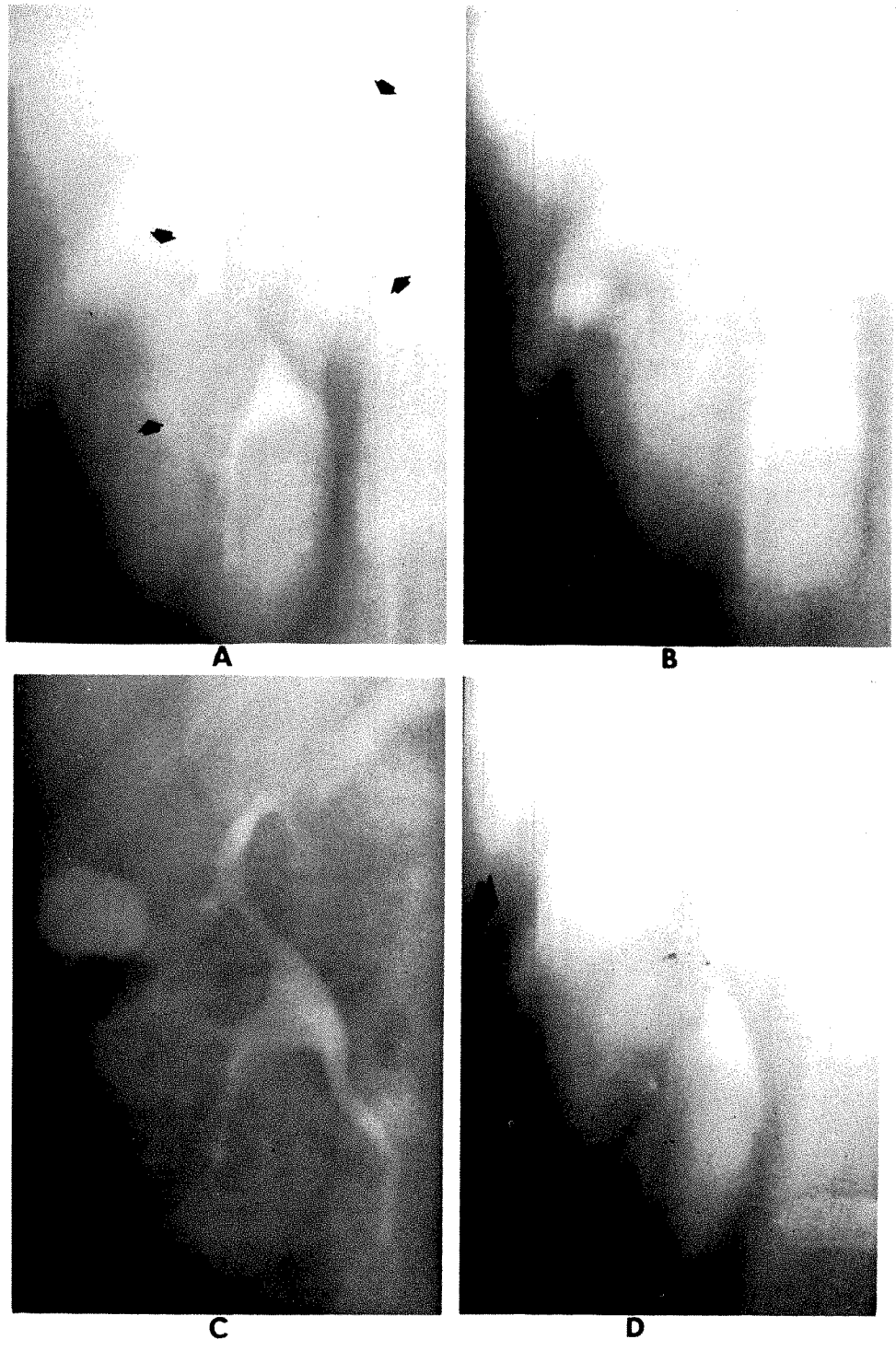



FIG. 3. (*A*) Nephrotomogram demonstrating radiolucent defects at the site of the ruptured lateral cyst (left arrows), and an intact upper pole medial cyst (right arrows). (*B*) Roentgenogram from the same study as *A*, several seconds later, demonstrating retrograde filling of the cyst from the communicating calyx. (*C*) Subsequent urogram visualizing the total extent of the cyst. (*D*) Repeat nephrotomogram 6 months after the initial trauma demonstrating no evidence of a residual cyst, and a cortical scar at the site of the previously ruptured cyst (arrow).

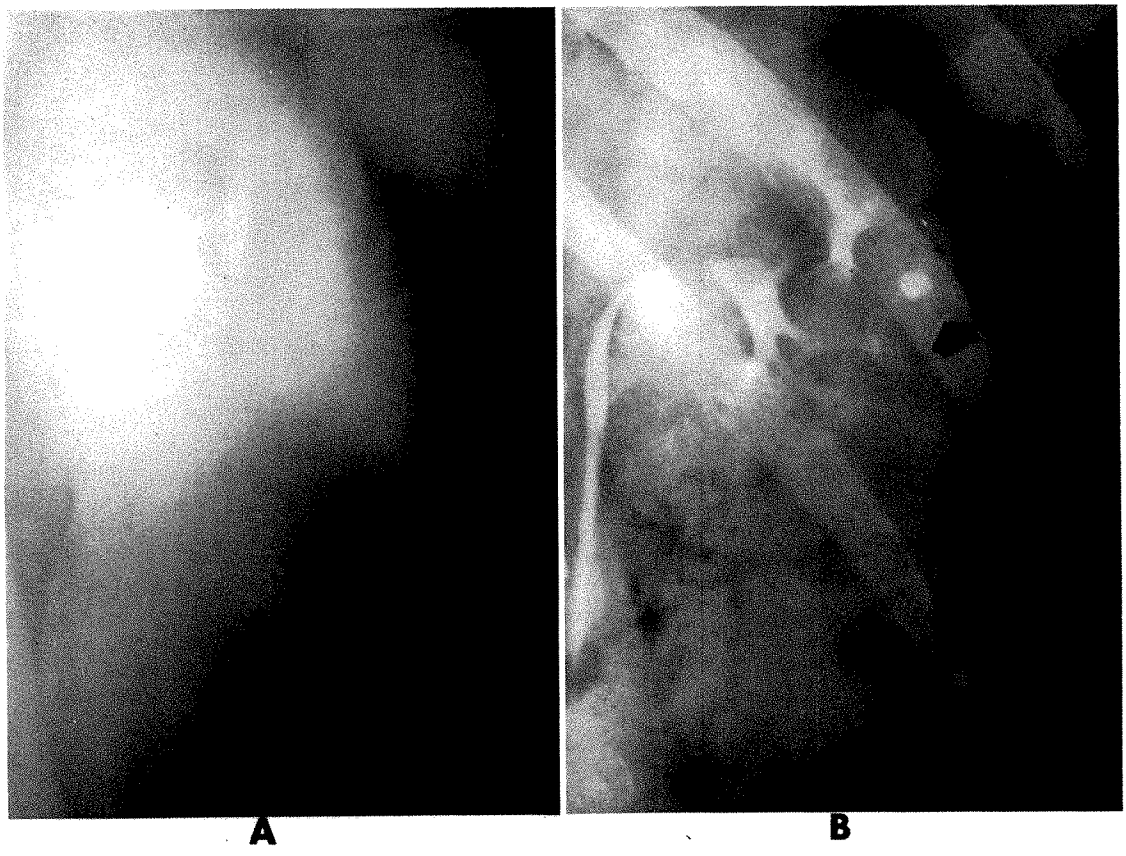



FIG. 4. (*A*) Large lower pole cyst of the left kidney. (*B*) Calyceal "diverticulum" (arrow).

been known to rupture spontaneously.[1,2] Spontaneous rupture of cortical cysts into the renal pelvis or into a calyx has been described, and an infectious etiology has been implied. Bauchart[1] states that rupture of cortical cysts into the collecting system is very common in their evolution, but not noted in most cases.

In the case described, the ruptured cyst was unequivocally demonstrated to communicate with a calyx and then heal, with obliteration of the channel into the collecting system, leaving a cortical scar. It is interesting that in this patient with multiple cortical cysts, a calyceal "diverticulum" was seen as well. One would wonder whether this calyceal "diverticulum" could represent a previously ruptured cyst, and whether this may be one way a ruptured cyst may resolve. Once the communication is made, differentiation between ruptured cyst, calyceal diverticulum, and hydrocalyx may be extremely difficult, if not impossible.[2,3,8] It is important to roentgenologically follow these cysts after rupture, whether the etiology is traumatic, infectious, or spontaneous, in an effort to elucidate their natural history.

SUMMARY

A documented case of post-traumatic rupture of a renal cortical cyst into the collecting system is reported.

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# PERCUTANEOUS ASPIRATION OF RENAL MASSES*

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WITH the development of television-controlled fluoroscopy and the opportunity for localization of a mass in the kidney, a guided approach to aspiration biopsy of renal masses has become possible.[6,10] Early investigators used roentgenograms to establish anatomic landmarks and achieved a fair degree of success.[1,4] Lindblom[10] has described and encouraged the use of needle aspiration biopsy in renal masses and has offered evidence that tumors are not apparently disseminated by this method. Our experience with this procedure has been limited but has encouraged us to continue its use, integrate it into the spectrum of available techniques and to report our results.

## METHOD

Approximately 10 minutes following the intravenous injection of urographic contrast medium, the patient is placed prone on the fluoroscopy table. The mass previously observed on the excretory urogram will then be again apparent and the skin overlying the mass is marked for puncture. The skin is cleansed and local anesthesia of the skin and subcutaneous tissue is carried out. A small incision of the skin with the tip of a scapel blade will more easily permit the use of a No. 14 needle of approximately 1½ inches length. This acts as a sleeve permitting easier movement of the biopsy needle. Through this is advanced an 8 inch thin-wall No. 20 spinal needle complete with stylet. Under fluoroscopic control, and while the patient suspends respiration, the needle is advanced into the mass. Usually the cyst wall will be felt to suddenly give away. The stylet is then removed and a short plastic tubing is attached. The fluid obtained from the cyst quickly fills the tubing and can be siphoned off. It will commonly be clear but may be faintly yellow in color, causing one to wonder if he has not entered the renal pelvis inadvertently. The quantity of fluid which may be obtained will vary with the size of the cyst and may be only a few milliliters, or larger in volume.

Following aspiration of a portion of the fluid, contrast medium is injected in a quantity sufficient to outline the cyst and roentgenograms are made in the prone, oblique and possibly lateral projections. As the needle is kept in place, some care is necessary in positioning the patient. The entire fluid content of the cyst is then aspirated and the patient is sent on his way.

In the case of a tumor, no fluid is obtained or a thick bloody material may be aspirated, as in Patient 12. A small quantity of contrast medium may be injected but this will probably not prove helpful. A small cyst can be missed as in Patient 10, but usually a mass of any size can be easily seen on the urogram produced and knowledge of the fact that the needle tip is in the mass can be gained through rotating the patient or the C-arm of the fluoroscopic apparatus.[7] The absence of fluid in the mass should be considered evidence that one is dealing with a neoplasm. The few drops of fluid aspirated from such a mass is smeared on slides, fixed promptly in 95 per cent ethyl alcohol and studied by the cytologist. The needle and syringe are also rinsed in normal saline and this is sent to the cytology department. Fluid from cysts is treated in the same way. We have not used the study of fat in the fluid as suggested by Lang[8] as a means of distinguishing between cyst and tumor of the kidney.

## MATERIAL

We have employed this procedure on 15 patients with pertinent information recorded in Table 1. The patients ranged in age from 38 to 74 years, were evenly divided as to sex, and were nearly all hospitalized.

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More of the lesions were right-sided than left. There was no morbidity or mortality. In Patient 10, a 1½ cm. diameter cyst was missed by 2 mm. The needle tract was identified at laparotomy performed the following day. This was our only misdiagnosis by this method. In Patients 4 and 12, the correct diagnosis of tumor was made; how-

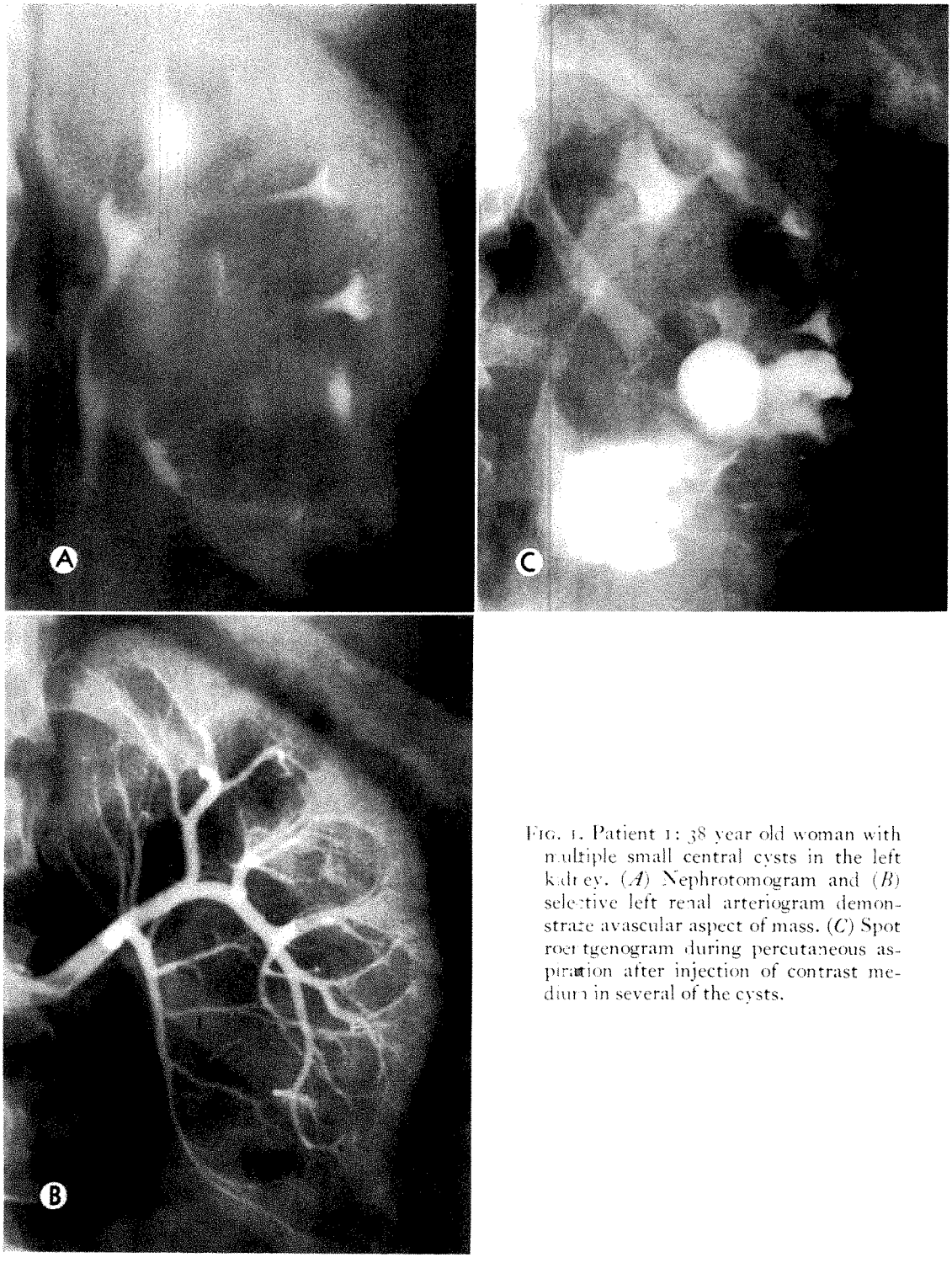



FIG. 1. Patient 1: 38 year old woman with multiple small central cysts in the left kidney. (*A*) Nephrotomogram and (*B*) selective left renal arteriogram demonstrate avascular aspect of mass. (*C*) Spot roentgenogram during percutaneous aspiration after injection of contrast medium in several of the cysts.

TABLE I

SUMMARY OF FINDINGS IN 15 PATIENTS

| Patient | Sex | Age | Side | Signs or Symptoms | Uro-graphic Diagnosis | Angio-graphic Diagnosis | Nephroto-mographic Diagnosis | Nature of Fluid | Cytology | Aspiration Diagnosis | Surgical Diagnosis | Final Diagnosis | Result |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | F | 38 | L | none | cyst | cyst | cyst | clear | acellular | multiple small cysts | none | cysts | beneficial |
| FIG. 1, A-C | | | | | | | | | | | | | |
| 2 | M | 62 | R | backache | cyst | none | cyst | clear, slight bleeding | acellular | cyst | none | cyst | beneficial |
| 3 | M | 54 | L | none | cyst | cyst | none | clear | acellular | cyst | cyst | cyst | beneficial |
| FIG. 2, A-C | | | | | | | | | | | | | |
| 4 | M | 65 | R | hematuria, flank pain | mass | none | none | few drops of serum | acellular | tumor | tumor | tumor | beneficial |
| 5 | F | 71 | L | backache | cyst | none | cyst | clear | acellular | cyst | none | cyst | beneficial |
| 6 | M | 56 | R | microscopic hematuria | mass | cyst | tumor? | clear | acellular | cyst | none | cyst | beneficial |
| 7 | M | 66 | L | none | cyst | none | cyst | clear | acellular | multiple cysts | none | cyst | beneficial |
| 8 | F | 64 | R | flank discomfort | cyst | cyst | none | clear | acellular | cyst | none | cyst | beneficial |
| 9 | M | 74 | L | none | cyst | none | cyst | clear | acellular | cyst | none | cyst | beneficial |
| 10 | F | 46 | R | backache | cyst | none | none | few drops of serum | acellular | tumor | 1½ cm. cyst | cyst | misdiagnosis |
| 11 | M | 66 | R | none | cyst | none | cyst | clear | acellular | cyst | none | cyst | beneficial |
| 12 | F | 55 | R | flank pain, hypertension | cyst | cyst | none | brown, thick | necrosis acellular | tumor | tumor | tumor | beneficial |
| 13 | F | 53 | R | none | cyst | cyst | cyst | clear | acellular | cyst | none | cyst | beneficial |
| 14 | M | 64 | L | backache | cyst | cyst | cyst | clear | acellular | cyst | none | cyst | beneficial |
| 15 | F | 66 | R | none | cyst | none | none | clear | acellular | cyst | none | cyst | beneficial |

ever, cytologic confirmation of a tumor was not obtained by this method. No evidence of metastatic disease has been observed in the renal carcinoma patients, 2 years after resection. This is in accord with the observations made by others.[8,10] Seven of the patients had selective angiograms which were correct in 6 cases. The seventh instance, Patient 12, gave the appearance of a cyst which is not uncommon in very necrotic tumors.[2,14] Nephrotomograms were used in 9 patients and were correct in indicating the diagnosis in 8.

## DISCUSSION

It is unfortunate that there continues to be opposition to percutaneous aspiration of renal masses.[12] Much time, effort, expense and hospitalization could be saved if most masses seen on the excretory urogram could be aspirated in the urographic laboratory before the patient was discharged. This would perhaps appear to be too precipitous an action but we feel that little would be lost and no serious dangers run with the exception of the exceedingly rare individual who has an unrecognized renal abscess. In such cases a septicemia may be precipitated by the use of the needle aspiration.

Association of cyst and tumor is often discussed but large series prove this to be of miniscule possible proportion.[3,13] In the study by Levine *et al.*,[9] of 1,007 cases of cyst and tumor, the close approximation of cyst and tumor occurred once and multiple degenerated cystadenomas appeared once, making an incidence of only 0.2 per cent. The essentially self-healed renal carcinoma may be mistaken for a cyst even at surgery.[2,14] These tumors appear to pose little threat to longevity.[2] The possible morbidity and mortality involved in laparotomy in the elderly must be weighed against the possible error of the aspiration.[5,11,12]

The possible spread of tumor along the needle tract is the usual objection to this procedure. No evidence to support this assumption is current and there is reason to believe that the trauma associated with surgical resection is much more likely to

be cause of dissemination than the use of a slender needle. In Lindblom's series[10] there was evidence that the aspirated group did better than the nonaspirated group after surgical extirpation. Hemorrhage could occur but has not developed in any of our patients and has not been reported to be a problem. We perform this examination on an out-patient basis and 3 of our patients were in this category.[10]

If one accepts that this is a safe, quick means of determining the nature of a renal mass and that operability and curability of renal tumors is not impaired by using it,

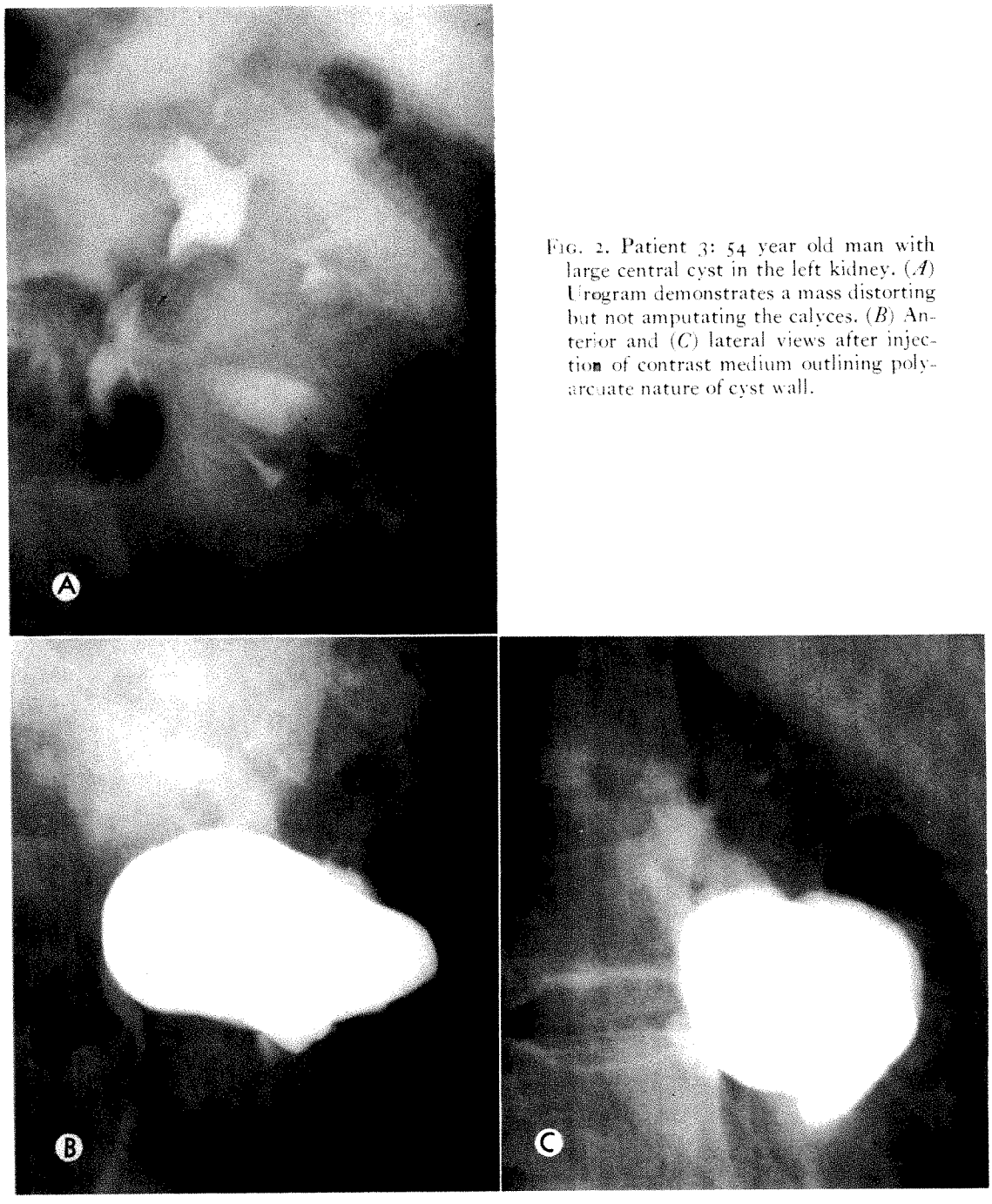



FIG. 2. Patient 3: 54 year old man with large central cyst in the left kidney. (*A*) Urogram demonstrates a mass distorting but not amputating the calyces. (*B*) Anterior and (*C*) lateral views after injection of contrast medium outlining polyarcuate nature of cyst wall.

then there is no cogent reason to forego it or to insist that it be preceded by nephrotomography or renal angiography.[5,7,10] There are those who state that it be used exclusively in obvious cases of cyst or not at all.[12] We would suggest that renal angiography be used following aspiration in those cases not proven to be obviously cysts by aspiration. In this way, the diagnosis of tumor will be confirmed, its extent delineated and operability ascertained.

By following the sequence of urogram then puncture followed by angiography, a logical quick approach to most renal masses would be established. There is no widely accepted evidence that all renal masses need to be removed and aspiration has often resulted in the disappearance of many renal cysts.[5,6,10]

The outline of a renal cyst can be studied and correlated with the mass distorting the urogram. The cysts are not always spherical and smooth-walled, and mild irregularities and polyarcuate outlines can be accepted as consistent with the diagnosis of cysts.[10] Of greater importance is the presence of a clear, acellular fluid in the aspirate. When the opacified cyst has not filled the entire volume of the mass, one must consider if there are other adjacent cysts or an adjacent tumor. The very occasional possibility of a tumor located next to a cyst must be kept in mind in this consideration.[5] Additional placements of the needle, aspiration and opacification can prove, as in Patients 1 and 7, that the mass on the urogram is composed of multiple cysts. Failure to resolve this problem is one clear indication for renal angiography and possible renal exploration.

## SUMMARY

The method and utilization of fluoroscopically-controlled television-monitored aspiration of renal masses following the production of a urogram is described and discussed. Fifteen patients examined by this method are reported and the results evaluated.

The desirability of performing this examination in most renal masses with the exception of suspected renal abscesses is stressed. It is recommended that the aspiration immediately follow the urography and precede the angiography or nephrotomography in the order of investigation of renal masses.

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# UNUSUAL RENAL CALCIFICATION FOLLOWING ACUTE BILATERAL RENAL CORTICAL NECROSIS*

## A CASE REPORT

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LONG term survival following acute bilateral renal cortical necrosis was unusual in the past. Renal dialysis and improved fluid and electrolyte management have prolonged patient survival time[6] so that new manifestations of the natural progression of this entity are being observed. This paper reports an unusual form of calcification which developed in a postpartum patient who experienced sickle cell crisis with subsequent acute renal cortical necrosis. We believe this report to be unique in a patient with sickle cell disease.*

### REPORT OF A CASE

An 18 year old Negro female with proven sickle cell disease delivered her first child following an uneventful pregnancy and an uncomplicated delivery on April 10, 1965. The sudden onset of profound shock appeared 9 hours postpartum with successful resuscitation after 1½ hours. This was followed shortly thereafter by severe bilateral flank pain, gross hematuria, and oliguria which was unresponsive to mannitol. Typical acute renal failure (renal cortical necrosis) with uremia followed and was treated by peritoneal dialysis and the usual measures with ultimate survival. Serum creatinine values reached a high of 13.2 mg. per cent, falling to 4 to 5 mg. per cent at discharge 60 days following delivery.

Renal calcification became visible on abdominal plain roentgenograms 44 days after delivery with progressive calcification obvious on follow-up roentgenograms (Fig. 1–4). The renal size also decreased progressively from 12.9 cm. length when first visible to 9.9 cm. at discharge, and 8.6 cm. when last seen 12 months post partum.

Since discharge, the patient has remained asymptomatic and active. She has continued severely anemic and in chronic renal failure with an endogenous creatinine clearance averaging 8–10 ml. per minute.

### DISCUSSION

The first reported case of bilateral renal cortical necrosis is attributed to Juhel-Renoy in 1886.[3] Since that time, well over 200[7] cases have been documented, usually at postmortem examination. The usual symptoms follow a characteristic pattern.[10] The abrupt onset of oliguria is seen in a

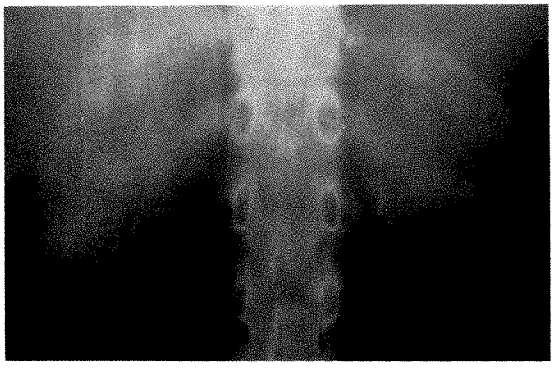


FIG. 1. *May 24, 1965*: Laminagram reveals a 1–2 mm. line of calcification in the subcapsular area. Plain roentgenograms suggested an increase in renal density. The plain longitudinal renal measurements is 12.9 cm. on the right and 12.4 cm. on the left.

* A detailed clinical report concerning this case will be published elsewhere.[2]

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This material has been reviewed by the Office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any indorsement of the opinions advanced or any recommendation of such products as may be named.
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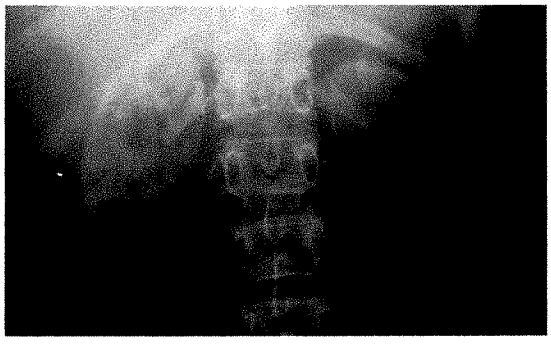


Fig. 2. *August 17, 1965*: Plain roentgenogram shows dense, diffuse, cortical calcification sparing the medulla. Longitudinal renal measurement is 9.8 cm. on the right and 9.9 cm. on the left.

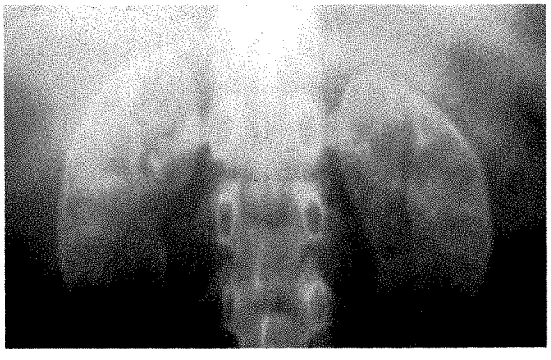


Fig. 3. *November 19, 1965*: Laminagram shows increased cortical density with the uninvolved medullary portions now appearing relatively more sharp and lucent. Plain film renal measurements are 9.6 cm. on the right and 9.7 cm. on the left.

patient who is experiencing sharp flank pain. Anuria and uremia rapidly follow. Gross hematuria is often present and is considered an important point in differential diagnosis. The temperature usually remains normal. Shock is a common but not constant finding.

The etiology of this entity is varied.[5,10] Two-thirds of the cases are reported in association with third trimester bleeding usually secondary to abruptio placentae. Other causes include severe trauma with associated shock and hemorrhage, overwhelming infection of any source with bacterial shock, incompatible blood transfusion, mercury or phosphorus poisoning, and severe dehydration in children after prolonged vomiting or diarrhea.

Renal ischemia is probably the direct cause of necrosis in the cortex.[8] This may result from multiple emboli, fibrin deposition, direct vascular damage, or vasoparalysis. Experimentally, similar lesions have been produced by bacterial toxins, vasoconstrictors, and toxic chemicals. Associated necrosis of the anterior pituitary gland and adrenals has also been noted. Stanley[8] reports microscopic changes consisting of large cortical areas of coagulation and liquefaction necrosis extending almost to the renal pyramids and covered externally by a thin subcapsular rim of viable tissue. The medulla is spared. Varying amounts of calcification are observed microscopically and grossly.

## ROENTGEN FINDINGS

Diffuse renal calcium deposition in the past has been rare due to rapid patient demise. With prolonged survival, more cases are being observed. Cramer and Fuglestad[1] described the eighth reported case in a patient surviving 2½ years.

Plain roentgenographic findings during the anuric phase may reveal swollen kidneys with indistinct outlines. The latter finding is due to retroperitoneal fluid collection and edema during the anuric phase. Following dialysis and diuresis, the outlines again become distinct. Calcification has not been observed before the fourth

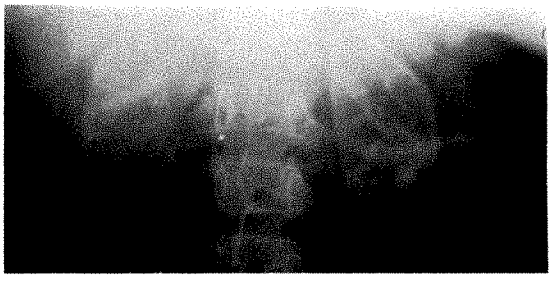


Fig. 4. *May 9, 1966*: Characteristic cortical calcification is revealed. Prominent decrease in renal size is seen compared with the early roentgenograms. The renal length is now 8.6 cm. on the right and 9.0 cm. on the left.

week and usually appears in 4 to 8 weeks.[5] The first impression is one of general overall increase in renal density which can best be demonstrated by laminagraphy. Lloyd-Thomas *et al.*[4] described thin parallel lines of calcification in the juxtamedullary and subcapsular regions which he called tram-line calcification. However, this appearance has not been noted by others, nor observed in the present case.

Serial roentgenograms show the renal density to increase gradually until a dense band of heavy calcification is seen throughout the cortical area with complete sparing of the medullary portions which appear as relatively lucent pyramids. This appearance is typical of bilateral renal cortical necrosis. As cortical density increases there is noted a progressive decrease in cortical thickness with eventual significant decrease of the renal measurements. No change in the medullary area is noted.

Although the observed calcification is certainly unique, differential diagnosis should include renal tubular acidosis, primary hyperparathyroidism, hypervitaminosis D, ingested toxic substances (mercury, sulfonamide), chronic glomerulonephritis, tuberculosis, sarcoid, hemorrhage, and extensive renal infarction.

## SUMMARY

The typical roentgenographic appearance and progression of calcification in a case of bilateral renal cortical necrosis in a postpartum patient with associated sickle cell disease are demonstrated. The shell-like cortical calcification with sparing of the medulla is characteristic. Due to prolonged patient survival, it is felt that this finding will be observed with increasing frequency.

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# MILK OF CALCIUM RENAL DISEASE

## REPORT OF CASES AND REVIEW OF THE LITERATURE

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MILK of calcium renal disease was first described by Howell[10] in 1959, and 8 additional reports[1,8,11,13–15,19,20] have appeared since then. All of these articles have discussed milk of calcium in calyceal diverticula.

The purpose of the author is: (1) to add a tenth such case and review the literature, particular attention being paid to the clinical and roentgenographic features; and (2) to report an unusual case of milk of calcium in a hydronephrotic kidney.

### I. REPORT OF AN ADDITIONAL CASE OF MILK OF CALCIUM IN A CALYCEAL DIVERTICULUM

Case 1. A 32 year old white female contracted left upper lobe pulmonary tuberculosis in 1954 and was treated with INH and streptomycin until 1956. There was no history of urinary tract disease. On a routine follow-up chest roentgenogram in 1963, in addition to a calcified pulmonary focus, a half-moon shaped calcific density in the right upper quadrant of the abdomen was seen. Subsequent roentgenograms showed this to be milk of calcium in the right kidney (Fig. 1) and not within the gallbladder (Fig. 2). Intravenous pyelography resulted in markedly increased opacification of the cystic lesion containing the calcifications (Fig. 3). A 2 year follow-up abdominal roentgenogram showed no change in the calcifications. Urinalyses have been consistently normal and the patient remains asymptomatic.

### DISCUSSION

Calyceal cysts or diverticula, or pyelogenic cysts as they may be called, are not uncommon findings, but milk of calcium within such a cyst is quite unusual. The calyceal diverticulum is thought to be a congenital abnormality, representing a developmental aberration of the wolffian ducts. The postinflammatory origin has not been discounted however.[4,9,17] It may or may not communicate with the collecting system of the kidney. This is in contrast to the simple parenchymal cyst which rarely if ever communicates with the calyces.[5,6] Ferguson and Ward-McQuaid[7] state that the diagnosis of calyceal diverticulum may be made regardless of a demonstrable communication. The wall of the diverticulum consists of smooth muscle with a transitional cell epithelial lining. This has been confirmed by Walker *et al.*[20] and Benendo and Litwak.[1] When

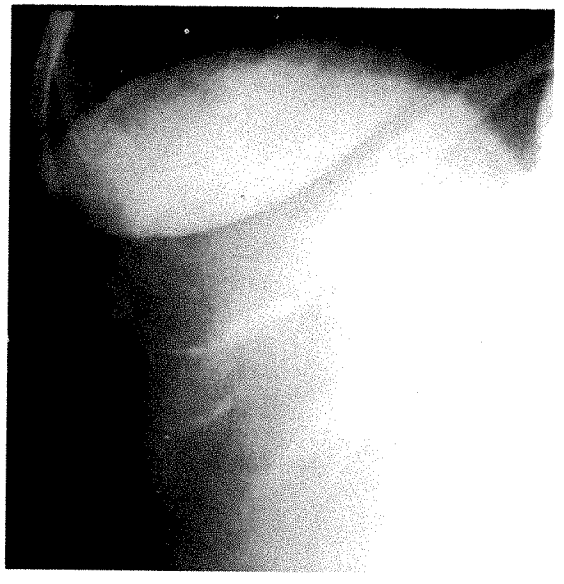


Fig. 1. Case 1. Erect roentgenogram of the right upper quadrant. The layering of the milk of calcium in the typical half-moon configuration is shown.

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This material has been reviewed by the Office of The Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any indorsement of the opinions advanced or any recommendation of such products as may be named.

communication with the calyx is not continuous, infection becomes possible and stone formation may ensue.[5] Calculi may also form without antecedent infection. In the series of 20 cases of calyceal diverticula reported by Yow and Bunts,[21] 11 had calcifications, and of these 7 were proven surgically. None of the cases had the peculiar milk of calcium however. These authors stated that inflammation of the diverticulum leads to infected urine and stone formation. This is analogous to the origin of milk of calcium in the gallbladder as a result of cystic duct obstruction with bile stasis.[2,12,18]

In the case of Walker *et al.*[20] there was chemical analysis of the stones; they were found to be calcium carbonate. Boyce[3] discussed milk of calcium in the kidney and showed typical plain roentgenograms but referred to the composition of the stones as calcium phosphate (hydroxyapatite).

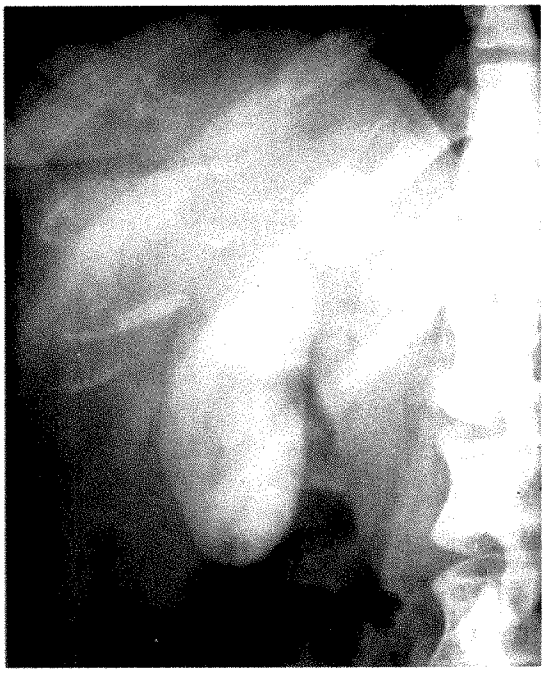


FIG. 2. Case 1. Oral cholecystogram. The calcifications within the cyst above the gallbladder are well demonstrated. The proximity to the gallbladder is fortuitous; the lesion is actually retroperitoneal.



FIG. 3. Case 1. Intravenous pyelogram. Oblique projection. A marked increase in the density of the calyceal diverticulum is noted (compare with Figure 2). The upper pole calyces are partly obscured but not distorted. No sinus tract is visible.

### INCIDENCE

In the 10 cases reviewed in this article sex distribution has been equal. The patients have ranged from 32 to 71 years in age with a mean age of 49 years. The milk of calcium has been in the upper half of the kidney in all cases; it was on the right side as often as it was on the left side.

Only in our Case 1 was there a history of pulmonary tuberculosis but this was apparently unrelated to the milk of calcium.

No patient had any evidence of hyperparathyroidism and 1 patient of the 10 had cancer.

### CLINICAL SYMPTOMS

In Case 1, and those of Walker *et al.*,[20] Maurer and Wilden,[13] Henken,[8] Iozzi *et al.*,[11] and Vandervort[19] renal milk of calcium was an incidental finding. The other 4 cases[1,10,14,15] presented with symptoms of kidney disease. Specifically, Howell's patient[10] had a long history of recurrent urinary tract infections without colic before the lesion was discovered. It was thought to be in the collecting system and surgery was done without finding the calcifications. There was a decrease in the amount of calcium within the calyceal cyst on long term follow-up roentgenograms but

this could have been due to chemical changes and not necessarily to the passage of calculi. The patient of Benendo and Litwak[1] had in addition to milk of calcium in a pyelogenic diverticulum, an obstructing ureteropelvic junction calculus with typical renal colic. It is possible that the milk of calcium in the cyst supplied the nidus for the larger stone. Roentgen studies showed no visible communication between the cyst and the adjacent calyces. In the case of Morin and Albert,[14] the milk of calcium was in a right calyceal cyst but the patient also had bilateral discrete kidney stones. Colic was manifested only on the left side in this instance. Similarly, the patient of Pullman and King[15] had renal milk of calcium on the right side, but initially present with flank pain on the left. Subsequently he complained of right costovertebral pain but there was considerable doubt whether this was renal or not.

In reviewing the symptomatology, it is apparent that no patient has presented with symptoms directly referable to the milk of calcium in the cyst. Also, on analyzing Yow and Bunts' tabulated list of 10 patients with calcifications in calyceal cysts, flank pain was almost always accompanied by fever and chills or pyuria, or both. One must therefore be careful not to ascribe upper urinary tract symptoms to milk of calcium in a calyceal cyst before excluding infection or other stones.

### ROENTGEN DIAGNOSIS

The roentgenographic appearance of the half-moon or hemispherical calcium-fluid level has been the unique and diagnostic feature in all cases. Supine and decubitus roentgenograms showed that the calcifications were mobile within the cyst and were heavier than the nonopaque fluid, which, according to Campbell,[5] has the same composition as urine. The lesion was round, or nearly round in the supine projection in 9 of the 10 cases, but the horizontal beam study demonstrated the straight superior and convex inferior contour of the milk of calcium collection in every instance. The calcifications are finely granular on the roentgenogram, and of importance is the absence of fixed calcium deposits in the wall of the cyst. It is not known why the milk of calcium does not solidify or why it does not deposit in the wall as occurs in the wall of solitary renal cysts, infected cavities, or areas of old hemorrhage.[6,16]

The precise localization of the calyceal cyst is easily made by intravenous pyelography, and the erect or decubitus roentgenogram is essential to show the characteristic calcium-fluid level. In the cases of Morin and Albert,[14] and Pullman and King,[15] as well as in our Case 1, there was increased opacification of the cyst on the intravenous pyelograms. This feature has not been demonstrated with retrograde pyelography, nor has a diverticulum-to-calyx communication been shown. Morin and Albert[14] state that the sinus tract may be too small to be seen on the roentgenographic study. They also postulate that functioning nephrons in the wall of the cyst might account for the increased opacification. My belief is that the former theory is less plausible than the latter and that if a sinus tract does exist, failure to show it on the retrograde examination might be due to edema or spasm of this communication.

The location of renal milk of calcium in the lateral view of the abdomen will differentiate it from milk of calcium bile. Oral cholecystography will provide additional confirmation. One must also exclude by history the presence of an intestinal diverticulum filled with contrast material or containing calculi. Mesenteric lymph node calcifications are not difficult to distinguish in the supine projection and of course do not show the fluid level in erect or decubitus views. With all of the roentgenographic features in mind, the radiologist should be able to make the diagnosis of renal milk of calcium with a minimum of time and special examinations.

TREATMENT

From the surgical standpoint it is particularly important to distinguish the calyceal cyst calcification from pelvocalyceal calculi. Medically we must recognize that urinary tract infection often complicates the clinical picture.

No patient in this series has had surgical intervention for removal of the milk of calcium. Whether or not this is the proper approach remains to be seen, but from the available evidence conservative treatment has yet to be proven harmful to the patient. Certainly, further observations of the incidental cases should prove of considerable value.

## 2. REPORT OF AN UNUSUAL CASE OF MILK OF CALCIUM IN A HYDRONEPHROTIC KIDNEY

Case II. A 37 year old Oriental male was admitted to the hospital with crampy right lower abdominal pain of one week's duration. The patient had experienced similar pain intermittently for the previous year and an intravenous pyelogram done many months before in his homeland reportedly showed right renal calcifications but no obstruction. The patient has never had any urinary tract symptoms however.

Past history included treatment for pulmonary tuberculosis 13 years ago, and a single episode of typhoid fever as a child. Family history was noncontributory. Review of systems was also not remarkable.

On physical examination the patient was an acutely ill but well nourished male. The only abnormal finding was a tender palpable mass, about 10 cm. in diameter, in the right lower quadrant. It was also tender to rectal examination.

Laboratory Findings. Hematocrit, serum calcium and phosphorus, electrolytes, and serology were normal. The white blood cell count was slightly elevated with a shift to the left. Urinalysis was normal except for an occasional white blood cell per high power field. Urine cultures, including those for acid-fast bacilli were normal. Stool guaiac examination was negative on several occasions; one specimen was "trace" positive for occult blood.



Fig. 4. Case II. Erect roentgenogram. Multiple calcium-fluid levels are the result of the layering of the milk of calcium (solid black triangles). The three large irregular calculi are within the hydronephrotic kidney. The most medial of these stones is impacted at the ureteropelvic junction (see Figure 5).

Skin test for tuberculosis was read as negative.

The mass was believed to be inflammatory cecal disease and under medical management it disappeared together with all symptoms in 2 weeks.

Initial abdominal roentgenograms showed three discrete irregular calculi and several amorphous calcified densities in the right upper quadrant. These were definitely separate from the palpable lower abdominal mass. A diagnosis of hydronephrosis with calcifications was made. On the erect roentgenogram the calcified zones formed multiple varying sized calcium-fluid levels (Fig. 4). The right kidney did not visualize on the intravenous pyelogram; the left kidney was normal but not hypertrophied. Intravenous cholangiography showed that the gallbladder and common bile duct were displaced medially by the hydronephrotic kidney. Upper gastrointestinal study was normal ex-

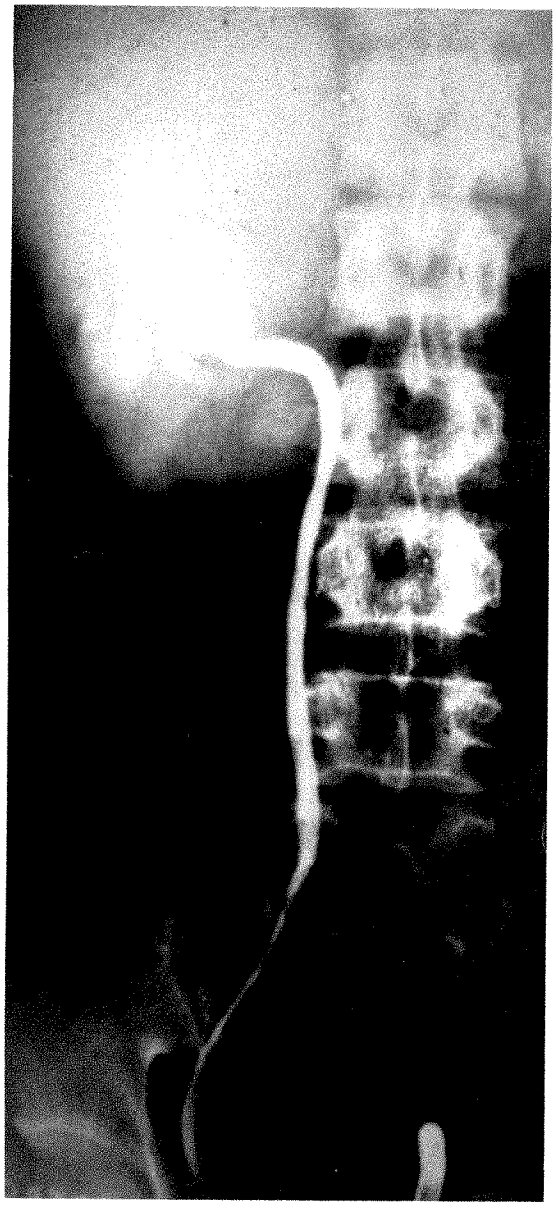


Fig. 5. Case II. Retrograde pyelogram. There is complete obstruction of the ureteropelvic junction.

cept for an extrinsic defect on the right side of the descending duodenum. Chest roentgenography was unremarkable aside from a fibrocalcific parenchymal scar in the right apex. Right retrograde pyelography demonstrated total obstruction by an irregular stone impacted at the ureteropelvic junction (Fig. 5).

Hospital Course. On antibiotics and bed rest the patient became asymptomatic in 2 weeks. Shortly thereafter percutaneous renal biopsy was attempted but it was unsuccessful and complicated by gross hematuria. Subsequently the patient had a right nephrectomy from which he made an uneventful recovery.

Pathology. Examination of the specimen showed a grossly hydronephrotic kidney with only minimal residual functioning tissue at the upper pole. Two large stones were impacted in the renal pelvis, one of which extended into the uppermost portion of the ureter. A third large irregular stone was found in a dilated calyx. Several hundred cubic centimeters of brownish fluid were present in the dilated pelvis and calyces, and there were numerous collections of tiny black and sand-white stones. The brown fluid contained many red blood cells and this was considered to be from the attempted renal biopsy. There was no evidence of tuberculosis on gross or microscopic sections although some portions of the kidney showed chronic inflammatory changes with fibrosis. Cultures of the brown fluid were sterile.

*Comment.* Calcifications in a hydronephrotic kidney are occasionally seen but milk of calcium has not been noted before. Like milk of calcium in calyceal cysts, the origin is not known. Whether or not there is a common etiologic factor to both is open to speculation. My belief is that there is, and that it is a chemical change in the urine as a result of obstruction with infection.

In this case, the hydronephrosis was a direct result of an impacted calculus of the ureteropelvic junction, but one cannot assume that the milk of calcium formed after this. It is possible that these fine sandy particles provided the nucleus upon which the obstructing calculus later grew. The resemblance to the case of Benendo and Litwak,[1] referred to earlier in this paper should not be overlooked.

The roentgenographic appearance of milk of calcium is similar to that noted in Case I. The calcifications are not deposited in the parenchyma of the kidney, and the erect roentgenogram clearly demonstrates the calcium-fluid levels. The variation in height and length of these collections is due to the trapping of the fine calculi in the dilated pelvis, major and minor calyces. Since there was some free communication

between parts of the collecting system, a decubitus roentgenogram might well have shown a different layering pattern. Regardless of this, the diagnosis of milk of calcium in a hydronephrotic kidney is unequivocal.

SUMMARY

1. A tenth case of milk of calcium in a calyceal diverticulum is reported.

2. The clinical and roentgenographic features of milk of calcium in calyceal diverticula or cysts are discussed.

3. The coexistence of infection or other renal calculi is invariably the cause of symptoms.

4. An interesting and unusual case of milk of calcium in a hydronephrotic kidney is also reported.

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Acknowledgement is made to Captain Richard D. Wolfe, USAF MC for his assistance in preparation of the Case 1 report.

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# MILK OF CALCIUM IN A RENAL CALYCEAL DIVERTICULUM*

## CASE REPORT AND REVIEW OF LITERATURE

*By* MURRAY A. ROSENBERG, M.D.
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IN 1948, Holm[4] writing on "pyelogenic renal cyst" presented a case which demonstrated layering of a calcium-laden sediment in the cyst. In 1959, Howell[5] first reported the entity as "milk of calcium renal stone;" his patient had had a history of repeated urinary tract infections. Walker, Pearson and Johnson,[9] in 1960, described a calyceal diverticulum with milk of renal stone as an incidental finding at autopsy. Benendo and Litwak,[1] in 1964, cited the first surgical confirmation of this entity, in a patient with a history of urinary calculosis.

Six cases of "milk of renal calcium cyst" have been reported since Holm's original observation (Table 1). The present case represents the seventh reported case and the second instance of *in vivo* surgical and histologic confirmation. Of all cases reported to the present time, half were discovered as incidental findings.

### REPORT OF A CASE

K.S. #1034-59. A 55 year old white female was admitted to the Bridgeport Park City Hospital January, 1959, because of left flank pain radiating to the groin and gross hematuria. She reported having seen several "pieces of gravel" in the urine on the morning of admission. On physical examination there was guarding and tenderness over the left upper and mid abdomen and over the suprapubic area. Left costovertebral angle tenderness was noted. The patient related of having passed kidney stones 2 years previously.

The admission urine showed an albumin of 4+ and was grossly bloody. The uric acid was 6.4 mg. per cent; blood calcium 9.5 mg. per cent; blood urea nitrogen 14 mg. per cent. A white blood cell count was 20,300, with 85

TABLE I

REPORTED CASES OF MILK OF CALCIUM RENAL CYST

| Author | Surgery | Histology | Signs and Symptoms |
|---|---|---|---|
| Howell[5] | No | No | Pyuria, tenderness, recurrent urinary tract infection |
| Walker, Pearson and Johnson[9] | No | Transitional epithelium | Incidental finding on autopsy |
| Pullman and King[8] | No | No | Pyuria, flank pain |
| Benendo and Litwak[1] | Yes | Transitional epithelium | History of urinary calculi, associated calculosis |
| Maurer and Wildin[6] | No | No | Incidental finding |
| Henken[3] | No | No | Incidental finding |
| Rosenberg (present case) | Yes | Transitional epithelium | Costovertebral angle tenderness and flank pain, gross hematuria |

* From The Department of Radiology, Park City Hospital, Bridgeport, Connecticut.

per cent polymorphonuclears; urine culture showed *E. coli*, and was negative for acid fast organism.

Scout roentgenogram of the abdomen demonstrated an amorphous collection of calcifications in the region of the upper pole of the left kidney, and a long linear calcification in the left pelvic area (Fig. 1).

Intravenous urography revealed normal calyces and pelves. The collection of calcifications noted on the scout roentgenogram maintained persistent relationship to the left superior calyx (Fig. 2); the linear calcification noted on the plain roentgenogram in the left pelvis was shown to lie within the left lower ureter (Fig. 3). Retrograde pyelography showed filling of a large cavity apparently communicating with the left upper calyx (Fig. 4). Erect and supine roentgenograms of the stomach taken during gastrointestinal series showed the collection of calcifications to assume a "half moon" shaped density (Fig. 5 and 6). The patient was subsequently explored because of her past history and presenting symptoms.

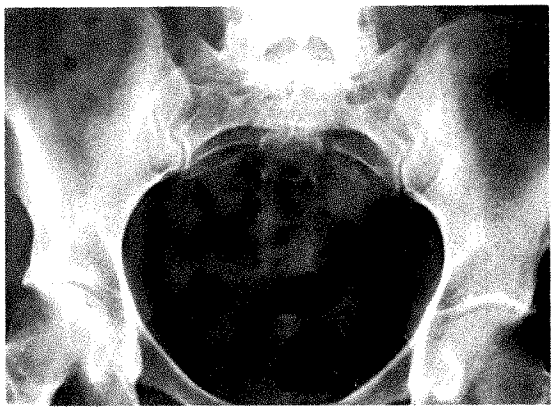


FIG. 1. Scout roentgenogram of the pelvis demonstrates the long curvilinear calcification in the region of the lower ureter on the left (arrow).

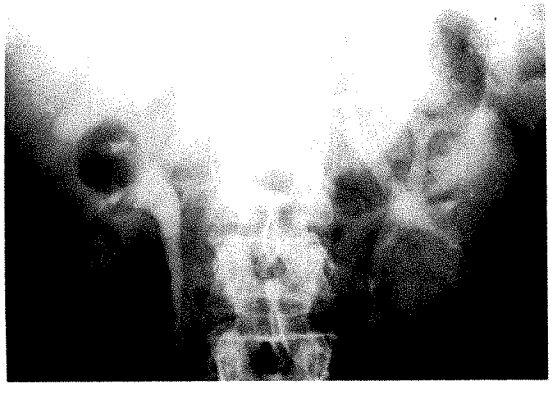


FIG. 2. Intravenous urogram demonstrates the collection of amorphous calcifications adjacent to the superior calyx on the left. (The calcifications are seen through the gas in the stomach.)

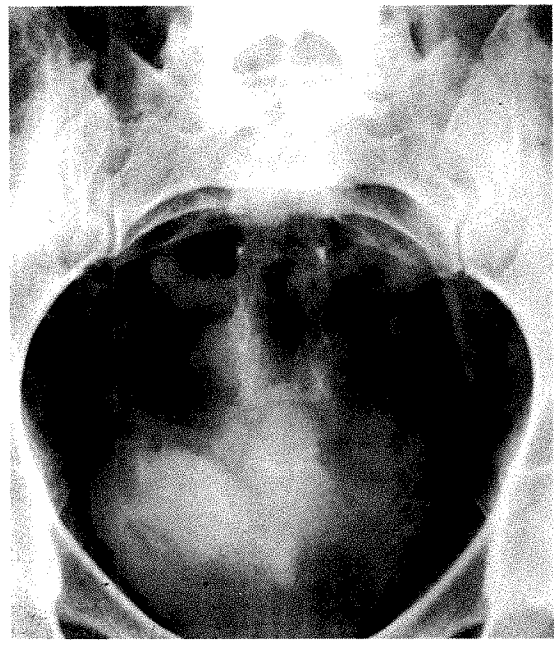


FIG. 3. The lower left ureter as seen on intravenous urogram. The calcifications noted on the scout roentgenogram of the pelvis are seen to lie within the left lower ureter.

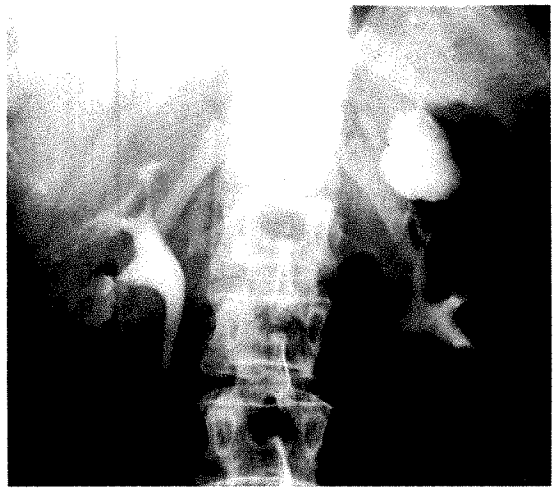


FIG. 4. Retrograde pyelogram shows filling of the diverticulum, demonstrating a patent connection with the superior calyx. The upper urinary tracts are otherwise normal.

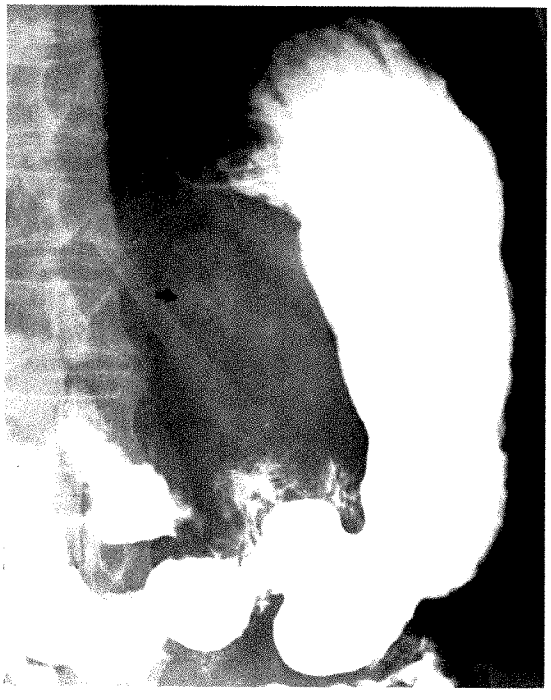


FIG. 5. Prone oblique roentgenogram of the stomach shows incidentally the amorphous collection of calcifications situated in the upper pole of the left kidney (arrow).

At surgery a large calyceal diverticulum was found protruding from the upper pole of the left kidney. The cavity was opened and cloudy, whitish liquid containing multiple black calculi was aspirated. The pathologic report was "pyelogenic cyst lined by transitional urinary epithelium and showing chronic inflammation" (Fig. 7). There were multiple hard, black-brown pellets about 1 mm. in diameter with smooth shiny surface. A test for calcium and phosphorus was positive.

The calcifications noted in the left side of the pelvis within the left ureter probably represented inspissated "milk of calcium" passed from the diverticulum. The material appears to have formed a cast of the lower ureter.

The patient has been seen subsequently on two occasions for unrelated medical problems at which time she denied any urinary symptoms. Repeat urography and urine analysis have been normal.

## DISCUSSION

Prather[7] first used the term calyceal diverticulum to describe a cavity connected to a minor calyx and lined with epithelium similar to the adjacent calyx. The diverticulum results from a developmental defect of the wolfian ducts. The communication which exists between the adjacent normal collecting system and the diverticulum is usually very narrow and may be completely obliterated. The diverticulum contains a viscid liquid suspension of calcium carbonate and calculi may be present within the suspension.[10] The communication between the calyceal system and the diverticulum may or may not be demonstrated on intravenous urography. The diverticulum will fill on retrograde studies if sufficient patency of the communicating channel is present. The diagnosis is often suspected by noting layering of the "milk of calcium" on an erect

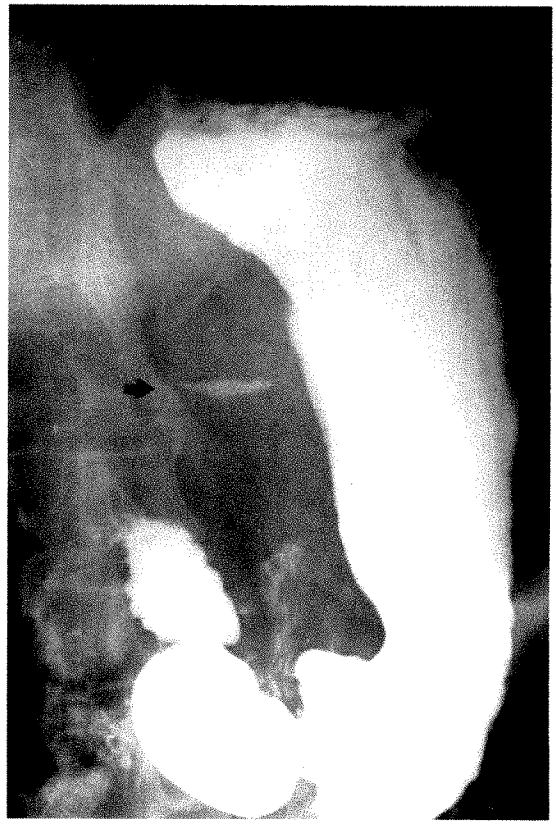


FIG. 6. The erect oblique roentgenogram demonstrates a typical "half-moon" shaped configuration in the upper pole of the left kidney, representing layering of the calcium sediment, and in the diverticulum.



Fig. 7. Histologic section of diverticulum (× 300). Transitional (urinary) epithelium with inflammatory reaction in the wall.

roentgenogram and it is confirmed by contrast study. Howell[3] remarked that a decrease in the amount of calcium provided proof of communication between the excretory system and the diverticulum.

The finding of a calyceal diverticulum is incidental in the asymptomatic patient. In such cases further management is unnecessary.[3] However, with stasis, infection may occur with resultant urinary tract infection[2] (pyuria, fever, flank pain, and possible hematuria). In such cases, surgery is indicated. The hematuria when present is usually caused by calculi formed within the diverticulum.

The simple parenchymal cyst arises from metanephric tissue and is readily differentiated from renal calyceal diverticulum[4] (Table II).

## SUMMARY

1. The patient demonstrating "milk of calcium" in a calyceal diverticulum may present with urinary tract infection and/or hematuria. In such cases surgery is indicated.

2. The present case concerns a patient with gross hematuria and urinary tract infection. Surgical removal resulted in cure.

Table II

COMPARATIVE SIGNS OF CALYCEAL DIVERTICULUM AND SIMPLE SOLITARY CYST

| Calyceal Diverticulum (pyelogenic cyst) | Simple Solitary Cyst |
|---|---|
| 1. Arises from wolfian duct | Arises from metanephric tissue |
| 2. Communicates with calyx (may be obliterated) | Does not communicate with calyx |
| 3. Wall of diverticulum is lined with transitional urinary epithelium | Cyst wall is composed of fibrous tissue with single layer of endothelium |
| 4. About 50 per cent contain stones | Never forms stones |
| 5. Contains thick, viscid fluid | Contains thin, serous fluid |

3. About half of the reported cases (including Holm's Case 2) were described as incidental findings.

4. This is the second instance of surgical and histologic confirmation of this condition *in vivo*.

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Vol. 101, No. 3

# RENAL VASCULAR IMPRESSIONS*

## INCIDENCE AND CLINICAL SIGNIFICANCE

*By* ROBERT A. NEBESAR, M.D.,† JAMES J. POLLARD, M.D.,† *and* ELWIN E. FRALEY, M.D.‡

THE renal collecting system frequently shows extrinsic impressions caused by renal arteries or renal veins.[1,6–8] Renal vascular impressions were considered to be a roentgenographic finding without physiologic or clinical significance until recently, when nephralgia and persistent superior infundibular obstruction producing localized caliectasis were shown to be caused by extrinsic vascular compression of the superior major calyx.[2,3]

The purpose of this report is to further document the frequency and location of renal arterial impressions and to describe the roentgenographic findings in patients with clinically significant intrarenal obstruction caused by crossing vessels.

### METHODS

A review was made of 150 consecutive renal angiograms obtained by the percutaneous Seldinger technique with serial film recording. The angiograms were evaluated together with the intravenous urograms to determine the incidence of arterial impressions and the arteries causing them. Thirty-five cases were excluded because an adequate intravenous pyelogram was not available or because the intrarenal collecting system was distorted by a renal mass. Of the remaining 115 cases, 81 were roentgenographically normal and 34 had the following diseases: arterial stenosis (12); renal cyst or tumor (10); hydronephrosis (4); nephrosclerosis (3); renal artery aneurysm (2); pyelonephritis (2); and hypoplastic kidney (1).

In addition to the above 115 cases, the intravenous and retrograde pyelogram and angiograms were evaluated in 2 patients with nephralgia and superior infundibular obstruction who were being studied while the review was in progress.

### RESULTS

Arterial impressions occurred in 18 per cent of all cases and were approximately 3 times more frequent on the right side than on the left. Although there was usually only a single impression, 2 or more were seen in 7 kidneys (Table I). The ventral artery or its branches were the most frequent causes of arterial filling defects. Arterial impressions were more common upon the renal pelvis (15) and superior infundibulum (12) than upon the inferior infundibulum (1) or ureteropelvic junction (1), (Table II and Fig. 1, *A* and *B*).

In only 1 patient of the 115 unselected cases did an artery appear to cause roentgenographically significant obstruction. A patient with stress incontinence had an intravenous pyelogram which suggested that there was a right renal mass associated with narrowing of the right superior

TABLE I

GENERAL INCIDENCE OF ARTERIAL IMPRESSIONS

| | |
|---|---|
| Total patients | 115 |
| Patients with impression | 21 |
| Total kidneys with impression | 22 |
| Right | 15 |
| Left | 5 |
| Bilateral | 1 |
| Kidneys with single impression | 15 |
| Kidneys with two impressions | 6 |
| Kidneys with multiple impressions | 1 |

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TABLE II

LOCATION OF IMPRESSION ON THE COLLECTION STRUCTURES AND ARTERY CAUSING IMPRESSION

| | No. |
|---|---|
| *Pelvis* | 15 |
| Ventral artery or branch | 12 |
| Dorsal artery or branch | 2 |
| Indeterminate | 1 |
| *Superior Infundibulum* | 12 |
| Ventral | 6 |
| Dorsal | 1 |
| Both ventral and dorsal | 2 |
| Indeterminate | 3 |
| *Inferior Infundibulum* | 1 |
| Dorsal | 1 |
| *Ureteropelvic Junction* | 1 |
| Ventral | 1 |

infundibulum and caliectasis. Renal angiography showed that the central branch of the ventral artery compressed the infundibulum and appeared to cause moderate obstruction. Because the patient was asymptomatic and had sterile urine, she is being observed.

Two patients with nephralgia and superior infundibular obstruction were admitted to the hospital while the review was in progress. Both patients had unexplained long-standing flank pain, and one had a persistent urinary tract infection. The roentgenographic findings were similar in both cases: there was a persistent narrowing of the superior infundibulum, superior caliectasis, interference with peristalsis in the infundibulum on the cine intravenous pyelogram, retention of contrast medium in the superior calyx on the delayed drainage roentgenograms of the retrograde pyelography, and on the angiogram an artery crossed and compressed the superior infundibulum at the point of narrowing. Since these patients had both symptoms

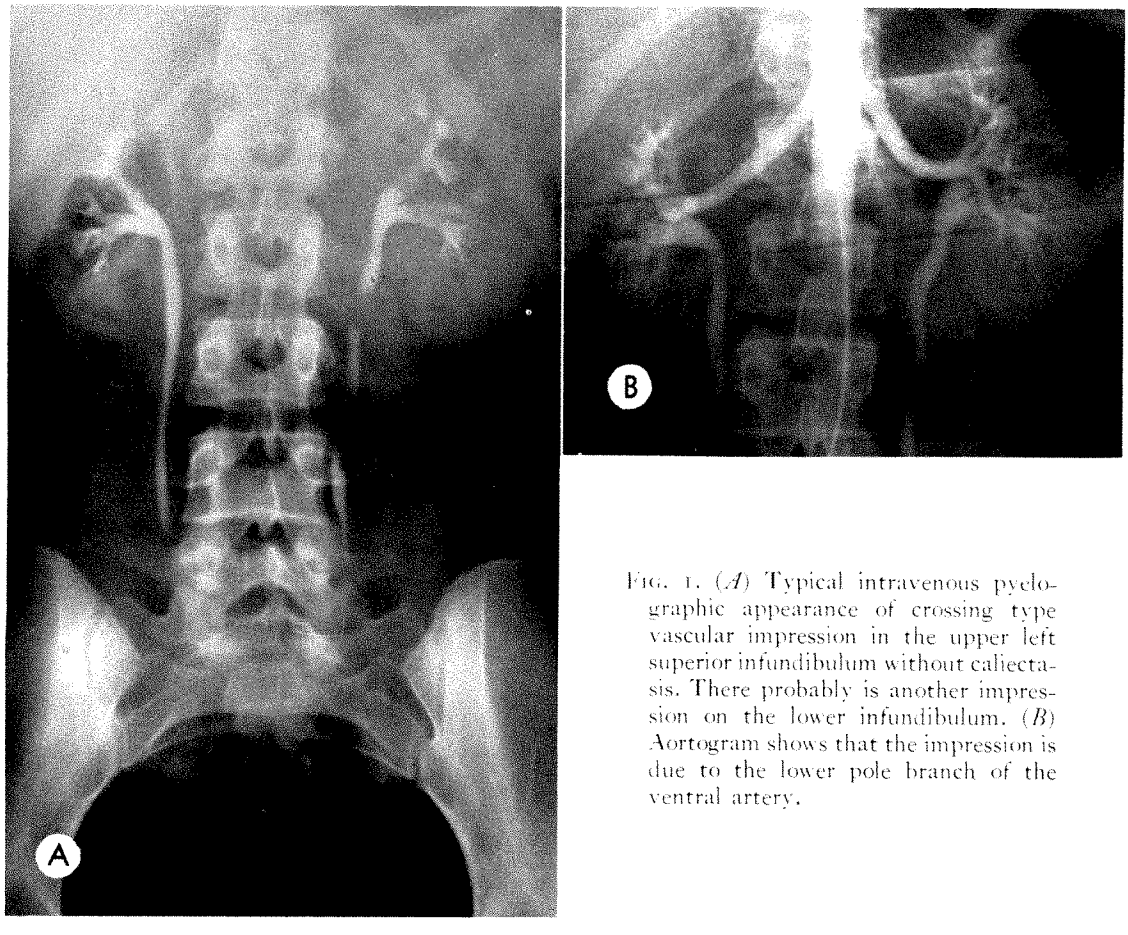



FIG. 1. (*A*) Typical intravenous pyelographic appearance of crossing type vascular impression in the upper left superior infundibulum without caliectasis. There probably is another impression on the lower infundibulum. (*B*) Aortogram shows that the impression is due to the lower pole branch of the ventral artery.

and significant roentgenographic findings, they were explored and were found to have a vessel obstructing the superior infundibulum. The patients were relieved of symptoms and infection following surgical removal of the obstruction (Fig. 2, *A–C*; and 3, *A–E*).

## DISCUSSION

Renal arterial impressions may appear as a well defined transverse linear defect crossing the pelvis or infundibulum or as a concave indentation upon the pelvis or infundibulum. A venous impression appears as a wide transverse band, usually flattening the entire upper infundibulum.[7] Vascular impressions frequently disappear completely with distention of the collecting structures.[6]

Vascular impressions can usually be distinguished on the pyelogram from abnormalities that extrinsically compress or narrow the proximal collecting system. Although a small tumor or cyst could be confused with a concave unilateral type of vascular compression defect, these intrinsic renal masses generally cause a more broad area of compression and produce displacement of the collecting system. Frequently, renal tumors or cysts also cause deformities

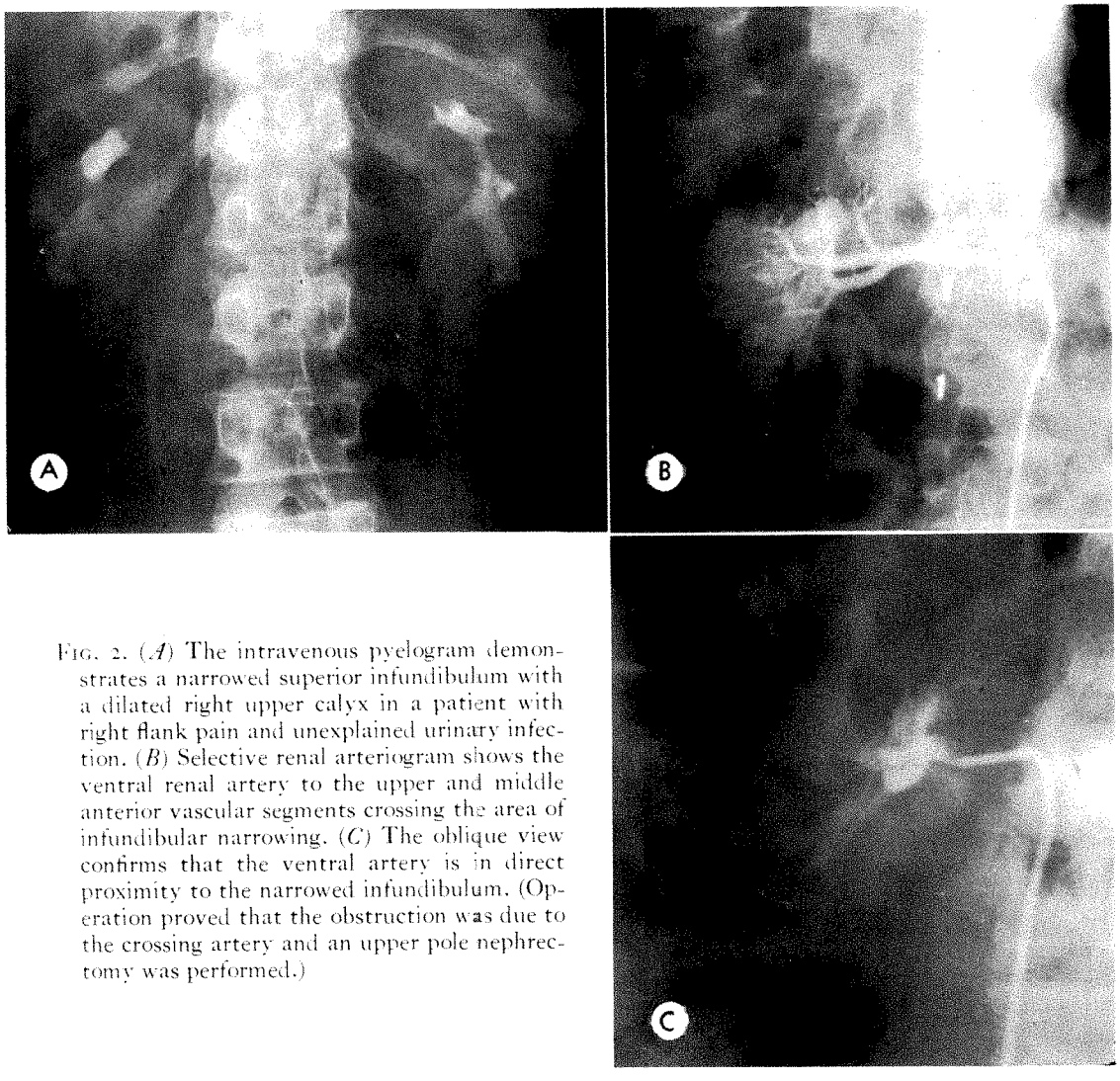



FIG. 2. (*A*) The intravenous pyelogram demonstrates a narrowed superior infundibulum with a dilated right upper calyx in a patient with right flank pain and unexplained urinary infection. (*B*) Selective renal arteriogram shows the ventral renal artery to the upper and middle anterior vascular segments crossing the area of infundibular narrowing. (*C*) The oblique view confirms that the ventral artery is in direct proximity to the narrowed infundibulum. (Operation proved that the obstruction was due to the crossing artery and an upper pole nephrectomy was performed.)

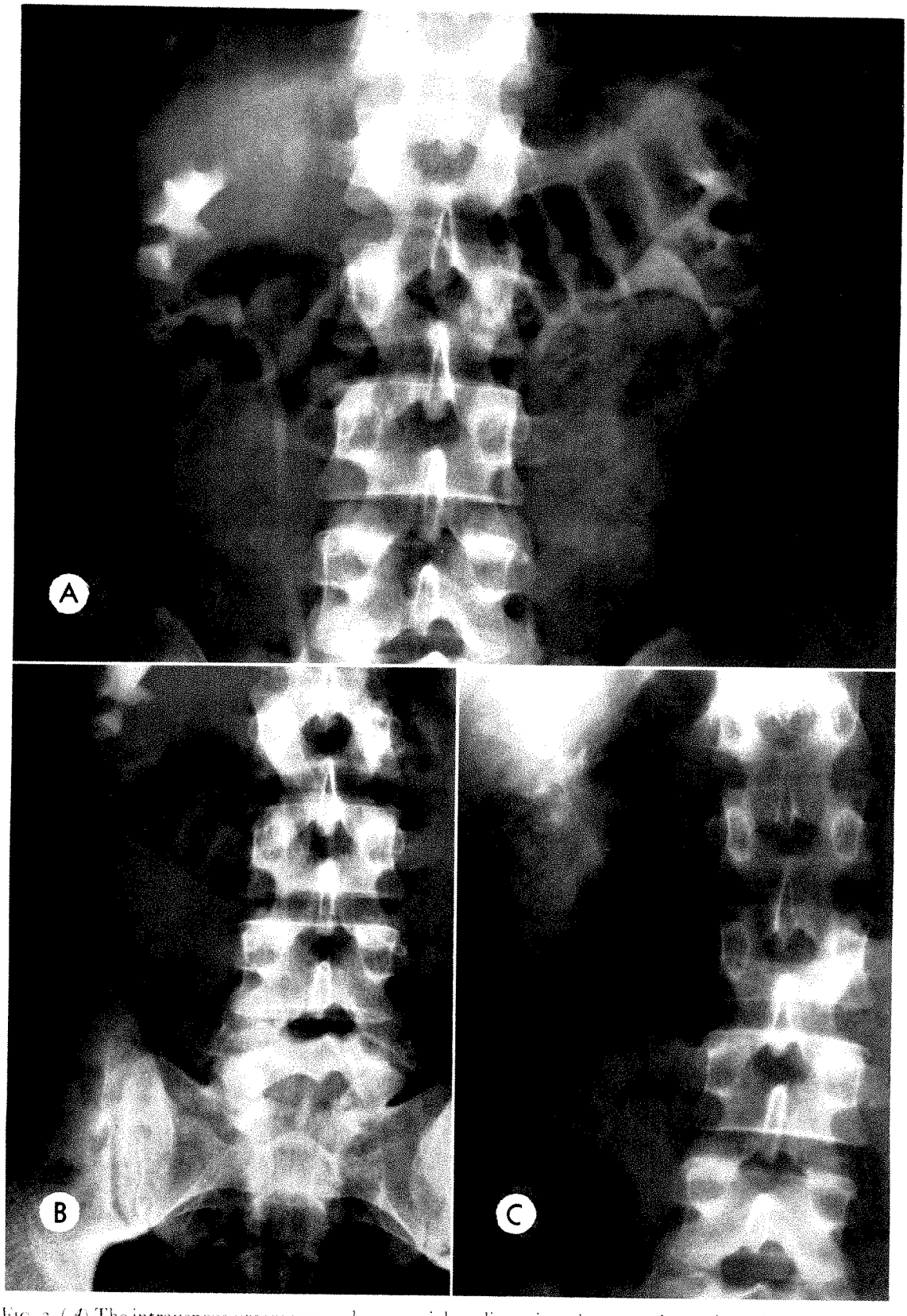



FIG. 3. (*A*) The intravenous urogram reveals upper right caliectasis and narrowed superior infundibulum in a patient with right flank pain. (*B*) Immediately after retrograde pyelography there is marked retention of contrast material in the dilated upper right calyx. (*C*) Retention of contrast material is shown on the 20 minute upright drainage roentgenogram after retrograde pyelography.

of the minor calyces which are not produced by renal vessels.

Vascular impressions also must be differentiated from intrinsic abnormalities of the collecting structures. The extrinsic unilateral smooth compression or transverse impression of an arterial defect is usually easily differentiated from the sharply marginated intraluminal filling defect produced by calculus or blood clot. Epithelial carcinoma is usually papillary and presents as a fixed irregular intraluminal filling defect, but when infiltrating it may present as a long stricture. Benign strictures, often secondary to tuberculosis, narrow the infundibulum concentrically over a longitudinal segment.

Previous reports have noted a higher incidence (20 and 42 per cent)[1,6] of vascular impressions than was found in this series (18 per cent). The fact that one or both oblique projections were used in addition to the anteroposterior projection in many cases may partially account for the lower incidence in our series, since oblique renal angiograms sometimes excluded a vessel as cause of an apparent compression defect (Fig. 4*C*). Two projections are necessary to establish with certainty that a given vessel is in apposition with a collecting structure and is thus responsible for an impression.

Although vascular impressions are common, usually they are of no clinical importance. Insignificant vascular impressions may even be associated with asymptomatic caliectasis. However, when there is roentgenographic evidence of obstruction at the site of a persistent vascular impression in a patient with symptoms or, particularly with an otherwise unexplained infection, a more thorough work-up is indicated (Table III).

The following roentgenographic criteria must be present on the pyelogram and the angiogram in order to establish the diagnosis of vascular obstruction. The narrowing, irrespective of its location, must be persistent and must cause proximal dilatation of the affected part. Delayed drainage roentgenograms following retrograde pyelography should show entrapment of contrast material behind the obstruction even after

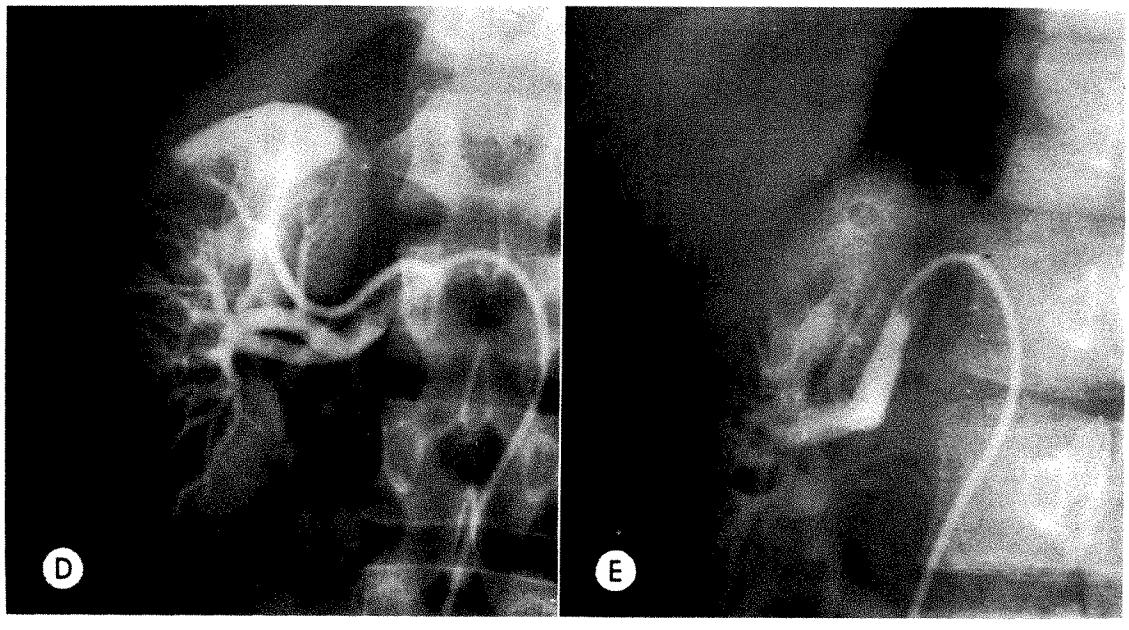



Fig. 3. (*D*) Selective arteriogram in anteroposterior projection demonstrates the ventral artery crossing the infundibulum at the point of narrowing. (*E*) The oblique view of the arteriogram confirms the apposition of the artery to the infundibulum. (Operation proved that the obstruction was due to the vessel and an infundibuloplasty was performed.)

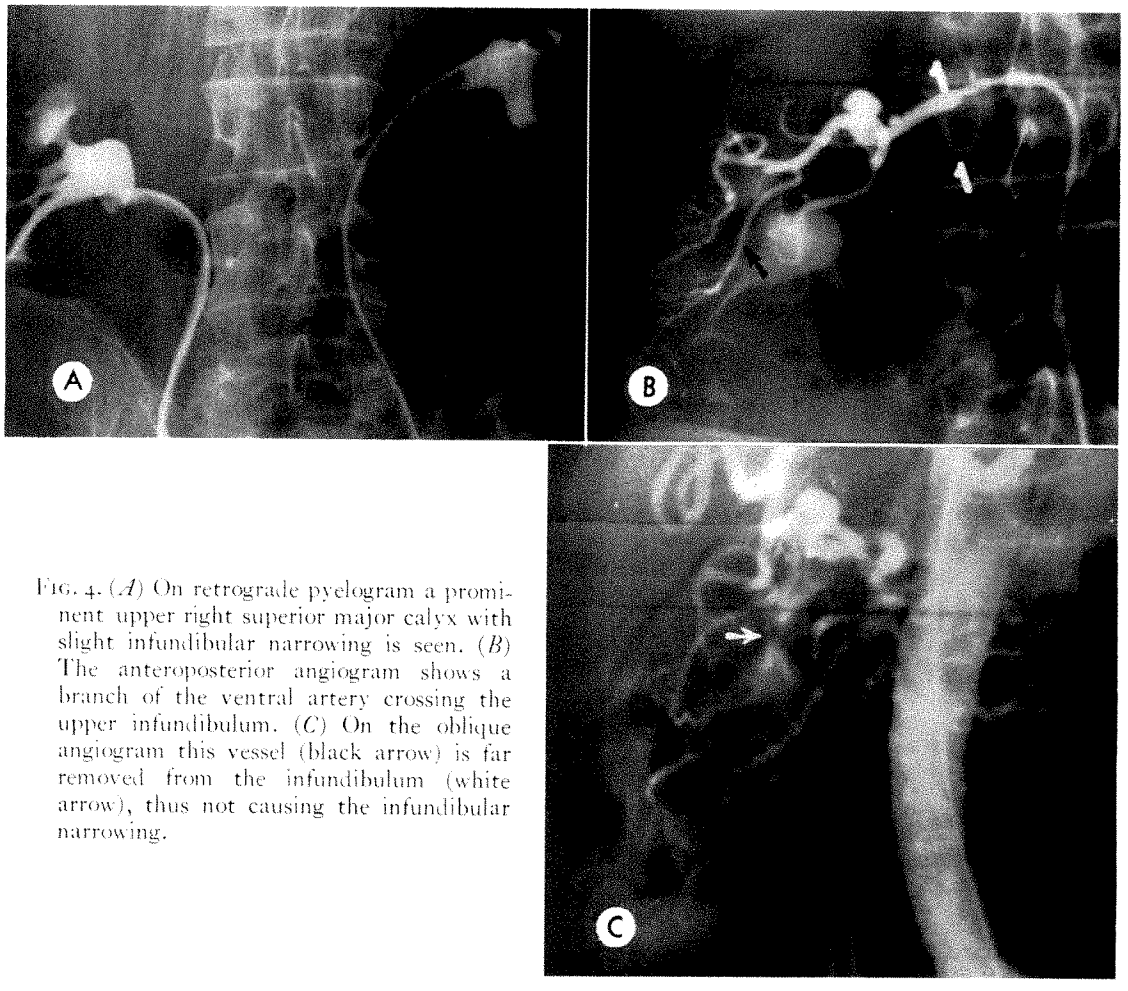



FIG. 4. (*A*) On retrograde pyelogram a prominent upper right superior major calyx with slight infundibular narrowing is seen. (*B*) The anteroposterior angiogram shows a branch of the ventral artery crossing the upper infundibulum. (*C*) On the oblique angiogram this vessel (black arrow) is far removed from the infundibulum (white arrow), thus not causing the infundibular narrowing.

the patient has been upright for as long as 30 minutes. Cine roentgenography should demonstrate interference with peristalsis at the site of vascular obstruction. Furthermore, the narrowing of the collecting structures must conform to the location of the artery as seen in 2 projections on an angiogram.

TABLE III

WORK-UP FOR VASCULAR IMPRESSION

1. *Roentgenographic*
   a. Intravenous pyelogram
   b. Retrograde pyelogram
   c. Cine intravenous pyelogram
   d. Renal arteriogram with oblique projection
   e. Renal venogram
2. *Urologic*
   a. Complete stone work-up
   b. Urine cytologies
   c. Careful evaluation to rule out genitourinary tuberculosis
   d. Endoscopy (b above)

Angiography is helpful both to demonstrate that an artery crosses the filling defect and to rule out other causes of narrowing such as tumor, cyst, or venous defect. The angiographic study should include an aortogram to visualize any aberrant or multiple renal arteries. Selective angiography of the renal arteries in both anteroposterior and oblique projections will best show the relationship of the renal arteries to the collecting structures and rule out a parenchymal renal lesion. When infundibular obstruction is present and the narrowing is in apposition to a vessel, it is reasonable to assume obstruc-

tion by the vessel, although, until surgical exploration is performed, an intrinsic lesion such as stricture cannot be excluded.

The renal veins occasionally will be adequately shown on the renal arteriogram. However, concentration of contrast agent within the veins is usually inadequate or masked by the overlying nephrogram. When a vein is suspected as the cause of an impression, a selective renal vein catheterization from a femoral vein (using a Seldinger technique) should be performed while the arterial catheter is still selectively in the renal artery. Since renal blood flow is rapid, a selective injection into the renal vein often will not fill it completely because blood washes out the contrast agent. Good filling of the renal vein can be obtained when renal venous blood flow is reduced by injection of 10 to 15 micrograms of epinephrine through the arterial catheter into the renal artery (Fig. 5, *A–C*).

The most common site of both benign vascular impressions and intrarenal ob-

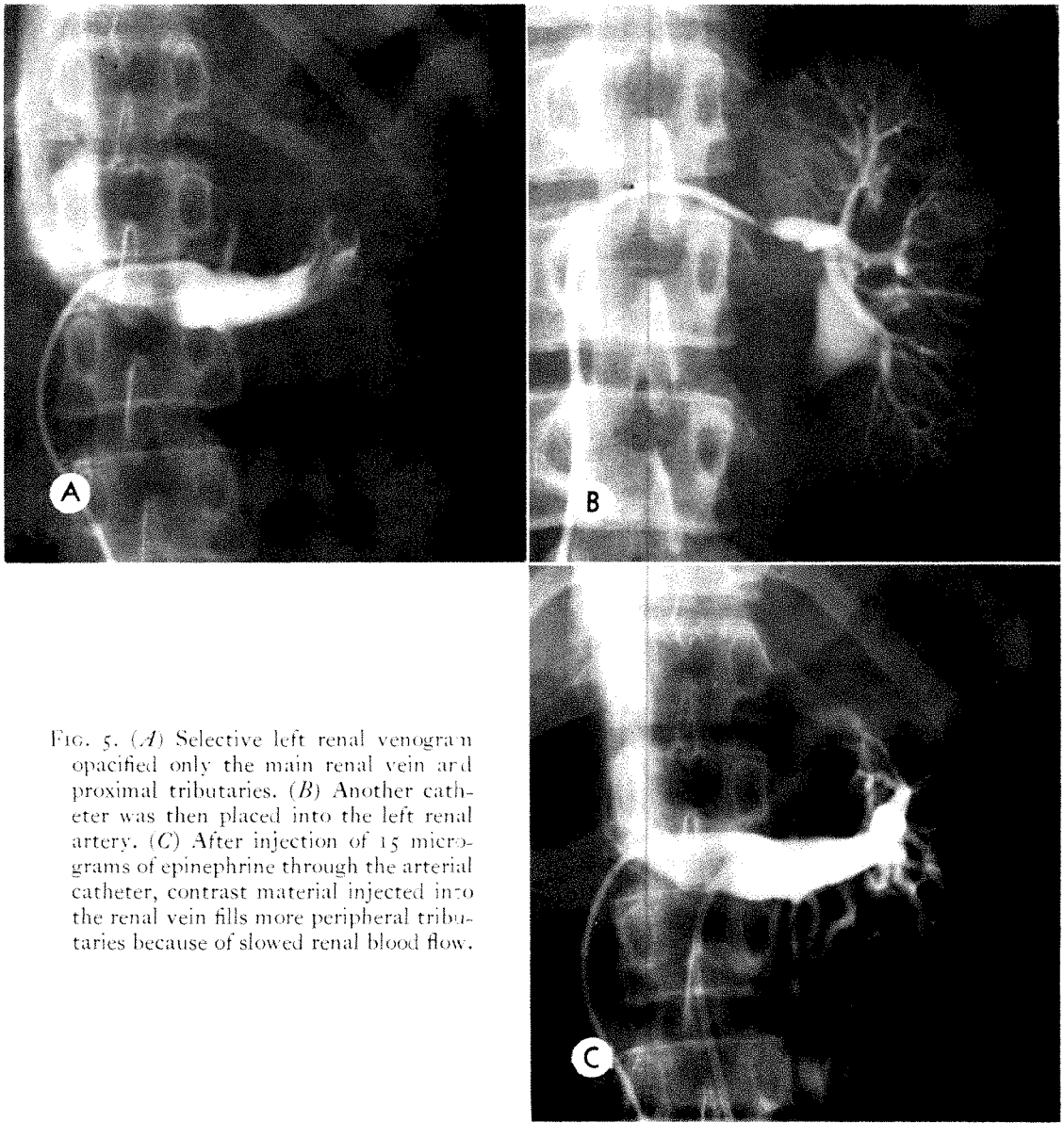



FIG. 5. (*A*) Selective left renal venogram opacified only the main renal vein and proximal tributaries. (*B*) Another catheter was then placed into the left renal artery. (*C*) After injection of 15 micrograms of epinephrine through the arterial catheter, contrast material injected into the renal vein fills more peripheral tributaries because of slowed renal blood flow.

structions due to renal vessels is the right superior infundibulum. In the past 2 years 10 cases of clinically significant vascular obstruction of the superior infundibulum have been documented.[2–6,9]

Obstruction of the infundibulum is rare despite the frequency of vascular impressions. Slight obstruction may be present without clinical significance. It is only when obstructions cause symptoms and especially infection that they should be regarded as clinically significant.

Although the present study and previously published findings[1,6] attest to the high incidence of vascular impressions in both the superior infundibulum and pelvis, we have not observed significant vascular obstruction of the renal pelvis. Therefore, it is reasonable to presume that the superior infundibulum is more prone to obstruction by crossing vessels than is the pelvis. The infundibulum is narrow anatomically and is intrarenal and thus immobile, while the pelvis is wide and redundant and usually extrarenal. Although the mechanism by which renal vessels cause intrarenal obstruction is not always certain, either abnormal renal mobility or unusual vascular anatomy or both were present in all cases.[2,8] The right kidney was involved more often than the left. It is the right kidney which more frequently shows increased mobility and ptosis.

All of the patients with surgically proven vascular obstruction of the superior infundibulum had nephralgia. Pain probably resulted from overdistention of the calyx and its surrounding smooth muscle. However, in addition, 4 of the 6 patients who were operated upon in this hospital had urinary tract infection, and 2 patients who are currently being considered for operation are infected. It is therefore possible that arterial obstructions of the proximal collecting system can cause enough residual urine to be an important factor in the initiation and perpetuation of severe urinary tract infections.

In the past, patients with clinically significant obstruction of the superior infundibulum were treated by upper pole nephrectomy. Recently, however, more conservative surgical procedures such as either division of the offending vessel or repositioning of the infundibulum have relieved these obstructions. All patients who have been operated upon are asymptomatic, and those who were infected now have sterile urine. A more detailed description of treatment can be found elsewhere.[2,8]

SUMMARY

Arterial impressions were seen in 18 per cent of 115 unselected angiograms and were 3 times more frequent on the right side. Arterial impressions were more common in the superior infundibulum and renal pelvis than in other portions of the collecting system. In only 1 case of the 115 unselected cases did there appear to be any significant caliectasis in association with an arterial impression.

Renal vascular impressions usually are insignificant but occasionally may be associated with symptomatic and clinically significant intrarenal obstruction. Two cases with nephralgia and proven superior infundibular obstruction due to crossing vessels are reported. A persistent vascular impression in the superior infundibulum is significant if there is also localized caliectasis and retention of contrast material on delayed drainage roentgenograms of retrograde pyelography in a symptomatic or infected patient with no other cause for infection. Cineroentgenographic evidence of interference with peristalsis should also be seen at the site of obstruction. If the area of narrowing is in apposition to a renal vessel on both anteroposterior and oblique angiograms, the vessel is assumed to be the cause of the obstruction, but surgical confirmation is necessary to exclude obstruction due to other causes.

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# RENAL DUPLICATION WITH HYDRONEPHROTIC SEGMENT*

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URETERAL duplication, one of the commonest urinary tract anomalies, is often associated with duplication of the renal pelvis and parenchyma. It is three times as common in women as in men and occurs with equal frequency on the right and the left side. The upper segment usually represents about 15 to 20 per cent of the renal substance and is frequently involved pathologically.[1,5,6]

The diagnosis of an asymptomatic renal duplication can usually be made by urography alone. The symptomatic duplication associated with conditions such as hydronephrosis, ectopic ureter, and ureteral reflux may require a complete urologic evaluation including cystourethrography, urography, cystoscopy with retrograde pyelography, and careful inspection of the perineum.[6]

A nonfunctioning renal segment may present a diagnostic problem. Urograms of such patients have on occasion been interpreted as normal or as cyst or tumor of the upper pole of the kidney.[1,2,3,5] In 2 recent cases selective renal arteriography was instrumental in establishing the correct diagnosis.

## REPORT OF CASES

CASE 1. J.T., a woman, aged 27, was admitted on June 18, 1964 with right flank pain, tender-

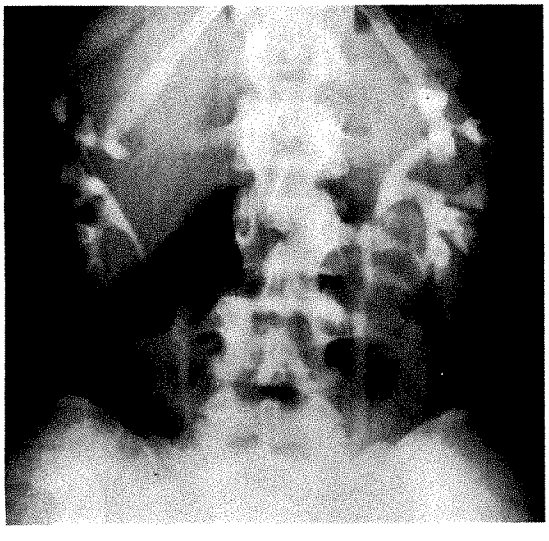


FIG. 1. Case 1. Urogram demonstrating an upper pole mass on the right side with displacement of the calyceal system laterally. There is sharp angulation of the ureter at the junction of its upper and middle thirds.

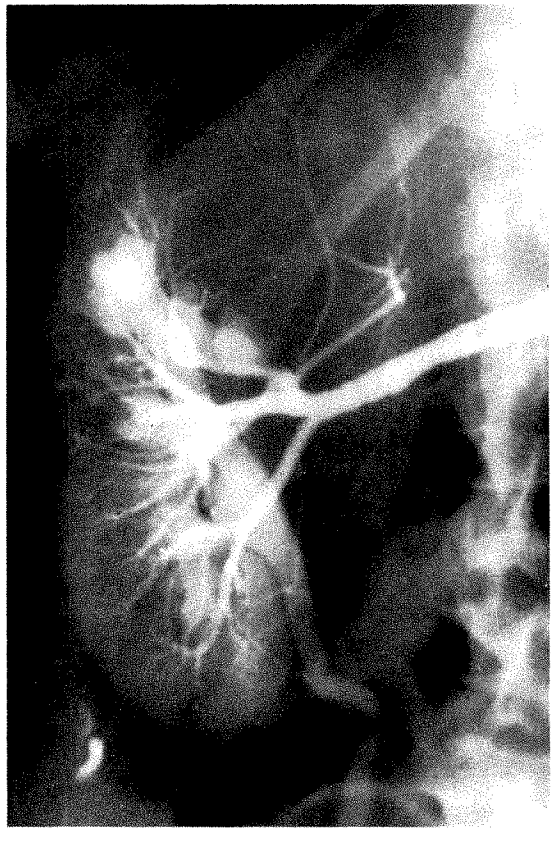


FIG. 2. Case 1. Arterial phase of a selective renal arteriogram demonstrating the diminished vascular supply to the upper half of the kidney and stretched narrowed vessels with deficient arborization.

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ness, and low grade fever. Urography appeared to demonstrate a mass in the upper pole of the right kidney, displacing the calyceal system laterally. The junction of the upper and middle thirds of the ureter was sharply angulated (Fig. 1). Selective renal arteriography demonstrated the vascular supply of the upper half of the kidney to be diminished and to consist of stretched narrowed vessels with deficient arborization (Fig. 2). The nephrogram of the same area was reduced and represented only by a faint rim of opacification (Fig. 3).

These findings suggested a duplicated kidney and prompted the search for the ectopic ureter which terminated in the urethra. On July 9, 1964 a right upper pole heminephrectomy with upper third ureterectomy was performed. A postoperative urogram showed reorientation of the calyces, pelvis, and ureter without the ureteral angulation seen preoperatively (Fig. 4).

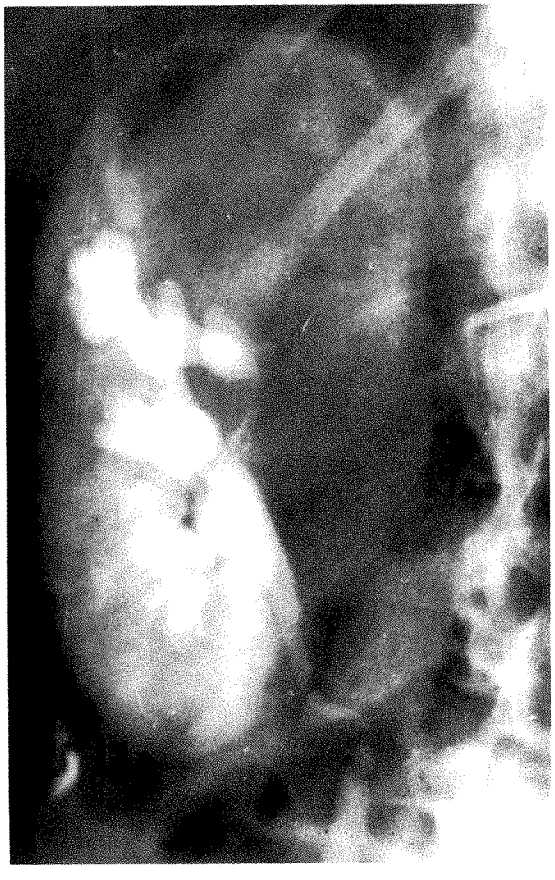


Fig. 3. Case I. The nephrogram phase of a selective renal arteriogram demonstrating a poor nephrogram sharply delineated by a faint rim of opacification.

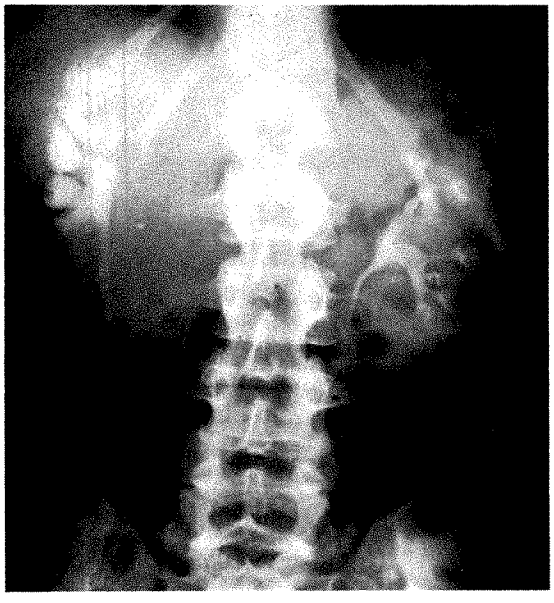


Fig. 4. Case I. Postoperative urogram showing reorientation of the calyces, pelvis, and ureter without the abnormal ureteral angulation seen preoperatively.

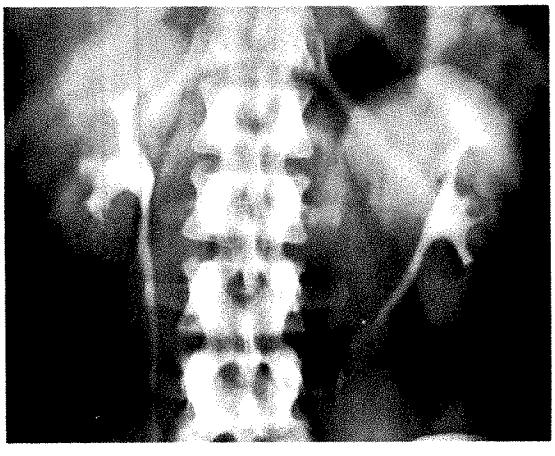


Fig. 5. Case II. Nephrotomogram demonstrating the left upper pole enlargement and abnormal ureteral angulation.

Case II. C.C., a woman, aged 34, with a 6 year history of recurrent flank pain and pyuria was admitted on July 21, 1966. Examination revealed a tender left flank mass. Urography and nephrotomography showed enlargement of the left upper pole of the kidney and angulation of the junction of the upper and middle thirds of the ureter similar to that demonstrated in the first case (Fig. 5). A selective left renal arterio-



FIG. 6. Case II. Selective renal arteriogram showing arterial deficiency with attenuation of the vessels of the upper portion of the left kidney.

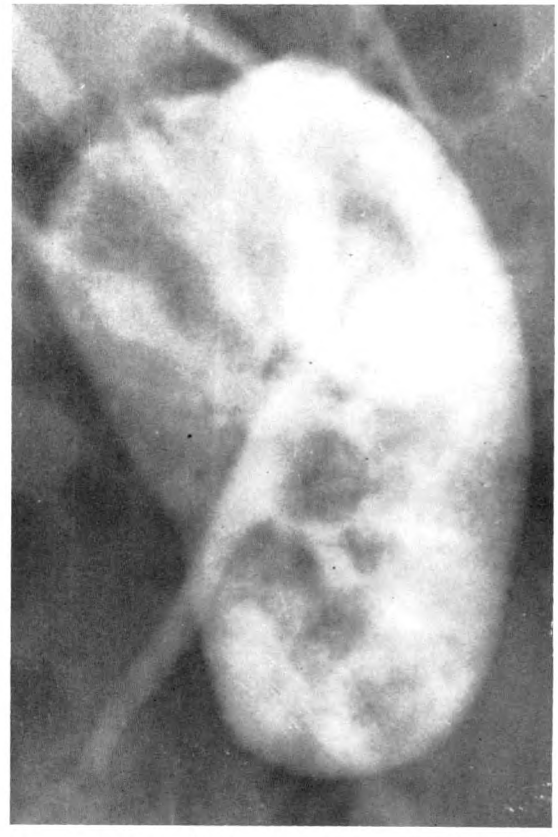


FIG. 7. Case II. Nephrogram phase of arteriogram demonstrating a relatively faint nephrogram of the upper portion of the kidney.

gram demonstrated arterial deficiency (Fig. 6) and a relatively faint nephrogram of the upper pole of the kidney (Fig. 7). On the basis of these findings, the diagnosis of a duplicated kidney with a nonfunctioning upper segment was made, and an unsuccessful search was made for a second ureteral orifice. On August 18, 1966 left renal exploration revealed an upper pole hydronephrosis and hydroureter. A left upper pole heminephrectomy with upper third ureterectomy was performed. The postoperative urogram demonstrated reorientation of the collecting system (Fig. 8).

## DISCUSSION

If a segment of a duplicated kidney is nonfunctioning, the abnormal element will not be demonstrated by urography. The urogram may appear normal, or the visualized collecting system may not be of characteristic shape. On occasion different roentgenologic appearances are obtained on the urogram and pyelogram since the urogram may show the functioning half of the duplication and the pyelogram the other.[1,2,4]

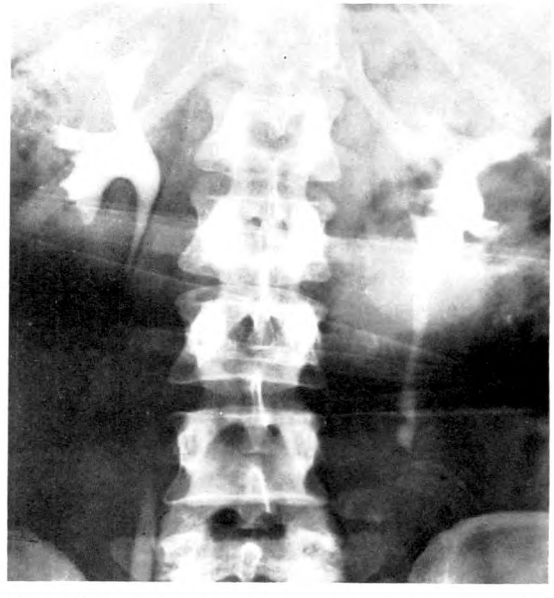


FIG. 8. Case II. Postoperative urogram demonstrating reorientation of the collecting system.

The possibility of duplication of the kidney should be considered if any of the following is noted on urography: (1) Excessive renal parenchyma without recognizable collecting system; (2) an abnormally small pelvis situated low in relation to the renal outline; (3) absence of one or more calyces;[1,2,3] and (4) abnormal angulation of the junction of the upper and middle thirds of the ureter.

The arteriographic findings of a renal mass in an area with no visible collecting system, a deficient nephrogram, and a blood supply consisting of a few small, thin stretched vessels are suggestive of renal duplication with a hydronephrotic segment and should stimulate the search for a second ureteral orifice.

SUMMARY

Renal duplication with a nonfunctioning hydronephrotic segment may lead to diagnostic difficulty and misinterpretation as tumor or cyst. Selective renal arteriography is an important adjunct to the diagnosis in such cases. The finding of a deficient arterial supply and a poor nephrogram phase in an area with no visible collecting system should suggest the diagnosis.

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# ANGIOGRAPHIC CHANGES IN THE ADRENAL GLANDS*

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THE value of the available roentgenographic methods for diagnosing pathologic alterations of the adrenal glands has been most discussed in recent years, and opinions vary widely. Among the more optimistic workers, Lang *et al.*[8] base their views on a series in which they used a good angiographic technique combined with the Ruiz Rivas[17] retroperitoneal gas injection. They considered it possible to distinguish between several pathologic conditions in the adrenals. On the other hand, Goodwin[5] is more cautious in this respect in his chapter on adrenal angiography in a recent textbook on angiography.

This difference of opinion prompted a study of the theoretic possibilities of visualizing vascular alterations in the adrenals by means of a suitable angiographic technique. The Department of Endocrinology at Karolinska Hospital, one of the few in this country, provided a suitable source of specimens from patients who had undergone clinical examination. The glands were examined by microangiographic and histologic techniques, and for some, comparison could be made with preoperative angiographic findings. Microangiography permits a quantitative evaluation of the vascular bed and examination of the width of individual vessels with regard to variations in the lumen and topographic features.[1] The specimens that had been examined with respect to vascular pattern were then submitted to histologic study. The microangiographic technique proved to be an ideal aid for ascertaining which vascular alterations can be visualized by clinical angiography and phlebography.

## MATERIAL

The material consisted of 16 primary adrenal tumors, 7 adrenal glands with metastases and 4 specimens of hyperplasia of the adrenal cortex.[3] Since the adrenal gland had not previously been examined by the technique in question, it was necessary for the sake of comparison to examine a normal series; such a study was performed on the vascular development of the adrenal gland in the fetus, the child and the adult.[6] In these earlier papers[3,6] the relevant clinical and angiographic data, technique and microangiographic and histologic findings were reported.

## DISCUSSION AND CONCLUSIONS

Lang *et al.*[8] considered "that a retroperitoneal gas study provides a most conclusive anatomic demonstration of a suprarenal mass and its topographic relationship to adjacent structures. The characteristic contour and appearance of the mass may frequently suggest a correct histologic diagnosis."[8] In anatomic studies, such as that carried out by Merklin and Michels,[11] it has, however, been found that the boundary of the normal adrenal gland is variable. In examining 90 patients by means of retroperitoneal pneumography, Fagerberg[4] found the result difficult to interpret, and held that a mistaken impression of the size of the gland was sometimes obtained because "it is not the suprarenal gland itself but the suprarenal space with its contained gland, fat, and connective tissue that is demonstrated by retroperitoneal pneumography." Our anatomic studies of the abnormal glands filled with contrast medium show how unreliable a diagnosis on the basis of contour can be. For instance, in a number of glands there were small adenomas, and also metastases of such extent as to suggest hyperplasia. In 2 glands there were metastases of mammary carcinoma that did not affect the contour

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or size. Nor was it always possible to distinguish between adenoma and pheochromocytoma. The claim of being able to make a correct histologic diagnosis by means of retroperitoneal pneumography would seem to be an exaggeration. At best it is possible to detect enlargement of one or both glands.

Merklin and Michels[11] have performed a statistical analysis of a large volume of data from the literature relating to the vascular supply of the kidneys and adrenals, and have themselves performed vascular dissections of these organs. They point out something that many authors have failed to observe, namely that the long accepted statement that the adrenals receive arteries from only three directions—the aorta, the renal and phrenic arteries—is incorrect. The adrenals can be supplied by from 10 to 60 branches of arteries to organs around the glands. The adrenal blood supply is never the same in any two bodies, or on opposite sides. Irrespective of which trunks supply the glands, these often have, according to the above authors, 3 locally defined branches—a superior, a medial and an inferior branch. From these a number of smaller branches are given off. While the glands are thus supplied by a number of arteries, they are drained by only 1 or 2 veins.[15]

Our microangiographic studies have shown that the normal adrenal has a characteristic vascular pattern. The arteries give off capsular and medullary branches which in their turn give off sinusoids at right angles. These empty into central medullary veins or further ramifications at right angles to the sinusoids. The central medullary veins join up to form a few draining veins.

In *cortical hyperplasia* the basic vascular pattern remains, but there is an increase not only in volume of the organ but also in the size and number of vessels. It should be possible to visualize this hypervascularity by clinical angiography (Fig. 1). Similarly, abnormally wide arteries were observed. In the hyperplastic areas there were no pathologic vessels.

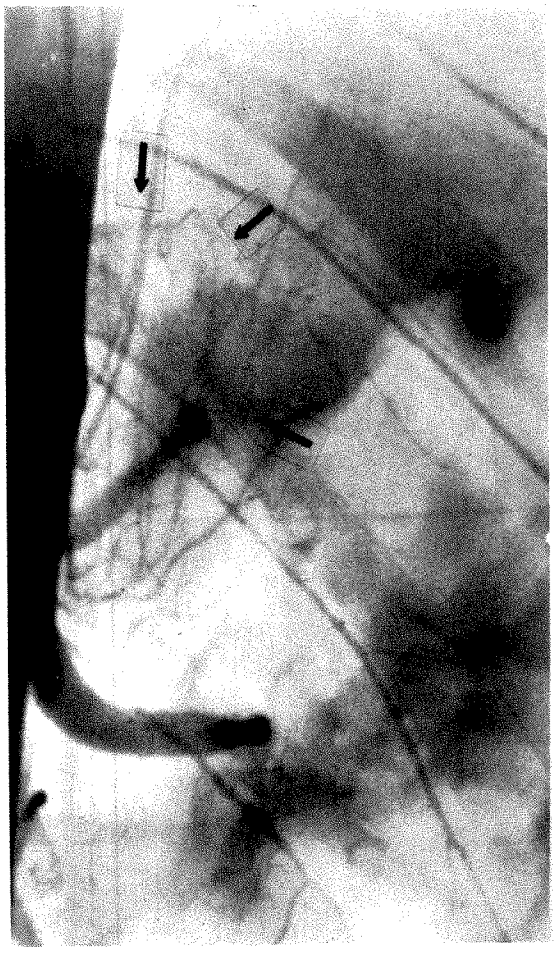


FIG. 1. Cushing's syndrome. Aortogram after injection of small quantities of adrenalin. The left adrenal is enlarged and hypervascularized (arrows). No pathologic vessels are seen. Apparent hyperplasia was confirmed at operation

In the cases of *cortical adenoma* the microangiographic vascular pattern varied: sometimes it was reminiscent of the normal gland and sometimes the architecture was entirely pathologic. The vascular density also varied, and in some cases the clinical angiograms showed hypervascularity (Fig. 5, *A* and *B*), occasionally with a number of pathologic vessels (Fig. 2, *A* and *B*); in other cases the vascular pattern was apparently normal. Small adenomas with normal vascular architecture can never be demonstrated, however well the roentgenographic technique is carried out.

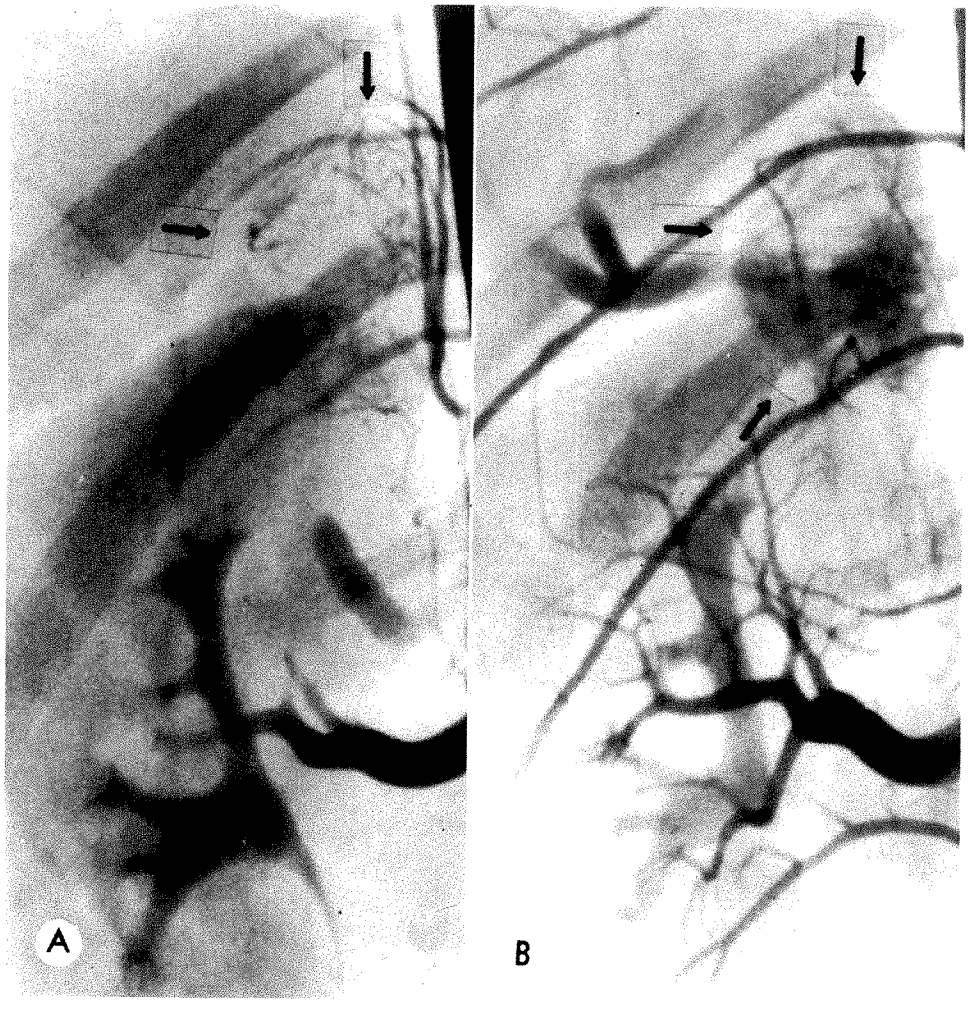



FIG. 2. Cushing's syndrome with adenoma, confirmed at operation. Aortogram after injection of small quantities of adrenalin. (*A*) Early vascular phase with hypervascularization of right adrenal (arrows), in which a number of small pathologic vessels are seen. (*B*) Late vascular phase, showing charging of the enlarged gland (arrows).

*Pheochromocytomas* are generally considered to be hypervascularized and should therefore stand out against the adenoma. Of the 6 specimens examined by microangiography only 1 was seen to be hypervascularized, and this had highly pathologic vessels. On the basis of the vascular pattern the others could not be distinguished from an adenoma by either angiography or microangiography (Fig. 3, *A* and *B*; and 4, *A* and *B*).

In contrast to many other angiographic studies it is not necessary to take account of the hypervascularity that might arise in inflammatory conditions in the interpretation of the vascular pattern of the adrenal gland,[7] since any inflammatory component in the adrenals has a rapid deleterious action and the neoplastic changes have proved to be devoid of such a component.

This study thus shows that the optimism displayed by certain authors regarding the possibility of distinguishing roentgenographically between various forms of adrenal alterations is groundless. In most cases in which there are changes in the adrenal glands there is, however, no need to rely on films for a differential diagnosis:

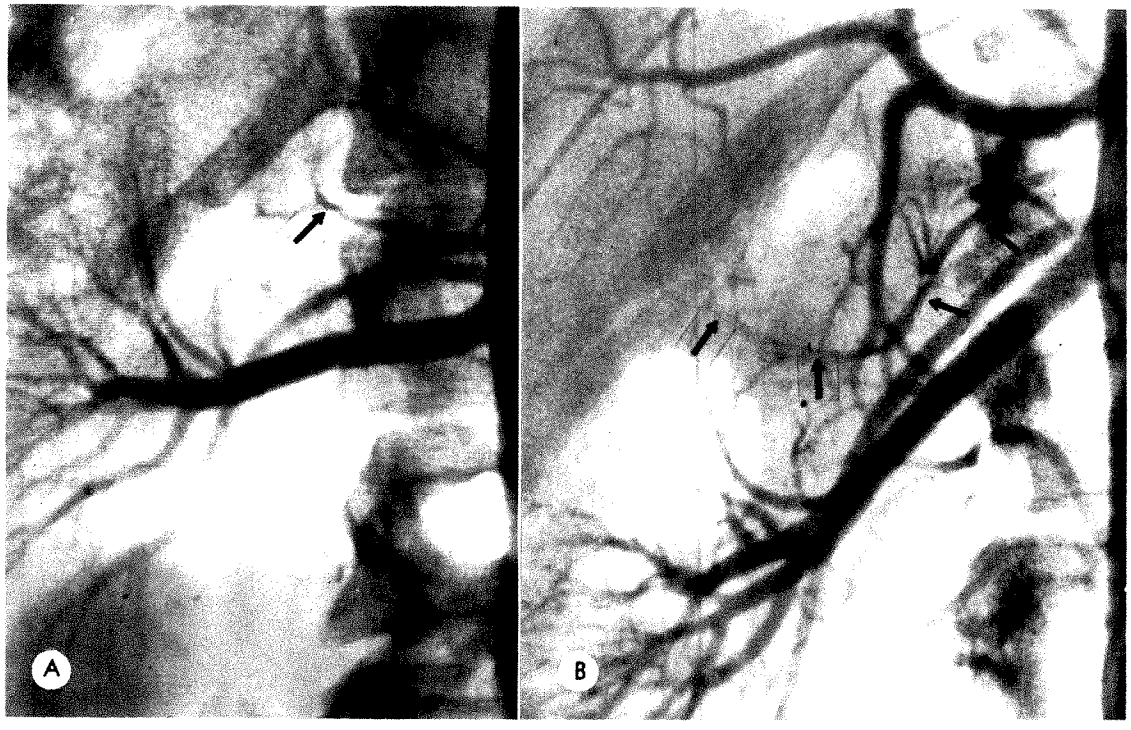



FIG. 3. Pheochromocytoma, confirmed at operation. (*A*) Aortogram showing normal conditions (arrows) 2 years before the diagnosis was confirmed. (*B*) Aortogram showing two branches from the right adrenal artery stretched around an enlarged gland (arrows). No pathologic vessels are noted.

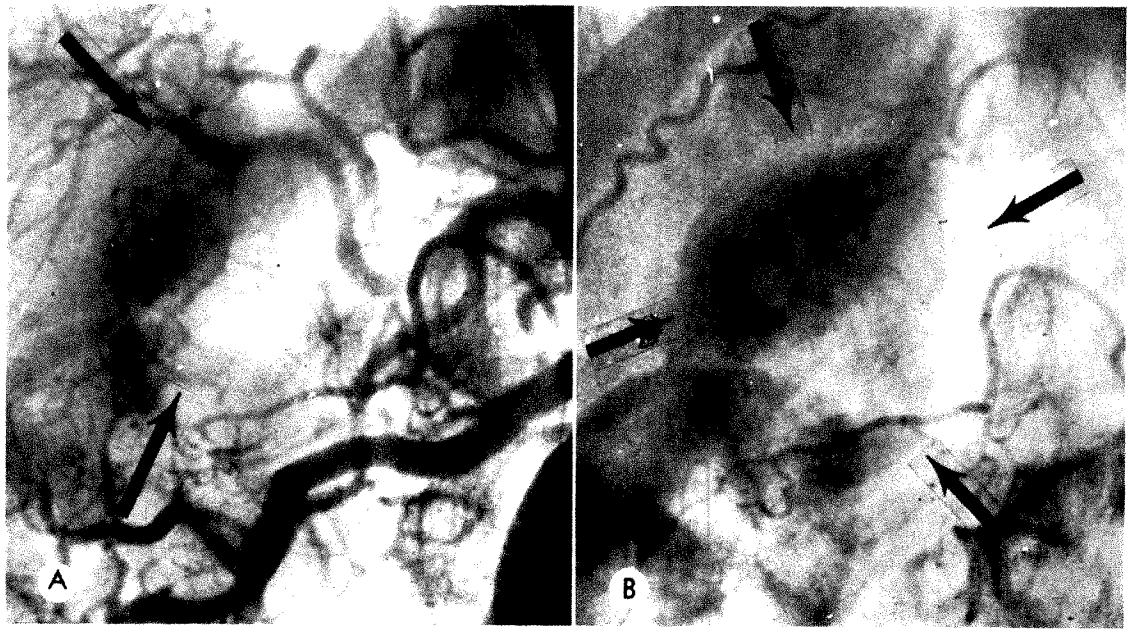



FIG. 4. Pheochromocytoma confirmed at operation. (*A*) Aortogram showing stretched adrenal vessels (arrows) with a moderate hypervascularization and a few small pathologic vessels. (*B*) Late phase from the same examination showing charging of an enlarged adrenal (arrows).

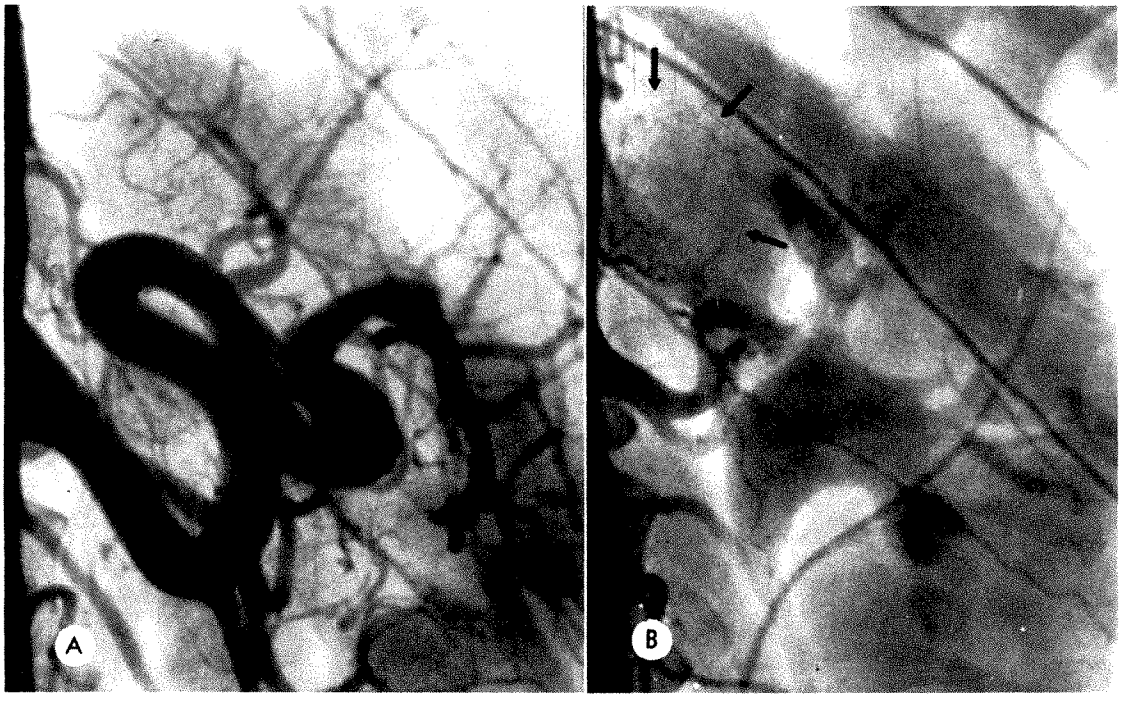



FIG. 5. Hyperaldosteronism with adenoma, confirmed at operation. (*A*) Aortogram where the lienal artery and small branches partly cover the left adrenal. Normal conditions prevail. (*B*) Aortogram after injection of small amount of adrenalin. A moderately enlarged adrenal gland with hypervascularization is now visualized (arrows).

this will often be provided by the clinical picture and hormonal findings, the roentgenographic examination being required only to determine the side affected and the possibility of an aberrant organ. This problem, too, can sometimes present difficulty, and the prospects of improving the information have been discussed. Merklin and Michels,[11] for instance, have recommended that as the arterial system of the adrenal gland is a complex one, contrast medium should be supplied not only to branches coming directly from the aorta but also to the branches of the celiac and superior mesenteric arteries. The same view has been expressed more recently by Ludin[10] in 1966, who has shown that in practice the chance of visualizing the adrenal arteries is improved if aortography can be supplemented with selective examination of the celiac and superior mesenteric arteries. Since the changes in the adrenals are often of minor extent the intra-arterial administration of small amounts of adrenalin prior to the injection of contrast medium will often improve the diagnostic prospects.[9] In this way a contraction of the arteries is obtained that reduces the flow of contrast medium to adjacent organs, thus improving the visualization of the adrenals (Fig. 5, *A* and *B*). Some authors recommend selective adrenal arterial angiography, in the belief that this provides better results,[13,16] but since the adrenals can be supplied by many vessels, this method would seem to be of little practical value.

A more promising procedure from the anatomic standpoint is to inject contrast medium into the venous side for angiography, since this is more regularly distributed. Olin and Reuter[14] have described a method in which small amounts of angiotensin or adrenalin are injected into the renal artery through a selectively placed catheter so as to reduce the renal

circulation, and then to inject the contrast medium into the catheterized renal vein. This method might be of value for examining the left adrenal, whose veins usually drain into the left renal vein. This technique has been developed further by Mikaelsson,[12] who performs bilateral selective adrenal phlebography, using suitably designed catheters for each side. This method has the advantage that it can be combined with hormonal level determination.[2] Because of the extremely thin wall of the adrenal veins the injection pressure must be very low if rupture is to be avoided. In some of the cases in our material the operation was preceded by selective adrenal phlebography, and in one instance it was found that the gland had ruptured and that there was a large hematoma in the surrounding tissue. By choosing a suitable technique it should be possible to avoid such complications. Adrenal phlebography performed hitherto shows that the method is useful for locating the side of suspected adrenal changes (Fig. 6). It is of no value, however, for detecting minor alterations when the vascular pattern is normal. It is evident from the above that the roentgenographic diagnosis of changes in the adrenal glands is difficult, and that the differential diagnosis is often impossible. The best results appear to be provided by selective adrenal phlebography, possibly combined with injection into the renal artery of small doses of adrenalin through selectively placed catheters so as to reduce the renal blood flow.



Fig. 6. Cushing's syndrome with adenoma, confirmed at operation. Selective adrenal phlebography showing an enlarged gland.

SUMMARY

On the basis of angiographic, microangiographic and histologic studies of normal and neoplastically altered adrenal glands suitable roentgenographic methods for examining suspected changes of the adrenal glands are discussed.

The difficulties of differential diagnosis are pointed out.

The most suitable examination technique from the anatomic aspect would seem to be bilateral adrenal phlebography, possibly combined with an injection of small doses of adrenalin via the renal arteries to reduce the blood flow.

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# SELECTIVE ANGIOGRAPHY OF THE ADRENAL GLANDS*

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CONVENTIONAL roentgenographic methods designed to detect adrenal pathology are best suited to the demonstration of large masses. These methods provide less than optimal visualization of the subtle morphologic changes which frequently accompany adrenal disease. We have applied a selective angiographic technique in an attempt to improve our diagnostic accuracy in such lesions. Direct catheterization of adrenal vessels[15] combined with selective vasoconstriction provides a method for demonstrating regularly the adrenal glands in spite of their variable blood supply.

## ANATOMIC CONSIDERATIONS

The adrenal blood supply is divided classically into superior, middle and inferior adrenal arteries arising respectively from the inferior phrenic artery, aorta and renal artery[5] (Fig. 1). In actuality, there are great variations in this pattern from patient to patient and from side to side.[4,13] The roentgenologic anatomy is not well described. The following brief description is based not only on the cases described in this report, but also on about 80 other partial selective adrenal arteriograms obtained coincidentally.

On the right side, one or more superior adrenal branches almost always arise from the posterior division or main trunk of the right inferior phrenic artery. These vessels supply the roughly triangular portion of the upper pole. Right middle adrenal branches are often small and difficult to identify. When seen, they usually supply the medial portion of the gland, and arise jointly with branches to renal capsule, perirenal fat and other portions of the retroperitoneum. The right inferior adrenal arteries often arise from the renal artery, but may also originate from accessory renal or gonadal arteries. They supply the flat lower portion of the gland. It is not unusual to see a vessel arising from the inferior phrenic artery with the classic "middle adrenal" distribution, or to see an "inferior adrenal" artery coming directly from the aorta. In these cases there is an obvious conflict between the anatomic definition of adrenal vessels in terms of their point of origin, and the more useful roentgenologic classification which is based on their area of distribution within the gland.

On the left side, the inferior phrenic artery usually provides fewer superior adrenal branches. The upper pole of the left gland is more rounded than on the right and usually thinner in the frontal projection. Consequently, the blush obtained on the arteriograms is usually less dense on the left side. The middle adrenal distribution is often quite extensive. In many cases, it appears to supply most of the gland. We have seen the left middle adrenal artery arise from the proximal part of the right inferior phrenic artery in a number of cases. The left inferior adrenal arteries usually supply a portion of the gland in close proximity to the renal hilus.

## PHARMACOLOGIC ASPECTS

Many arteries show intense vasoconstriction for a brief period following the intra-arterial injection of minute amounts of epinephrine. This is most dramatically demonstrated in the renal arteries.[6] Unlike most other visceral branches, the adrenal

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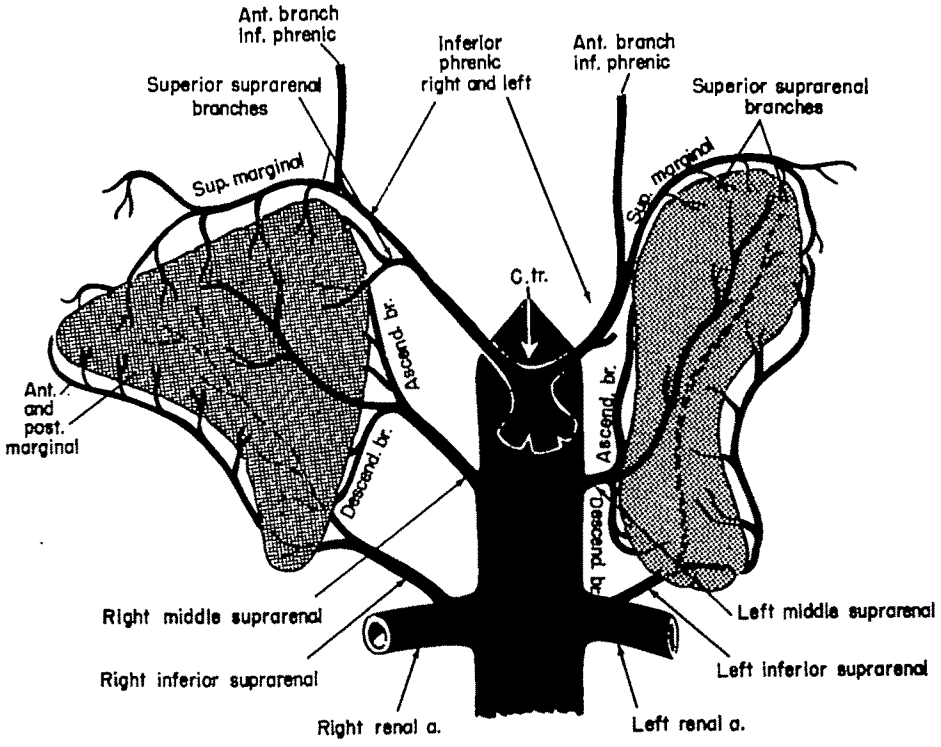



FIG. 1. Schematic diagram of adrenal circulation. After Gérard.[5] Some of the variations are discussed in the text.

arteries exhibit a striking unresponsiveness to epinephrine.[18] Consequently, the post-epinephrine arteriograms of the renal and celiac arteries tend to visualize adrenal branches selectively whenever they are present.

## METHOD

Aortography is a necessary prerequisite for selective adrenal angiography. A J tipped clear polyethylene catheter (PE 240) with side holes is inserted into the abdominal aorta via a femoral artery by the Seldinger technique. The tip is positioned at the level of the twelfth thoracic vertebra. A serial arteriogram is obtained during a Valsalva maneuver using 30 to 50 cc. of 60 per cent methylglucamine diatrizoate delivered in 1 to 2 seconds.

The J shaped catheter is exchanged for a precurved opaque polyethylene catheter (B-D RPX 054) which is placed into a renal artery. Ten micrograms of epinephrine in 10 cc. of saline are injected rapidly into this vessel. Three seconds later, a serial arteriogram is obtained using 10 to 20 cc. of contrast material delivered in 5 to 10 seconds. The procedure is repeated in the opposite renal artery.

The aortogram is now analyzed carefully and the origin of the inferior phrenic and middle adrenal arteries identified whenever possible. Selective catheterization of the inferior phrenic arteries (Fig. 2) is now attempted. Details of this technique are described elsewhere.[7] If one or both of these vessels are seen to arise from the celiac axis, an epinephrine celiac arteriography is performed. Serial roentgenograms are obtained using 20 $\mu$g. of epinephrine followed by an injection of 20 to 35 cc. of contrast material.

Selective middle adrenal arteriography is then undertaken (Fig. 3). The technique of catheterization of small vessels varies slightly from that employed in large arteries. One does not usually see or feel the tip of the catheter entering the vessel. Free backflow of blood is often not obtained.

The catheter is therefore manipulated into the area in which the artery is expected to originate. A syringe containing contrast material is attached to the catheter. While probing the area gently, numerous 0.5 cc. injections are made in the hope of visualizing the vessel. The operator must not use undue force in making these test injections.

Contrast material injected into adrenal vessels causes some discomfort to the patient even when used in small quantities. Injections into the inferior phrenic arteries cause pain referred to the shoulder, whereas middle adrenal injections cause pain in the flank. The volume of contrast material used varies with the size of the vessel and the tolerance of the patient. Frequently, the catheter occludes the artery, as evidenced by delayed emptying seen under fluoroscopy. In this situation, the catheter should be withdrawn immediately after the injection for arteriography is completed.

## RESULTS

Selective angiography performed primarily for the demonstration of adrenal pathology was undertaken in 14 patients (Table 1). Adequate adrenal visualization was achieved in all cases. There were no complications. It was possible to evaluate the size and shape of the adrenal glands by adding the composite blushes from the various adrenal injections. Thus, the diagnosis of adrenal hyperplasia was made (Fig. 4, *A*, *B* and *C*). A small adrenal adenoma, not visualized by any other roentgenologic method, was also detected readily by this selective technique (Fig. 5, *A* and *B*). Adrenal metastases from a carcinoma of the lung were also identified.[7]

Selective adrenal angiography was not required routinely for the localization of pheochromocytomas. However, in one patient with an extra-adrenal tumor situated between the aorta and inferior vena cava, selective angiography localized a lesion which could have been overlooked on the aortogram and selective renal arteriogram (Fig. 6, *A-D*). Adrenal venography further defined the tumor (Fig. 7).

Determining the origin of tumors in the suprarenal area depends upon demonstrating their principal blood supply. Selective angiography identified the origin of an adrenal carcinoma (Fig. 8, *A-D*), a large malignant adrenal adenoma, and a neuro-

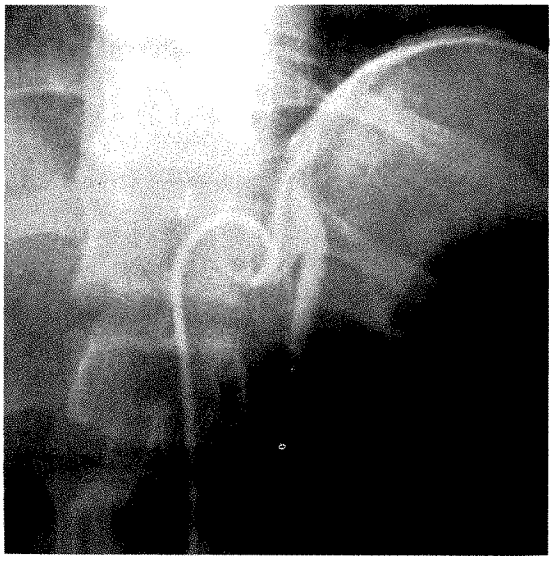


FIG. 2. Selective left inferior phrenic arteriogram demonstrates a blush in the upper pole of a normal adrenal gland.

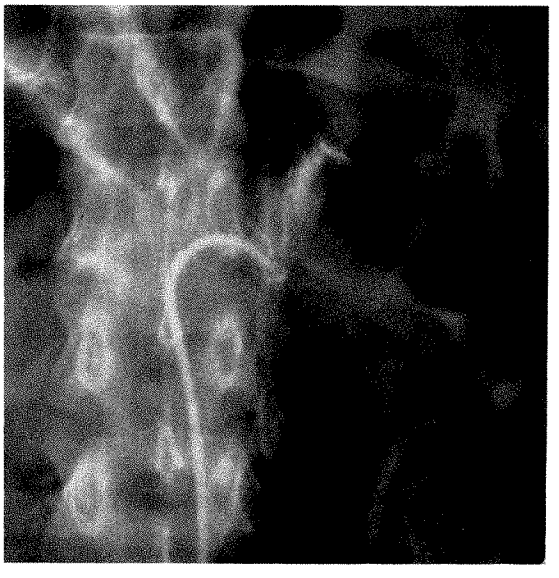


FIG. 3. Selective left middle adrenal arteriogram shows a blush in the midportion of a normal adrenal gland.

TABLE I

CASE MATERIAL

| Case No. | Age, Sex | Indication for Adrenal Arteriography | Pertinent Injections | Diagnosis |
|---|---|---|---|---|
| 1 | 32 F | Cushing's syndrome | Aorta<br>Right middle adrenal<br>Right inferior phrenic<br>Right renal (epinephrine) | Bilateral adrenal hyperplasia |
| 2 | 39 M | Cushing's syndrome | Aorta<br>Right inferior phrenic<br>Left middle adrenal | Left adrenal adenoma |
| 3 | 42 M | Rule out Cushing's syndrome | Aorta<br>Right inferior phrenic<br>Left inferior phrenic<br>Left middle adrenal | Borderline adrenal enlargement |
| 4 | 65 F | Rule out Cushing's syndrome | Aorta<br>Right renal (epinephrine)<br>Left renal (epinephrine)<br>Celiac (epinephrine) | Normal adrenals |
| 5 | 24 F | Rule out Cushing's syndrome | Aorta<br>Celiac (epinephrine)<br>Right renal (epinephrine)<br>Left renal (epinephrine)<br>Left middle adrenal | Normal adrenals |
| 6 | 17 F | Locate pheochromocytoma | Aorta<br>Right renal<br>Left renal<br>Right middle adrenal<br>Left middle adrenal<br>Right adrenal vein | Right extra-adrenal pheochromocytoma |
| 7 | 50 M | Evaluate left supra-renal mass | Aorta<br>Left inferior phrenic<br>Left middle adrenal<br>Left renal (epinephrine) | Left adrenal carcinoma |
| 8 | 2 F | Evaluate left retro-peritoneal mass | Aorta<br>Left inferior phrenic<br>Left middle adrenal<br>Left renal (epinephrine) | Neuroblastoma, left adrenal region |
| 9 | 72 M | Evaluate left abdominal mass | Aorta<br>Left middle adrenal | Malignant adenoma left adrenal |
| 10 | 28 F | Rule out aldosteronoma | Aorta<br>Right renal (epinephrine)<br>Right accessory renal<br>Left inferior phrenic<br>Left renal (epinephrine) | Normal adrenals |

Table I—(*Continued*)

| Case No. | Age, Sex | Indication for Adrenal Arteriography | Pertinent Injections | Diagnosis |
|---|---|---|---|---|
| 11 | 50 M | Rule out aldosteronoma | Aorta<br>Right inferior phrenic<br>Right renal (epinephrine)<br>Left renal (epinephrine) | Normal adrenals |
| 12 | 20 M | Rule out aldosteronoma | Aorta<br>Right inferior phrenic<br>Left inferior phrenic<br>Celiac (epinephrine) | Normal adrenals |
| 13 | 60 F | Rule out aldosteronoma | Aorta<br>Right inferior phrenic<br>Left inferior phrenic<br>Right renal (epinephrine)<br>Left renal (epinephrine) | Normal adrenals |
| 14 | 47 F | Rule out aldosteronoma | Aorta<br>Right inferior phrenic<br>Left inferior phrenic<br>Right renal (epinephrine) | Normal adrenals |

blastoma (Fig. 9, *A* and *B*). In each of these cases, aortography failed to demonstrate the feeding vessels.

An attempt to locate aldosterone-secreting tumors was made in 5 cases. No lesions were demonstrated. In only one instance was there sufficient clinical indication to perform surgery, and in this case no tumor was found.

## DISCUSSION

The roentgenologic methods currently used frequently permit the diagnosis of adrenal lesions. Plain roentgenograms and intravenous urograms, particularly when supplemented by nephrotomograms,[3] usually demonstrate large masses. Retroperitoneal pneumography[14] represents a further refinement in roentgenologic diagnosis and, when technically successful, occasionally shows lesions less than 2 cm. in size. Aortography generally, but not always, demonstrates highly vascular tumors,[9,11] but does not permit a very detailed analysis of the adrenal structure. The combination of aortography and retroperitoneal pneumography[10] results in a technically complex examination often yielding little additional information.

Two principles are involved in our technique of selective adrenal angiography. First, it has been shown that the adrenal glands are quite unresponsive to doses of epinephrine which constrict most visceral branches.[18] Second, a relatively simple technique for catheterizing small aortic branches has been developed. As a result, it has been possible to obtain highly detailed adrenal angiograms in the 14 patients presented here.

Unlike the retroperitoneal pneumogram, the selective adrenal arteriogram shows the gland without its fatty envelope. This permits a better estimation of its size in patients with Cushing's disease. With more experience, it should be possible to develop rather precise criteria for the diagnosis of adrenal hyperplasia.

The vascularity of functioning adrenal adenomas is such that, while not readily

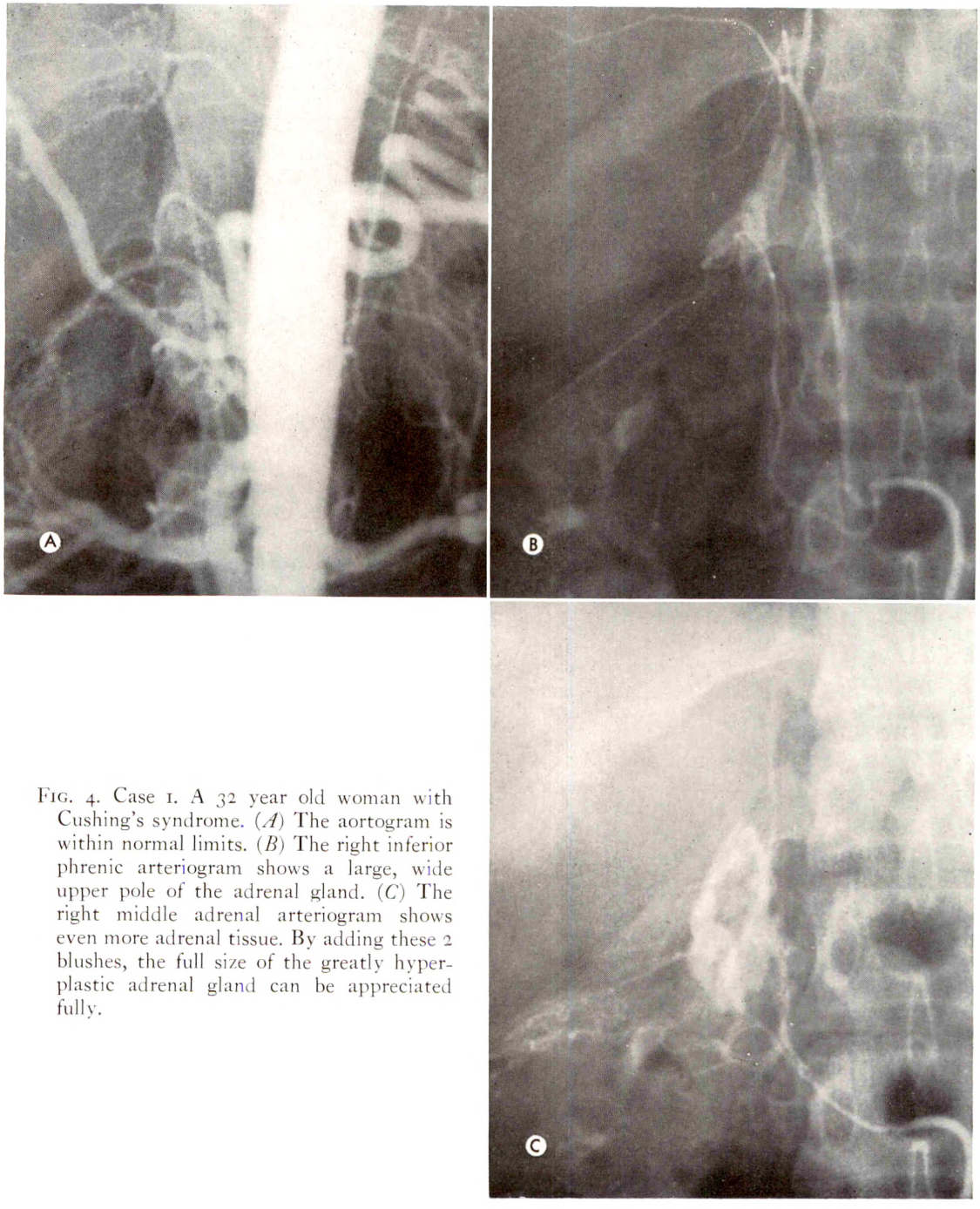



FIG. 4. Case 1. A 32 year old woman with Cushing's syndrome. (*A*) The aortogram is within normal limits. (*B*) The right inferior phrenic arteriogram shows a large, wide upper pole of the adrenal gland. (*C*) The right middle adrenal arteriogram shows even more adrenal tissue. By adding these 2 blushes, the full size of the greatly hyperplastic adrenal gland can be appreciated fully.

detected on aortograms, they are identified easily on selective angiograms. Not only does this permit the diagnosis of tumors too small to be seen by pneumography, but overcomes the technical problems encountered frequently in dissecting gas around a diseased left adrenal gland.

When Cushing's syndrome is present, the demonstration of one large adrenal gland, free of tumor, is sufficient to permit

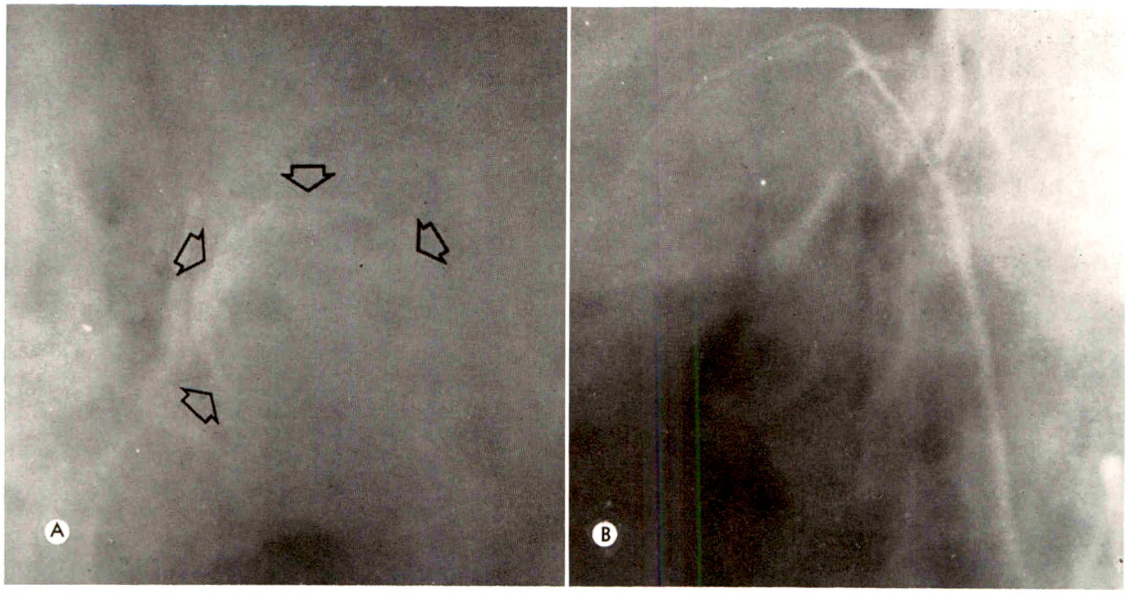



Fig. 5. Case 2. A 39 year old man with Cushing's syndrome. An aortogram and retroperitoneal pneumogram were negative. (*A*) Selective left middle adrenal arteriogram shows a round 3.5 cm. mass (arrows) just lateral to the adrenal gland. (*B*) A selective right inferior phrenic arteriogram shows a small adrenal gland on the right. At operation a small adenoma was found in the left adrenal gland.

the presumptive diagnosis of bilateral hyperplasia. In the presence of a functioning adenoma, the opposite gland atrophies.

In patients with pheochromocytomas, the surgeon is aided significantly when the lesion is localized preoperatively.[1,16] This is particularly true in the case of extra-adrenal pheochromocytomas. When proper precautions are taken, aortography and even selective arteriography[12] can be carried out safely in these patients. Constant blood pressure monitoring is particularly useful in this situation. While pheochromocytomas are frequently so vascular that they show up clearly on aortograms, selective arteriography will be required to demonstrate the lesion in occasional cases. Furthermore, there may be a place for selective angiography in excluding small lesions on the side opposite to the demonstrated pheochromocytoma.

Many of the larger adrenal tumors present as avascular lesions on aortograms. The selective injection of adrenal arteries not only demonstrates the organ of origin of the tumor, but also shows a surprising vascularity which more closely resembles the appearance of the pathologic specimen. The surgeon's approach may be influenced by this angiographic localization.

It is hoped that selective adrenal angiography may prove useful in the diagnosis of aldosterone-secreting tumors which are too small frequently to be shown by other roentgenologic methods. We have not yet had the opportunity to find such a lesion. We have, however, in a number of patients with advanced hypertension, noted a bilateral symmetric adrenal enlargement which we attribute to secondary hyperplasia.

Adrenal venography appears to be a promising adjunctive technique.[17] In addition to giving some anatomic information, blood samples are obtained simultaneously for biochemical studies.[2]

## SUMMARY

A method of selective adrenal angiography is described. It utilizes the direct catheterization of small aortic branches as well as the pharmacologic unresponsiveness of the adrenal gland to epinephrine.

Among 14 patients studied, this method

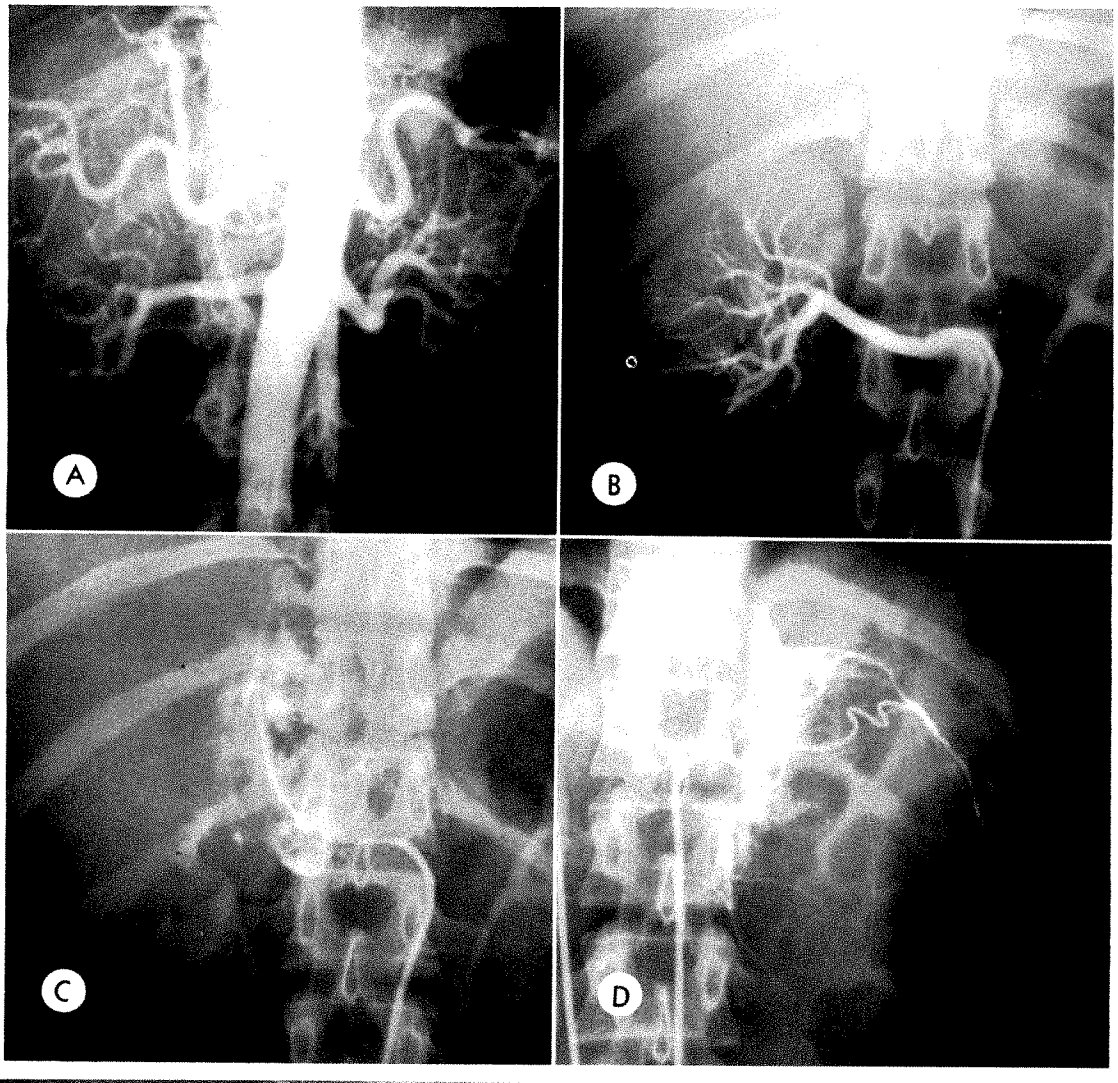



FIG. 6. Case 6. A 17 year old girl with diagnosed pheochromocytoma. (*A*) The aortogram shows slight deviation of the upper aorta to the left, but no definite tumor blush. (*B*) Selective right renal arteriogram is normal. A selective left renal arteriogram was also normal. (*C*) A right middle adrenal arteriogram shows a tumor blush in a para-aortic location. The blush corresponds in size and contour to the egg-sized lesion found at surgery between the aorta and the inferior vena cava. (*D*) A selective left middle adrenal arteriogram is normal.

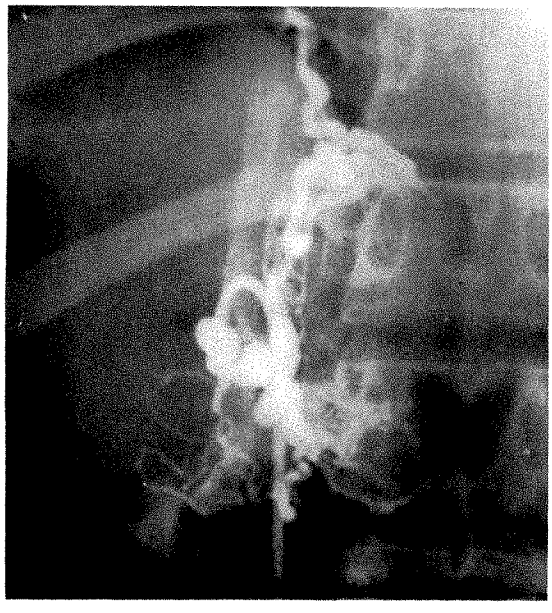


FIG. 7. Same patient as Figure 6. A selective adrenal venogram further outlines the pheochromocytoma.

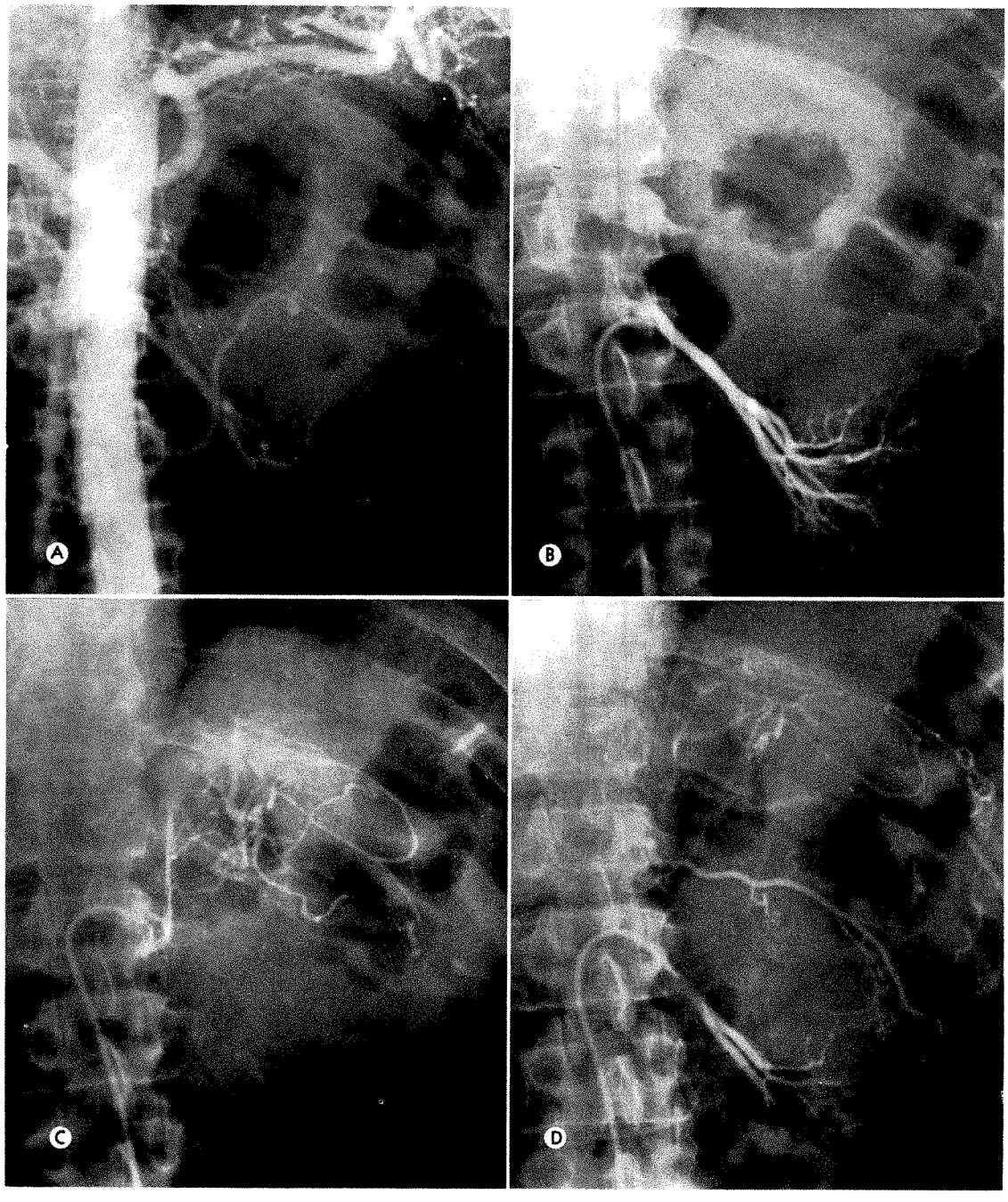



Fig. 8. Case 7. A 50 year old man with a suprarenal mass detected incidentally on intravenous pyelogram. (*A*) The aortogram shows displacement of the kidney and splenic artery, and an apparently avascular tumor. (*B*) The selective renal arteriogram fills only one small capsular artery to the mass. (*C*) The selective middle adrenal arteriogram permits extensive filling of irregular tumor vessels, indicating the adrenal origin of this tumor. (*D*) A renal arteriogram performed after epinephrine also demonstrates tumor vessels (RA = renal artery). At surgery a large adrenal carcinoma was found.

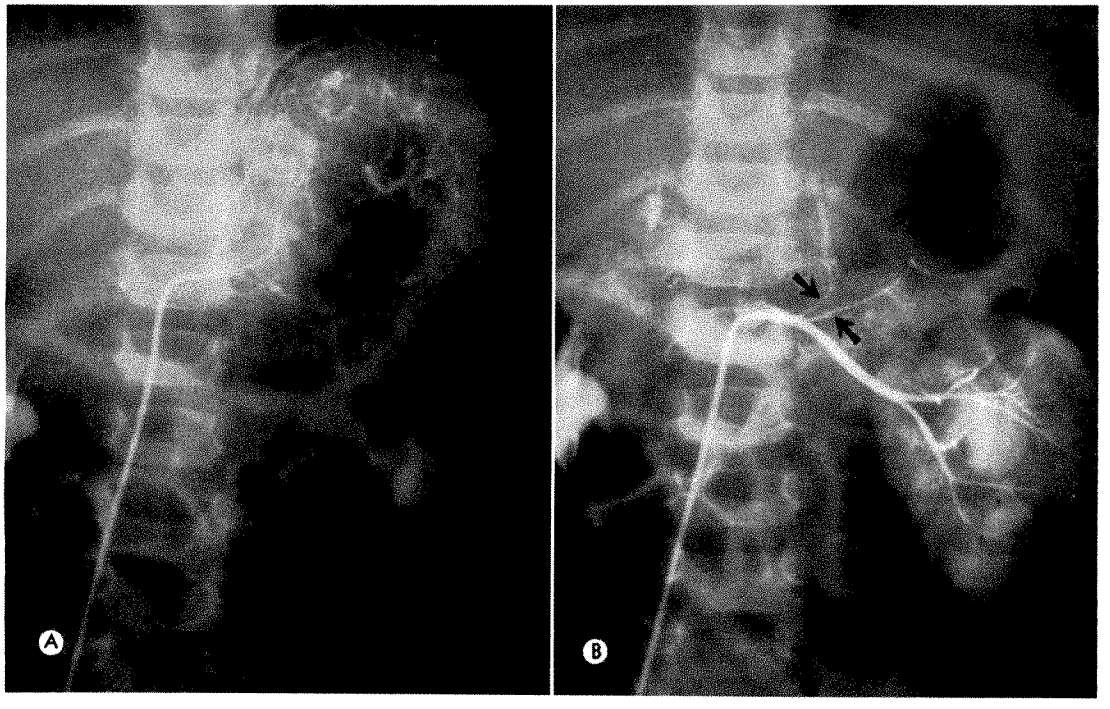



FIG. 9. Case 9. A 2 year old girl with suspected inoperable neuroblastoma being evaluated prior to biopsy and radiation therapy. An aortogram showed displacement of the lower aorta to the right, and downward and lateral displacement of the kidney with partial ureteric obstruction. Tumor vessels were not identified. (*A*) Selective left inferior phrenic arteriogram shows extensive tumor blood supply extending almost to the diaphragm. (*B*) Selective left renal arteriogram after epinephrine shows an additional segment of tumor supplied from inferior adrenal arteries (arrows). A small portion of the normal right adrenal gland is also seen.

has been found useful in diagnosing adrenal hyperplasia and adrenal adenomas, in localizing pheochromocytomas, in demonstrating adrenal metastases, and in determining the tissue of origin and extent of suprarenal masses.

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*Representatives on the American Board of Radiology:* J. F. Roach, Albany, N. Y.; C. A. Good, Rochester, Minn.; and C. A. Stevenson, Spokane, Wash.

*Director of Instruction Courses:* H. O. Peterson, Minneapolis, Minn.; *Associate Director:* D. G. Mosser, Minneapolis, Minn.

*Manager of the Annual Meeting:* J. C. Cook, LL 14 Professional Plaza, Detroit, Mich. 48201.

*Editor:* T. Leucutia, 401 Professional Building, Detroit, Mich. 48201.

*Representative on the Board of Chancellors of the American College of Radiology:* S. F. Ochsner.

*Sixty-ninth Annual Meeting:* Jung Hotel, New Orleans, La., Oct. 1–4, 1968.

## AMERICAN RADIUM SOCIETY

*President:* John L. Pool, New York, N. Y.; *President-Elect:* Juan A. del Regato, Colorado Springs, Colorado; *1st Vice-President:* Sol B. Gusberg, New York, N. Y.; *2nd Vice-President:* E. Richard King, Richmond, Va.; *Secretary:* Fernando G. Bloedorn, University of Maryland Hospital, Baltimore, Md. 21201; *Treasurer:* James F. Nolan, 1407 South Hope Street, Los Angeles, Calif. 90015.

*Executive Committee:* Joseph H. Farrow, Chairman, New York, N. Y.; John L. Pool; Juan A. del Regato; Sol B. Gusberg; E. Richard King; Fernando G. Bloedorn; James F. Nolan; Justin J. Stein; Milton Friedman.

*Scientific Program Committee:* John L. Pool, Chairman; Juan A. del Regato; Vincent P. Collins; Arthur J. Holleb; Richard H. Jesse; Ralph F. Phillips.

*Arrangement Committee:* Ivor Fix, Chairman, Miami Beach, Fla.; Luther W. Brady, Chairman-designate; Everett Shocket; Ashbel C. Williams; Mario Vuksanovic; John S. Stewart; Harvey P. Groesbeck.

*Publication Committee:* Harry Hauser, Chairman, Cleveland, Ohio; Martin Van Herik; Robert J. Bloor.

*Public Relations Committee:* Everett Shocket, Chairman, Miami Beach, Fla.; Harvey P. Groesbeck; Antolin Raventos; Luther W. Brady, ex officio.

*Janeway Lecture Committee:* Clifford L. Ash, Chairman, '69, Toronto, Canada; A. N. Arneson, '71; Milford D. Schulz, '73.

*Representatives on the American Board of Radiology:* Donald S. Childs, Rochester, Minn.; Justin J. Stein, Los Angeles, Calif.; Ralph M. Scott, Louisville, Ky.

*Liaison Representatives on the National Council on Radiation Protection and Measurements:* Herbert M. Parker, Richland, Wash.; Marvin M. D. Williams, Rochester, Minn.

*Representative on the Board of Chancellors of the American College of Radiology:* Charles G. Stetson.

The Fiftieth Annual Meeting of the Society will be held at the Hotel Fontainebleau in Miami Beach, Fla., April 7–11, 1968.

# EDITORIAL

## THE EDUCATION OF THE RADIOLOGY RESIDENT

THOSE of us who have been in the field of radiology for 20 or more years are very aware of the tremendous changes which have taken place in that seemingly short period of time. What used to be essentially a gross anatomic and pathologic discipline has had the dimension of physiology added. In addition, the depth of application of the other basic sciences has made commonplace the radiologic investigation of diseases and conditions previously untouchable or unheard of. The development within the general field of radiology of the subspecialties of pediatric and neuroradiology, of cardioradiology and angiography, of nuclear medicine and radiobiology is evidence of the huge wealth of radiologic detail to be acquired by the resident before his future colleagues put the stamp of approval on him. In the face of this, can one say that the existing plan of medical education is capable of successfully bringing to fruition the training of the young radiologist in the most economical fashion? Before we can answer that question, we must take stock of the needs, present and future. The growth of clinical radiology in terms of patient examinations and patient treatments has, over the years, shown a steady annual increase, usually in the neighborhood of 7 or 8 per cent. There is no reason to believe that this will not continue. Indeed, it is reasonable to expect the increment of increase to become greater. Thus, the need for trained radiologists will grow, but there already is a shortage of radiologists—this seems especially true of radiotherapists. The residency program director is accustomed to the constant stream of letters which come across his desk from people all over the country in search of radiologists. Wherever one travels or whenever radiologists congregate, the question is heard: "Do you know where I can find an associate?". The NACOR (National Advisory Committee on Radiology) report to the Surgeon General of the U. S. Public Health Service dated April 1966 clearly indicates that this shortage is going to continue and, unless a drastic change occurs in the numbers now entering the field of radiology, it is bound to become critical. It, therefore, seems quite obvious that with the growing service loads and the increasing shortage of radiologists something needs to be done to attract more people to the field and perhaps shorten the time period which adequate training requires. It is especially to this latter point that I wish to address myself, for if we can shorten the period of training we have thereby increased the available supply of radiologists by a proportionate amount.

The present system of medical education requires most applicants to undertake a four year college program with inclusion of many subjects of a non-medical and even non-scientific nature. To be sure, the bachelor degree holder is probably more academically rounded and perhaps better balanced by his college years, but will he thereby become a better physician in this highly technical age? Can we spare the time to philosophy, the romance languages, fine arts, etc., or should it be devoted to mathematics, sociology, physics and, to coin a word, computerology? Then medical school! This also consumes four years and the curriculum includes not only the basic

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sciences, those building blocks of medicine, but a myriad of unrelated courses in medical and surgical specialties, all of which demand great effort and time on the part of the student. Should not the role of the medical school be planned to de-emphasize the specialties and provide the candidate with a really solid foundation in general medical sciences, coupled with the trained ability and know-how to build thereon? Many of us have been exposed to "speeded-up programs" which are on trial in several universities and which are designed to bring the student to completion of the college and medical school training in something less than the usual eight years, commonly six. These programs are not to be confused with the speeded-up medical school curricula of World War II which suffered so greatly from the absence of teachers. Although these trial programs as yet lack sufficient depth in time to make a real evaluation possible, preliminary judgment suggests that they have great merit and warrant much further study, development and probable expansion.

What is today's product of college and medical school? Is he ready to undertake his life's work? The answer is, of course, no; he must now undergo an internship with a minimum length of one year. It does not have to be any particular kind of internship; it can be a straight medical or a straight surgical, a mixed or rotating, a good internship or something less than good, as long as it is an approved internship. Does not this lack of specificity suggest that we really do not consider what takes place in that internship year to be of vital importance? Should we not question the value of such a year, a large part of which is, at best, necessarily devoted to non-productive activities? Is the fledgling radiology resident better prepared to begin his specialty study because of his experiences of that year? Would it not be wiser to take that year and make it a coordinated integrated part of the total residency? Should it not be a radiology internship? In other words, should we not take the time spent in internship and make it available for specialty training?

Now for the residency itself. Although the content has been increased, our training requirements in relation to time are essentially the same today as they were 20 years ago, notwithstanding the fact that in recent years we have inserted a year of waiting between the completion of the residency and the taking of the American Board examinations. Although many candidates do remain in an academic atmosphere for their fourth year, since there is no requirement to do so, many do not; and for some the fourth year is a waste of time. Indeed, it is possible that there are those who are better prepared for examination at the end of the third year of residency than after the fourth year. Thus, it seems logical to question the desirability of requiring or accepting such a permissive year. Would it not be an improvement to first determine how many years it should take the average resident to comprehend that body of knowledge which considerate judgment says is necessary and thus establish the length of the training period, accepting the expectation that when an individual successfully completes that training period he is ready for both examination and independent practice?

To this point we have done little more than tear things apart, not with the intent of being destructive but solely to permit closer inspection of the pieces with the hope that with those fragments we can then reconstitute a program that will more adequately and economically satisfy our needs. Specifically then, what can we build? Perhaps we should consider something along the following lines.

*College and Medical School.* Instead of two separate courses of study—one liberal and one medical—they should be combined into one integrated study pattern designed to provide a man with some liberal background but chiefly directed towards an intellectually strong foundation in the basic sciences, some of the social sciences and general medicine and surgery, with an

accent on graded clinical experience spread throughout the medical portion of the training. Preferably this phase of the resident's education should be completed in six years.

*The Residency*. Training should begin immediately after graduation from the combined college and medical school and the training period in radiology needs to be a minimum of four years. *Three separate and distinct programs* should be offered in our particular specialty.

The *first* is the general radiologic residency designed to prepare an individual for practice in the community hospital and/or private office. This would entail one and a half years of diagnosis, one and a half years of therapy, and one year elective in either diagnosis or therapy or divided between the two. The elective year would permit the resident to concentrate in one or more areas of his choice, such as neuroradiology or orthovoltage therapy.

The *second* program is in straight diagnosis. Three years would be devoted to general diagnostic radiology and an elective fourth year would be added which would permit continuation of general diagnostic work or provide an opportunity for concentration in a subspecialty field within diagnostic radiology, such as nuclear medicine.

The *third* program is in straight therapy. This program would entail three years in general radiation therapy with a fourth elective year for, again, greater depth of experience in general therapy or concentration on a portion thereof.

The second and third programs are intended to provide highly skilled individuals for practice in the urban medical center.

What advantages or improvements might we expect from these programs?

1. The graduate would be a well educated individual capable of independent thought and judgment.

2. He would be better trained in radiology than his counterpart of today. He would be well prepared to undertake with no external help not only the usual radiologic procedures but many of the complicated ones too.

3. He would arrive at a point where he is ready to enter upon his life's work some three years earlier than the man of today.

Needless to say, I can't expect all to agree with the plan offered above. Perhaps I should expect none to approve, for in truth, I have sincere doubts myself; but the suggested program is presented to you with the hope that it will stimulate your thoughts and possibly thereby a different and wiser plan will eventually emerge which might play a significant role in solving some aspect of the very difficult manpower problem facing the future of radiology.

John F. Roach, M.D.

Albany Medical College
of Union University
Albany, New York 12208

# NEWS ITEMS

## NEW OFFICERS OF THE AMERICAN ROENTGEN RAY SOCIETY

At the Sixty-eighth Annual Meeting of the American Roentgen Ray Society held at the Washington Hilton Hotel, Washington, D. C., September 26–29, 1967 the following officers were elected: *President:* Dr. C. Allen Good, Rochester, Minnesota; *President-Elect:* Dr. Stephen W. Brown, Augusta, Georgia; *1st Vice-President:* Dr. J. Scott Dunbar, Montreal, Quebec, Canada; *2nd Vice-President:* Dr. A. Bradley Soule, Jr., Burlington, Vermont; *Secretary:* Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Georgia 30322; *Treasurer:* Dr. Colin B. Holman, Mayo Clinic, Rochester, Minnesota 55901.

The new Chairman of the Executive Council is Dr. Seymour Fiske Ochsner, Ochsner Clinic, 1514 Jefferson Highway, New Orleans, Louisiana 70121.

The Sixty-ninth Annual Meeting of the Society will be held at the Jung Hotel, New Orleans, Louisiana, October 1–4, 1968.

## SOCIETY FOR PEDIATRIC RADIOLOGY

The Tenth Annual Meeting of the Society for Pediatric Radiology was held September 25, 1967, in conjunction with the Sixty-eighth Annual Meeting of the American Roentgen Ray Society, at the Washington Hilton Hotel, Washington, D. C.

The following officers were elected: *President:* Dr. Martin H. Wittenborg, Boston, Massachusetts; *President-Elect:* Dr. David H. Baker, New York, New York; *Secretary-Treasurer:* Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Boulevard, Los Angeles, California 90027.

The Eleventh Annual Meeting of the Society will be held at the Jung Hotel, New Orleans, Louisiana, September 30, 1968.

## CONFERENCE ON THYROID CANCERS

Hôpital Cantonal Universitaire
Lausanne, Switzerland

The International Union against Cancer will hold a Conference on Thyroid Cancers in Lausanne, Switzerland, May 8–10, 1968.

This Conference will deal with various aspects of thyroid cancer: epidemiology, functional activity, experimental research, results of therapy, as well as the basis of a new clinical and pathologic classification.

The International Union against Cancer is an organization which devotes its activity to extend the fight against cancer throughout the world. Its members belong to organizations or research institutes in 106 countries.

The interested persons will be able, if they wish, to obtain more information from the President of the Committee for the organization: Professor G. Candardjis, Institut universitaire de radiologie médicale, Hôpital cantonal, 1000 Lausanne (Switzerland), or by writing to Union Internationale Contre le Cancer (UICC), Rue du Conseil-Général 3, 1200 Genève (Switzerland).

# BOOK REVIEW

*Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.*

Radiology in Neuro-Ophthalmology. By Guido Lombardi, M.D., Chief Radiologist, Istituto Neurologico di Milano; Consultant Radiologist, Istituto Oftalmico di Milano. Cloth. Pp. 244, with 167 illustrations. Price, $13.25. The Williams & Wilkins Company, Baltimore 2, 1967.

This is the second book by Lombardi to appear in English translation and is an excellent account of the author's vast experience with neuro-ophthalmologic problems. Dr. Lombardi is chief radiologist at the large Neurological Institute of Milan and consultant to the Ophthalmologic Clinic. In this double role he has had the unique opportunity of observing many neuro-ophthalmologic patients and has collected his experiences from the standpoint of a radiologist interested in neuroradiology as well as in diseases of the eye and of the orbit. This was done in France by Hartman and Gilles in 1949 and Dr. Lombardi's book may be considered a refinement of the book by the two French authors while presenting the additional information available now.

After an introductory chapter, Dr. Lombardi describes the techniques of examination employed. These include straight radiographic and tomographic procedures as well as contrast orbitography, and the angiography of the ophthalmic artery and veins. The third chapter deals with the cranio-facial malformations affecting the orbit and the eye. The fourth chapter deals with vascular diseases with particular emphasis on carotid-cavernous fistulas and aneurysms of the carotid system as they affect the optic or the motor nerves of the eye. The fifth chapter deals with orbito-facial injuries. The sixth chapter discusses space-occupying lesions of the orbit. Chapters seven, eight and nine deal with the eye involvement in diseases of the sinuses, nasal cavity, base of the skull and sellar region. Chapter ten deals with miscellaneous conditions as they affect the orbit or the eye.

At first a general radiologist may be confused by the collection of such an heterogeneous group of conditions and diseases in a single book. It must be admitted, however, that such a procedure represents a very practical approach in a highly specialized field.

Dr. Lombardi presents his vast experience in excellent and concise English. The illustrations are excellent and the printing is readable and clear.

Cesare Gianturco, M.D.

## BOOKS RECEIVED

Atlas of X-Ray Diagnosis of Early Gastric Cancer. By Hikoo Shirakabe, M.D., Heizaburo Ichikawa, M.D., Kenji Kumakura, M.D., Mamoru Nishizawa, M.D., Kyo Higurashi, M.D., Hisao Hayakawa, M.D., and Tadashige Murakami, M. D. Cloth. Pp. 244, with many illustrations. Price, $28.00. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1967.

Cancer in Pregnancy. By Larry McGowan, B.S., M.D., Assistant Professor, Department of Obstetrics and Gynecology, Hahnemann Medical College and Hospital, Philadelphia, Pa.; Director of Gynecology and Obstetrics, Thomas M. Fitzgerald Mercy Hospital, Darby, Pa. Cloth. Pp. 142. Price, $7.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1967.

The Office Assistant in Medical Practice. By Portia M. Frederick, Instructor in Medical Office Assisting, West Valley Junior College, Campbell, Calif.; and Mary E. Kinn, C.P.S., Chairman of the Certifying Board, American Association of Medical Assistants. Third edition. Cloth. Pp. 461, with many illustrations. Price, $7.50. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

Tumors of the Intestines. By David A. Wood, M.D., Director, Cancer Research Institute; Professor of Pathology, University of California School of Medicine, San Francisco, Calif. Atlas of Tumor Pathology, Section VI, Fascicle 22. Published by the Armed Forces Institute of Pathology. Paper. Pp. 261, with many illustrations. For sale by the American Registry of Pathology, Armed Forces Institute of Pathology, Washington, D. C. 20305, 1967.

Musculoskeletal Disorders of Childhood. Edited by Henry H. Banks, M.D. Reprinted from the Pediatric Clinics of North America, 1967, Volume 14, No. 2 and 3. Cloth. Two volume set.

Pp. Vol. I—299–532, Vol. II—533–722. Price, $12.50 the set. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

CLINICAL RADIOISOTOPE SCANNING. By Yen Wang, M.D., D.Sc. (Med.), Associate Professor of Radiology, University of Pittsburgh School of Medicine; Director of Nuclear Medicine, Presbyterian-University and Magee-Womens Hospitals, Pittsburgh; Editor, and Chairman Advisory Board, Chemical Rubber Company, Cleveland, Ohio; Consultant in Nuclear Medicine, Veterans Administration Hospital–Pittsburgh, Pa. Cloth. Pp. 246, with many illustrations. Price, $12.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1967.

THE URINARY TRACT: ROENTGEN DIAGNOSIS. By John Thornbury, M.D., Assistant Professor of Radiology, University of Washington School of Medicine; formerly Assistant Professor of Radiology, University of Iowa College of Medicine; and David A. Culp, M.D., Professor of Urology, University of Iowa College of Medicine. Cloth. Pp. 431, with many illustrations. Price, $13.00. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Ill. 60601, 1967.

INSURANCE FOR THE DOCTOR. By Harvey Saner, LL.B., and Herbert C. Lassiter, LL.B. Cloth. Pp. 193. Price, $9.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

I RADIOISOTOPI IN PNEUMOLOGIA. By B. Rescigno. Cloth. Pp. 300, with some illustrations. Archivio di Tisiologia e delle Malattie dell'Apparato Respiratorio, Napoli. Editrice Archivio di Tisiologia, Napoli, 1967.

# SOCIETY PROCEEDINGS

## MEETINGS OF RADIOLOGICAL SOCIETIES*

### UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
*Secretary*, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Jung Hotel, New Orleans, La., Oct. 1–4, 1968.

AMERICAN RADIUM SOCIETY
*Secretary*, Dr. Fernando G. Bloedorn, Division of Radiotherapy, University of Maryland Hosp., Baltimore, Md. 21201. Annual meeting: Hotel Fontainebleau, Miami Beach, Fla., April 7–11, 1968.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
*Secretary-Treasurer*, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., Nov. 26–Dec. 1, 1967.

AMERICAN COLLEGE OF RADIOLOGY
*Executive Director*, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Drake Hotel, Chicago, Ill., Feb. 6–10, 1968.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
*Secretary*, Dr. Kenneth L. Krabbenhoft, Harper Hospital, Detroit, Mich. 48201. Annual meeting: San Francisco, Calif., June 16–20, 1968.

AMERICAN BOARD OF RADIOLOGY
*Secretary*, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn.

The Fall 1967 examination will be held at the Statler Hilton Hotel, Dallas, Texas, December 4–8, inclusive. The deadline for filing applications was June 30, 1967.

The Spring 1968 oral examination will be held at the Fontainebleau Hotel, Miami Beach, Florida, June 10–14, inclusive. The deadline for filing applications is December 31, 1967. Candidates eligible for this examination will not be required to take the written examination.

The first written examination will be held the latter half of June 1968 in various centers of the country for all residents having completed 3 years of approved training as of June 30, 1968. Deadline for filing for this examination and the oral examination of December 1968 is December 31, 1967.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
*Secretary*, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
*Secretary*, Dr. J. A. del Regato, Penrose Cancer Hospital, Colorado Springs, Colo. 80907.

AMERICAN SOCIETY FOR DIAGNOSTIC ULTRASOUND
*Secretary*, Dr. Charles C. Grossman, 552 N. Neville St., Pittsburgh, Pa. 15213.

TWELFTH INTERNATIONAL CONGRESS OF RADIOLOGY
*President*, Dr. Kempo Tsukamoto, 9-1, 4-chome, Angewa, Chiba, Japan. Meeting: Hotel New Otane, Tokyo, Japan, Oct. 6–11, 1969.

NINTH INTER-AMERICAN CONGRESS OF RADIOLOGY
*Counselor for the United States*, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.
*President*, Dr. Leandro Zubiaurre, Montevideo, Uruguay. Meeting: Hotel San Rafael, Punta del Este, Uruguay, Dec. 6–12, 1967.

INTER-AMERICAN COLLEGE OF RADIOLOGY
*President*, Dr. Oscar Soto, H. Urteaga 480, Lima, Perú.

ALABAMA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Walter Brower, Birmingham, Ala. Meets time and place of Alabama State Medical Association.

ARIZONA RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Robert E. Steyskal, 550 W. Thomas Rd., Phoenix, Ariz. 85013. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS CHAPTER OF AMERICAN COLLEGE OF RADIOLOGY
*Secretary-Treasurer*, Dr. William J. Rhinehart, St. Vincent Infirmary, Little Rock, Ark. 72205.

ARKANSAS RADIOLOGICAL SOCIETY
*Secretary*, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
*Secretary-Treasurer*, Dr. Harry Z. Mellins, S.U.N.Y. College of Medicine, Brooklyn, New York 11201. Annual Meeting: Ohio State University School of Medicine, Columbus, Ohio, May 9–11, 1968.

ATLANTA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Donald R. Rooney, Burnt Hickory Road, Marietta, Ga. Meets monthly except during three summer months, on third Tuesday, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
*Secretary*, Colonel Kurt Harrell, Landstuhl Army Medical Center, Landstuhl, Germany. Meets quarterly.

BLOCKLEY RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. R. John Gould, 141 Lombardy Rd., Drexel Hill, Pa. 19026.

BLUEGRASS RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Joe Bernard, Central Baptist Hosp., Lexington, Kentucky 40503. Meets quarterly.

BROOKLYN RADIOLOGICAL SOCIETY
*Secretary*, Dr. Skottowe DePass, 69-13 Forest Ave., Brooklyn, N. Y. 11227. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
*Secretary*, Dr. Richard Sheehan, 36 Briarlee Drive, Tonawanda, N. Y. Meets second Monday evening each month, October to May inclusive.

CALIFORNIA RADIOLOGICAL SOCIETY
*Secretary*, Dr. James J. McCort, Santa Clara Valley Med. Ctr., San Jose, Calif. Meets annually during meeting of California Medical Association.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Emmett R. White, P. O. Box 303, Rutherford College, N. C. 28671. Meets every Friday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:00 NOON.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Robert A. Bornhurst, State Univ. Hospital, 750 E. Adams St., Syracuse, N. Y. 13210. Meets first Monday each month, October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
*Secretary*, Dr. Ollie E. Southard, 2787 Tudor Rd., Columbus, O. 43209. Meets second Thursday in October, November, January, and March 15 and May 19 at Fort Hayes Hotel, Columbus, Ohio.

CHICAGO ROENTGEN SOCIETY
*Secretary-Treasurer*, Dr. Fredric D. Lake, 2548 N. Lakeview Ave., Chicago, Ill. 60614. Meets second Thursday of each month, October to April, except December, at the Pick-Congress Hotel at 8:00 P.M.

CLEVELAND RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Theodore J. Castele, 18869 Canyon Rd. Parkview Park, Ohio 44126. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

COLORADO RADIOLOGICAL SOCIETY
*Secretary*, Dr. George F. Wertz, 1801 High St., Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
*Secretary*, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, George E. Plum, 712 N. Washington Ave., Dallas, Tex. 75246. Meets monthly, third Monday, at Southwest International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
*Secretary*, Dr. Robert L. Willis, Harper Hospital, Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

EAST BAY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Tom H. Piatt, 12 Camino Encinas, Orinda, Calif. 94563. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. T. F. Haase, Jr., 205 Medical Arts Building, Knoxville, Tenn. Meets in January and September.

FLORIDA RADIOLOGICAL SOCIETY
*Secretary*, Dr. John C. Jowett, Orlando, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Garth R. Drewry, Tampa General Hospital, Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

GEORGIA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Al Rayle, Jr., 938 Peachtree St., N. E., Atlanta, Ga. Meets in spring and fall with Annual State Society Meeting.

GREATER CINCINNATI RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

GREATER LOUISVILLE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

GREATER MIAMI RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Thomas W. Tufts, Broward General Hospital, 1600 S. Andrews Ave., Ft. Lauderdale, Fla. Meets monthly, third Wednesday at 8:00 P.M., at Jackson Memorial Hospital, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
*Secretary-Treasurer*, Dr. Alexander J. Link, 7215 Maryland, St. Louis, Mo. 63130.

HAWAII RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. R. K. Wang, P.O. Box 256, Wahiawa, Oahu 96786. Meets third Monday of each month at 7:30 P.M.

HEALTH PHYSICS SOCIETY
*Secretary*, John W. Thomas, Philadelphia, Pa. Annual Meeting: Denver-Hilton Hotel, Denver, Colo., June 16–20, 1968.

HOUSTON RADIOLOGICAL SOCIETY
*Secretary*, Dr. Zoltan Petrany, 1200 Moursond Drive, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at the Doctors' Club, 8:00 P.M., Houston, Tex.

IDAHO STATE RADIOLOGICAL SOCIETY
*Secretary*, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY
*Secretary*, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC.
*Secretary*, Dr. Edwin F. Koch, Jr., 915 University Ave., Muncie, Ind. 47303. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

IOWA RADIOLOGICAL SOCIETY
*Secretary*, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Robert C. Lawson, 310 Medical Arts Bldg., 10th and Horne, Topeka, Kan. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER, AMERICAN COLLEGE OF RADIOLOGY
*Secretary-Treasurer*, Dr. Ralph C. Quillin, 1221 S. Broadway, Lexington, Ky. 40504. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
*Secretary*, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
*Secretary*, Dr. Samuel Gelband. Meets second Tuesday of the month in February, April, June, October and December.

LOS ANGELES RADIOLOGICAL SOCIETY
*Secretary*, Dr. Harvey I. Meyers, 2010 Wilshire Blvd., Los Angeles, Calif. 90057. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Texas 77701.

MAINE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Robert A. Bearor, Maine Medical Center, Portland, Maine 04102. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY
*Secretary*, Dr. Henry Startzman, Medical Arts Building, Baltimore, Md.

MEMPHIS ROENTGEN SOCIETY
*Secretary-Treasurer*, Dr. Vernon L. Smith, Jr., Suite 203, 1085 Madison Ave., Memphis, Tenn. 38104. Meets first Monday of each month at John Gaston Hospital.

MIAMI VALLEY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Indiana. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MID-HUDSON RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
*Secretary-Treasurer*, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Edward A. Peterson, 572 Lowry Medical Arts Bldg., St. Paul, Minn. Meets twice annually, fall and winter.

MISSISSIPPI RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Dan T. Keel, Jr., 504 Chippewa St., Brookhaven, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

MISSOURI RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Harold B. Rapp, Cape Girardeau, Mo.

MONTANA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Clark Grimm, Great Falls, Montana. Meets at least once a year.

NEBRASKA STATE RADIOLOGICAL SOCIETY
*Secretary*, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEVADA RADIOLOGICAL SOCIETY
*Secretary*, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.

NEW ENGLAND ROENTGEN RAY SOCIETY
*Secretary*, Dr. Morris Simon, 330 Brookline Ave., Boston, Mass. 02153. Meets third Friday of each month, October through May, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society
*Secretary*, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New Mexico Association of Radiologists
*Secretary-Treasurer*, Dr. Justin J. Wolfson, Department of Radiology, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

New Mexico Society of Radiologists
*Secretary*, Dr. Phil Fox, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
*Secretary*, Dr. Arnold L. Bachman, Francis Delafield Hosp., 99 Fort Washington Ave., New York, N. Y. 10032. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M. Annual meeting to be announced.

North Carolina Chapter of ACR.
*Secretary-Treasurer*, Dr. Ira Bell, Hickory, N. C. Annual meeting to be announced.

North Carolina Radiological Society
*Secretary*, Dr. E. H. Schultz, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in the spring and fall each year.

North Dakota Radiological Society
*Secretary*, Dr. A. Ohrt, 408 Medical Arts Bldg., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
*Secretary*, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
*Secretary*, Dr. Anthony J. Tabacco, 621 Central Ave., Albany 6, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
*Secretary-Treasurer*, Ivan D. Siddons, 3701 J. St., Suite 106, Sacramento, Calif. 95816. Meets fourth Monday of Sept., Nov., Jan., March and May at the Sutter Club in Sacramento.

Northwestern Ohio Radiological Society
*Secretary*, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society
*Secretary*, Dr. Robert D. Berkebile, Elyria Memorial Hospital, Elyria, Ohio 44035.

Oklahoma State Radiological Society
*Secretary*, Dr. Donald F. Mauritson, 100 Utica Square Med. Center, Tulsa, Okla. 74114. Meets in January, May and October.

Orange County Radiological Society
*Secretary*, Dr. George W. Logan, 301 Newport Blvd., Newport Beach, Calif. Meets fourth Tuesday of every month at Orange County Medical Association Building.

Oregon Radiological Society
*Secretary-Treasurer*, Dr. Irving J. Horowitz, 2311 N.W. Northrup Str., Portland, Ore. 97210. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
*Secretary*, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
*Secretary-Treasurer*, Dr. Willis Taylor, 1118 9th Ave., Seattle, Washington. Annual meeting to be announced.

Pennsylvania Radiological Society
*Secretary*, Dr. T. Frederick Weiland, 619 Ridgeway Ave., Grove City, Pa. 16127. Annual meeting to be announced.

Philadelphia Roentgen Ray Society
*Secretary*, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
*Secretary*, Dr. Edward R. Seitz, 601 Jenkins Bldg., Pittsburgh, Pa. 15222. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiological Society of Connecticut, Inc.
*Secretary-Treasurer*, Dr. Henry J. Fox, 10 Washington Ave., Bridgeport, Conn. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
*Secretary*, Dr. Harold N. Margolin, 6159 Tulane Road, Cincinnati, Ohio. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
*Secretary*, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
*Secretary*, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
*Secretary*, Dr. Lester W. Eavenson, 2700 Napoleon Ave., New Orleans 15, La. Meets semiannually, during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey
*Secretary*, Dr. John W. Marquis, 12 Hawthorne Ave., East Orange, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island
*Secretary-Treasurer*, Dr. John M. Vesey, 1196 Elmwood Ave., Cranston, R. I.

Radiological Society of South Dakota
*Secretary-Treasurer*, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
*Secretary-Treasurer*, Dr. Robert G. Williams, The Santa Barbara Medical Clinic, P.O. Box 1200, Santa Barbara, Calif. 93102. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
*Secretary-Treasurer*, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y.

Redwood Empire Radiological Society
*Secretary*, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
*Secretary*, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
*Secretary*, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
*Secretary-Treasurer*, Dr. Robert W. Lackey, 4200 E. Ninth Ave., Denver, Colo. Annual meeting to be announced.

San Antonio-Military Radiological Society
*Secretary*, Dr. Hugho F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday of each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
*President-Secretary*, Charles R. Henkelmann, 3909 Palm Drive, Bonita, Calif. 92002. Meets first Wednesday of each month at the Town & Country Motel.

San Francisco Radiological Society
*Secretary*, Dr. H. Joachim Burhenne, Children's Hospital and Adult Medical Center, 3700 California St., San Francisco, Calif. 94119. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco, Calif. 94118.

Santa Clara County Radiological Society
*Secretary*, Dr. Bill G. Karras. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

Section on Radiology, California Medical Association
*Secretary*, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
*Secretary-Treasurer*, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
*Secretary*, Dr. Andrew F. Giesen, Jr., White-Wilson Clinic, Fort Walton Beach, Fla. 32548. Annual meeting: Miami Beach, Fla., Nov. 13–16, 1967.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
*Secretary*, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
*Secretary*, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
*Secretary*, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Jung Hotel, New Orleans, La., Sept. 30, 1968.

SOCIETY OF NUCLEAR MEDICINE
*Secretary*, Mr. C. Craig Harris, Oak Ridge National Laboratories, Oak Ridge, Tenn. *Administrator*, Mr. Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting to be announced.

SOUTH BAY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

SOUTH CAROLINA RADIOLOGICAL SOCIETY
*Secretary*, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

SOUTH DAKOTA RADIOLOGICAL SOCIETY
*Secretary*, Dr. Donald J. Peik, 1417 S. Minnesota Ave., Sioux Falls, S. Dak. Meets in spring with State Medical Society and in fall.

SOUTHERN CALIFORNIA RADIATION THERAPY SOCIETY
*Secretary-Treasurer*, Dr. Aaron G. Fingerhut, 1000 W. Carson St., Torrance, Calif. 90502. Mets quarterely.

SOUTHERN RADIOLOGICAL CONFERENCE
*Secretary-Treasurer*, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. 36604. Annual meeting: Grand Hotel, Point Clear, Ala. 36504, Jan. 26–28, 1968.

SOUTHWESTERN RADIOLOGICAL SOCIETY
*Secretary*, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

TENNESSEE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Marion E. Spurgeon, Memorial Hosp., Clarksville, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

TEXAS RADIOLOGICAL SOCIETY
*Secretary*, Dr. Herman C. Sehested, 815 Medical Arts Bldg., Fort Worth 2, Tex. Annual meeting to be announced.

TRI-STATE RADIOLOGICAL SOCIETY
*Secretary*, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

UPPER PENINSULA RADIOLOGICAL SOCIETY
*Secretary*, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

UTAH STATE RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Dr. Jack J. Davis, Salt Lake Clinic, 333 South Ninth East, Salt Lake City, Utah 84102. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

VERMONT RADIOLOGICAL SOCIETY
*Secretary*, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

VIRGINIA RADIOLOGICAL SOCIETY
*Secretary*, Dr. John M. Ratliff, Mary Immaculate Hospital, Newport News, Va.

WASHINGTON STATE RADIOLOGICAL SOCIETY
*Secretary*, Dr. Owen Marten, 930 Terry Avenue, Seattle, Wash. Meets quarterly.

WEST VIRGINIA RADIOLOGICAL SOCIETY
*Secretary*, Dr. George G. Green, Morgantown, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

WESTCHESTER COUNTY RADIOLOGICAL SOCIETY
*Secretary*, Dr. Arnold Gerson, Medical Arts Bldg., Mt. Vernon, N. Y. Meets on third Tuesday of January and October and on two other dates.

WISCONSIN RADIOLOGICAL SOCIETY
*Secretary-Treasurer*, Harold F. Ibach, 2400 W. Villard Ave., Milwaukee, Wis. 53209. Meets twice a year, May and September.

WYOMING RADIOLOGICAL SOCIETY
*Secretary*, Dr. J. D. Grant, Memorial Hosp., Sheridan, Wyo. Meets in fall with State Medical Society and in spring on call of President.

## CUBA, MEXICO, PUERTO RICO AND CENTRAL AMERICA

ASOCIACIÓN DE RADIÓLOGOS DE CENTRO AMERICA Y PANAMÁ. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
*Secretary-General*, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

SOCIEDAD DE RADIOLOGÍA DE EL SALVADOR
*Secretary*, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

SOCIEDAD DE RADIOLOGÍA DE GUATEMALA
*Secretary*, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPÍA CUBANA
*Secretary*, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

SOCIEDAD COSTARRICENSE DE RADIOLOGIA
*Secretary*, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

SOCIEDAD MEXICANA DE RADIOLOGÍA, A.C.
Coahuila No. 35, México 7, D. F.
*Secretary-General*, Dr. Ramón Ruenes.
Meets first Monday of each month.

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA
*Secretary*, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

SOCIEDAD RADIOLÓGICA PANAMEÑA
*Secretary*, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

SOCIEDAD RADIOLÓGICA DE PUERTO RICO
*Secretary*, Dr. Felipe N. de Jesús, Apt. 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

## BRITISH COMMONWEALTH OF NATIONS

ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC
*Secretary*, Dr. R. Robillard, Notre-Dame Hospital, 1560 Sherbrooke St., East, Montreal, Que., Canada. Meets four times a year.

BRITISH INSTITUTE OF RADIOLOGY
*Honorary Secretary*, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May. Annual meeting to be announced.

CANADIAN ASSOCIATION OF PHYSICISTS, DIVISION OF MEDICAL AND BIOLOGICAL PHYSICS.
*Honorary Secretary-Treasurer*, Paul M. Pfalzner, Dept. of Therapeutic Radiology, University of Western Ontario, London, Ont., Canada. Annual meeting to be announced.

EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY
*Secretary*, J. D. R. Miller, M.B., University of Alberta Hospital, Edmonton, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

FACULTY OF RADIOLOGISTS
*Honorary Secretary*, Dr. J. N. Pattinson, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting: London, England, June 21–22, 1968.

FACULTY OF RADIOLOGISTS, ROYAL COLLEGE OF SURGEONS IN IRELAND
*Registrar*, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
*Honorary Secretary-Treasurer*, Dr. Maurice Dufresne, *Associate Honorary Secretary-Treasurer*, Dr. F. Robert MacDonald, 1555 Summerhill Ave., Montreal 25, Que., Canada. Thirty-first Annual Meeting, Chateau Frontenac, Quebec, Que., March 4–9, 1968.

Montreal Radiological Study Club
*Secretary*, Dr. Leonard Rosenthall, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
*Secretary*, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie
*Secretary General*, Dr. Jacques Lespérance, 5415 Boul. L'Assomption, Montreal 26, P. Q., Canada. Meets every third Tuesday from October to April. Annual meeting to be announced.

Toronto Radiological Society
*Secretary*, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

College of Radiologists of Australasia
*Honorary Secretary*, Dr. T. P. Loneragan, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

## South America

Asociación Argentina de Radiología
*Secretary*, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
*Secretary*, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
*Secretary-General*, Dr. Miguel Maric Céntola, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiologia
*Secretary-General*, Dr. Jorge Enrique Barragué, Azeuénaga 888, Buenos Aires. Meetings are held monthly.

Sociedad Boliviana de Radiología
*Secretary*, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
*Secretary*, Dr. Armando Rocha Amoédo, Cxa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
*Secretary*, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
*Secretary*, Dr. Manuel Concha, Casilla 13426, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
*Secretary-General*, Dr. Armando Uribe, Hospital Militar Central, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
*Secretary*, Dr. Carlos Palau, Av. Bogotá 206, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
*Secretary*, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
*Secretary-General*, Dra. Ladys del Pino, Instituto de Radiología "Cayetano Heredia" Hospital Arzobispo Loayza, Lima, Perú. Meets monthly except during January, February and March.

Sociedad de Radiologica del Atlantico
*Secretary*, Dr. Raul Fernandez, Calle 40 #41–110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología Cancerología y Física Médica del Uruguay
*Secretary-General*, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
*Secretary*, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
*Secretary-General*, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad Venezolana de Radiología
*Secretary-General*, Dr. Luis F. Muro, Apartado No. 9362 Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

## Continental Europe

Österreichische Röntgen-Gesellschaft
*President*, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. Annual meeting to be announced.

Société Belge de Radiologie
*General Secretary*, Prof. Simon Masy, Louvain, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
*Permanent Secretary*, Dr. Jaques Sauvegrain, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France.
*General Secretary*, Dr. H. Ludin, Department of Roentgenology, Basler Kinderspital, Basel, Switzerland. Annual meeting to be announced.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
*Secretary-General*, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
*Secretary*, Dr. Robert Poch, Praha 12, Srobárova 50, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
*Secretary*, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut, Lagenbeckstr. 1, Mainz, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
*Secretary*, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
*Secretary*, Dr. H. F. O. Stricker, Schalklaar, Netherlands.

Scandinavian Radiological Society
*Secretary-General*, Dr. C.-E. Unnérus, Hagalund-Tapiola, Havsvindsvägen 5 C., Finland. Annual meeting: Copenhagen, Denmark, 1968.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
*Secretary*, Juan Gomez Lopez, Villanueva, 11, Madrid 1. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid. Annual general meeting to be announced.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
*Secretary*, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

## Asia

Indian Radiological Association
*Secretary*, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

Indonesian Radiological Society
*Secretary*, Professor Sjahriar Rasad, Taman Tjut Mutiah I Diakarta, Indonesia

Iranian Radiological Society
*Secretary*, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

## STATEMENT OF OWNERSHIP, MANAGEMENT AND CIRCULATION

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7. OWNER *(If owned by a corporation, its name and address must be stated and also immediately thereunder the names and addresses of stockholders owning or holding 1 percent or more of total amount of stock. If not owned by a corporation, the names and addresses of the individual owners must be given. If owned by a partnership or other unincorporated firm, its name and address, as well as that of each individual must be given.)*

| NAME | ADDRESS |
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VOL. 101, NO. 3

# ABSTRACTS OF RADIOLOGICAL LITERATURE

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## ROENTGEN DIAGNOSIS

### SKELETAL SYSTEM

SILVERSTEIN, MURRAY N., and KELLY, PATRICK J. Osteoarticular manifestations of Gaucher's disease. *Am. J. M. Sc.*, May, 1967, *253*, 569–577. (From: Mayo Clinic and Mayo Foundation: Section of Medicine and of Orthopedic Surgery, Rochester, Minn.)

A clinical appraisal of the osteoarticular manifestations of Gaucher's disease was made with respect to 38 patients who were seen at the Mayo Clinic. About 75 per cent of children and adults had osseous lesions on roentgenographic examination, but only 35 per cent of the patients having roentgenographic skeletal surveys had any complaints relative to bone. The most frequently involved bone was the femur and the most frequent roentgenographic finding was a fusiform widening of the lower end of the femur, the so called "Erlenmeyer-flask" deformity. Lesions were also observed in the tibia, humerus, vertebra, pelvis and skull. Isolated defects of the skull are extremely rare.

The symptoms from osseous lesions which occur in Gaucher's disease appear to be related to the presence of avascular necrosis of the femoral head, pathologic fractures, vertebral involvement, and involvement of the humeral head with narrowing of the adjacent joint space. Previous authors have also reported severe serious vertebral involvement, with collapse, gibbus formation and cord compression. In this series no patients were seen who had the previously reported syndrome of periostitis and severe constitutional reaction which has been associated with Gaucher's disease.

Only 4 of 10 patients having no osseous changes were anemic, whereas 78 per cent of those patients having osseous changes were anemic. Furthermore, none of 10 patients without osseous changes had splenectomy, whereas 10 of 28 patients having such changes had submitted to this procedure. The data suggest that anemia and splenectomy may contribute to the osseous changes in Gaucher's disease; the former by stimulating activity of more marrow space for invasion by Gaucher's disease, and the latter by eliminating a large reservoir for Gaucher's disease.—*E. Nicholas Sargent, M.D.*

CRANE, A. REYNOLDS, and SCARANO, JOSEPH A. Synovial cysts (ganglia) of bone: report of two cases. *J. Bone & Joint Surg.*, March, 1967, *49A*, 355–361. (Address: A. Reynolds Crane, M.D., Ayer Clinical Laboratory, Pennsylvania Hospital, Eighth & Spruce Streets, Philadelphia, Pa. 19107.)

The occurrence of extra-articular synovial extrusion in Baker's cyst or villonodular synovitis is well known. This article calls attention to the entity of proliferation of synovium into bone to produce a benign cystic lesion.

Two cases are reported with lesions in the distal tibia, and a lesion in the carpal lunate. In both cases, pathologic confirmation was obtained following surgery. One case, in fact, was associated with a soft tissue ganglion.

A discussion of the possible etiologic factors details several possible modes of origin. The authors seem to favor the origin due to synovial extrusion or projection of synovial fluid through a defect in bone, resulting in the development of the cyst.

The histologic picture is typical of that seen in soft tissue ganglia.

Radiographically, synovial cysts present as expanding lytic lesions with a shell of sclerosed bone adjacent to a joint surface. A connection with the joint may be evident.

The preferred treatment is enucleation and packing with bone chips.—*Burton Must, M.D.*

JACOBSON, DAVID P., and MCCLAIN, EDWARD J. Hypophosphatasia in monozygotic twins: a case report. *J. Bone & Joint Surg.*, March, 1967, *49A*, 377–380. (Address: David P. Jacobson, M.D., Department of Orthopedics, Children's Hospital, DeSoto Street, Pittsburgh, Pa. 15213.)

The authors believe that this is the first case report of hypophosphatasia in identical twins. Rathbun described this rare metabolic disorder in 1948. Hypophosphatasia is characterized by abnormal ossification and decreased serum alkaline phosphatase. There is also associated urinary excretion of phosphoethanolamine.

The patients lost their teeth over a 3 week period at the age of 15 months. The mother felt that the locomotor development had also lagged behind that of their siblings.

On examination, the twins demonstrated many findings associated with this disorder. There was poor skeletal growth, with resulting small stature, bone deformity, craniosynostosis (with secondary mental retardation) and premature loss of deciduous teeth.

The laboratory findings were normal except for hypophosphatasia and normal or slightly elevated serum calcium and phosphoethanolamine in the urine.

Roentgenograms revealed changes similar to rickets except for cranial suture closure, loss of deciduous teeth, and a tongue-like protrusion of uncalcified cartilage extending from the mid-portion of the widened epiphysis into the metaphysis.

Physical examinations of the parents and siblings were normal. Roentgenographic studies of knees and wrists of the parents and 7 siblings were unremarkable.

Serum alkaline phosphatase of the father and

mother were 1.4 and 0.3 Bodansky units respectively (normal value is 2 to 8 Bodansky units). Two other female siblings had low level of phosphatase.

The authors conclude that the occurrence in identical twins, with evidence of the disease in heterozygous siblings and in one parent, and the normal karyotype chromosome studies all point to the fact that this is an autosomal recessive disease.—*Mary Ann Hilz, M.D.*

JOHNSON, HUGH A. Formation of a functional thumb post with sensation in phocomelia. *J. Bone & Joint Surg.*, March, 1967, *49A*, 327–332. (Address: Rockland Memorial Med. Building, 2500 N. Rockton Avenue, Rockford, Ill. 61103.)

The author reports a case of formation of a functional thumb post in a 2 year old child, who was born with congenital absence of the distal three-quarters of one hand. This was accomplished by 3 operators in an 8 week period by using a 5 cm. portion of a resected rib and skin grafting.

The child was followed for a 6 year period and was found to be using the hand far beyond expectation. There is good sensation over the entire area.

Fourteen drawings, photographs, and roentgenographic reproductions show the entire process of the formation of the hand.

The author feels that this surpasses any prosthetic appliance for mobility and grasp.—*Alan Belcher, M.D.*

ARKLESS, RICHARD. Rheumatoid wrists: cineradiography. *Radiology*, March, 1967, *88*, 543–549. (From: Radiology Department, University of Washington Hospital, Seattle, Wash.)

The author reports dynamic alterations noted with cineradiography in the involved wrists of 55 patients with rheumatoid arthritis. This method is most useful in diagnosing the disease in its early stages, when routine roentgenograms reveal only minimal or no wrist abnormalities.

Following a description of the dynamics of the normal wrist, changes in the rheumatoid wrist are discussed. These include: diminished or absent rotation of the distal navicular in and out of the palm with radioulnar wrist deflections; failure of the lunate to return to normal position with ulnar wrist deflection; restricted dorsopalmar lunate motion; navicular-lunate and radio-ulnar separations; abutment of the triquetrum against the distal ulna and, at times, the ulnar surface of the radius; and excessive overriding of the hamate on the lunate with ulnar deflection. These abnormal dynamics occur either singly or in some combination.

Further, it was noted that where areas of sclerosis existed in the lunate and triquetrum, and adjacent radius and ulna, the dynamics of these carpal bones were such that they tended to rub or lift up and hit down against their proximal articulations. It is postulated that these traumatic movements may account for the paradoxical secondary hypertrophic and sclerotic changes seen in this disease of primary hypomineralization of bone.

These same dynamic alterations may occur in traumatic and gouty arthritic wrists but are not seen in the wrists of patients who have bone and joint abnormalities secondary to steroid therapy for conditions other than rheumatoid arthritis. Thus, the changes in rheumatoid wrists are probably not related to steroid therapy, nor are they confined entirely to rheumatoid arthritis.—*V. Brasseur, M.D.*

MCARTHUR, R. G., and EDWARDS, J. H. de Lange syndrome: report of 20 cases. *Canad. M. A. J.*, April 29, 1967, *96*, 1185–1198. (Address: Dr. McArthur, Fellow in Pediatric Endocrinology, Mayo Clinic, Rochester, Minn.)

In 1933 Cornelia de Lange described the condition in 2 infant girls from Amsterdam under the name Typus Degenerativus Amstelodamensis. The term "de Lange syndrome" and "Amsterdam dwarf" are in common use. The latter term is not desirable as dwarfism is not invariable.

The characteristic face has a low hairline, prominent eyebrows which meet in the midline, long curved eyelashes, uplifting nose with upturned nostrils, flat nose bridge, narrow palpebral fissures, epicanthic folds, elongated philtrum, thin upper lip, carp-like mouth, and a receding chin.

Microcephaly is usual. The ears are low set and the neck often short and webbed. Hypertrichosis is particularly evident in the nape of the neck, back and forearms. The hair is low posteriorly. The hands are small with short tapering fingers, a small incurved little finger and a proximally placed thumb with a small thenar eminence. Syndactyly and gross deformities including oligodactyly, micromelia and phocomelia may occur. Syndactyly of the second and third toes is common, as is limitation of movement at the elbow joint with impairment of supination. The skin is dry and rough. Congenital heart disease, usually ventricular septal defect, may occur and an increased incidence of gastrointestinal anomalies is reported. In the male the testes usually fail to descend and hypospadias may be present.

The roentgenologic studies show a number of deformities, the most consistent of which are a small or deformed skull, delayed ossifications, short metacarpals (particularly the first), short metatarsals, short phalanges, carpal bone abnormalities and posterior subluxation of the radius at the elbow joint.

The diagnosis is made on the basis of the clinical

many believe or is it of multicentric origin with random lymphatic and blood stream spread? Based on the information obtained from lymphography, liver and spleen scanning (and, more recently, scanning of mediastinal lymph nodes), as well as an analysis of the site of initial occurrence and recurrence, he concludes that the evidence favors multicentric origin with random spread. Support of this position is seen in the demonstration of tumor cells in the blood stream in patients with known Hodgkin's disease.

Expanding on these findings the author presents his own theory of origin and dissemination. He proposes that since the system affected is widely spread throughout the body and may be subject to the same influences at any point, there is no reason why the disease cannot arise multicentrically in some cases just as other cancers do. Tumor cells could then spread throughout the blood stream with a definite "homing" tendency to certain other target areas.—*Howard West, M.D.*

Smithers, D. W. Hodgkin's disease—II. *Brit. M. J.*, May 6, 1967, *2*, 337–341. (From: Radiotherapy Department, Royal Marsden Hospital and Institute of Cancer Research: Royal Cancer Hospital, London S.W.3, England.)

Part II of the Lecture deals initially with some general manifestations of Hodgkin's disease, particularly anergy (as shown by the acceptance of foreign skin grafts by many Hodgkin's sufferers). Its well known association with several other diseases, notably herpes zoster, infectious mononucleosis and sarcoid—all of which are associated with disorders of the immune mechanism—is also documented.

Pursuing the problem of autoimmune disease the author points out the similarity between Hodgkin's disease and a series of immunologic disorders in animals known as homologus disease. This latter disease is characterized principally by wasting, anemia and lymphoid depletion and is apparently due to the reaction of foreign lymphoid cells against the host.

Based on this information, an experiment was devised at the Royal Marsden Hospital in which lymphoid cells from a parental strain were injected into $F_1$ hybrid rats after whole body irradiation. Some of the rats acquired the ability to accept repeated injections and several of these eventually developed either leukemia or splenomegaly and thymic enlargement. Subsequently the experiment was refined and repeated without the use of irradiation. Other workers in the field, using similar techniques, have subsequently produced what is believed to be the first Hodgkin's disease—like tumors in laboratory animals.

Another related development has been the production of a disease similar to Burkitt's lymphoma in mice injected with spleen cells of other mice suffering from an acute infection of viral etiology (reovirus 3). Finally, homologous disease has now been shown to progress to lymphoma production which mimics lymphatic leukemia, lymphosarcoma, reticulum cell sarcoma, Burkitt's lymphoma and Hodgkin's disease.

The author concludes that Hodgkin's disease should be classified as an "immunoproliferative disorder" (to use Dameshek's term) and that one way the body's lymphocytes could be immunologically altered to cause such a disorder would be through certain viruses which have a preference for access through the upper respiratory tract.—*Howard West, M.D.*

Pike, M. C., Williams, E. H., and Wright, Barbara. Burkitt's tumour in the West Nile district of Uganda 1961–65. *Brit. M. J.*, May 13, 1967, *2*, 395–399. (From: Medical Research Council's Statistical Research Unit, University College Hospital Medical School, London W.C.1, on secondment to Department of Pathology, Makerere Medical School, P.L. Box 2072, Kampala, Uganda.)

In an effort to test the hypothesis that Burkitt's tumor is virus induced the authors have analyzed the cases reported in a region of Uganda known as the West Nile District.

The 3 hospitals within this district reported a total of 51 known or suspected cases of this disease from 1961 to 1965. The authors have analyzed each of these cases according to time of detection and location. Two methods of statistical analysis were then applied to determine whether the distribution of the cases indicated spread by an infectious agent rather than a random appearance throughout the area.

Both methods of analysis provide strong evidence of epidemic characteristics for this entity. The authors also suggest the possibility of "low" and "high" intensity areas within the district itself.—*Howard West, M.D.*

Perez, Carlos A., Vietti, Teresa, Ackerman, Lauren V., Eagleton, Mark D., and Powers, William E. Tumors of the sympathetic nervous system in children: an appraisal of treatment and results. *Radiology*, April, 1967, *88*, 750–760. (From: E. Mallinckrodt Institute of Radiology and the Department of Pediatrics, and Surgical Pathology, Washington University School of Medicine, Saint Louis, Mo.)

Forty-two children with tumors of the sympathetic nervous system were treated at the St. Louis Children's Hospital and the Mallinckrodt Institute of Radiology from 1949 through December, 1963.

Thirty-six of these were included in this study; 6 were excluded because of initial treatment elsewhere or their pathologic slides were not available for review.

The main feature of these tumors, including age and sex distribution and clinical laboratory findings, are summarized in the first two tables. Their sites of metastatic spread and pathologic classification are listed in the third and fourth tables respectively. The roentgenographic findings revealed that in all 9 cases of mediastinal tumors, a mass was in evidence posteriorly sometimes eroding adjacent vertebrae and ribs, and in one case it actually invaded the spinal canal. In the 25 cases of abdominal tumors, a suprarenal mass was observed in all on either roentgenograms of the abdomen or intravenous pyelograms, and 12 of these showed gross calcifications. It was noted that the mass most frequently displaced the kidney inferiorly and anteriorly.

Since this study is concerned with tumors of the sympathetic nervous system, only ganglioneuromas, ganglioneuroblastomas, and neuroblastomas are included. They are placed into three groups: (a) well encapsulated without metastatic spread; (b) extension to contiguous areas without metastatic spread; and (c) disseminated distant metastases. The first two groups received aggressive postoperative irradiation, the last group received lesser amounts of radiation.

Of the 36 cases reported, 2 received no definitive therapy and in 3 irradiation was interrupted because of far advanced disease. Surgery alone was performed in 2 cases, postoperative irradiation in 34 cases, adjuvant chemotherapy in 4 cases, and vitamin $B_{12}$ was given in 2 cases. The techniques of irradiation are described, including preoperative, postoperative management of the metastatic lesions and the time dose relationships.

The results are analyzed and tabulated as well as the factors influencing the prognosis and the complications and the sequelae of treatment. The crude two-year survival rate was 55.6 per cent and the crude five-year rate was 50 per cent. Forty per cent of patients with distant metastases survived tumor free 2 or more years. Two cases of mediastinal neuroblastoma and one adrenal neuroblastoma showed histologic evidence of maturation to a ganglioneuroma.

Aggressive irradiation is advocated in the treatment of these tumors, even when distant metastases are present, depending upon the patient's clinical condition.

Six cases are reported in sufficient detail to reveal their results including the complications from treatment. An excellent discussion concludes the study.—*P. M. Kroening, M.D.*

JOHNSTONE, GEORGE. Prepubertal gynaecomastia in association with an interstitial-cell tumour of the testis. *Brit. J. Urol.*, April, 1967, *39*, 220–221. (Address: P.O. Box 635, Queenstown, Tasmania, Australia.)

Interstitial-cell tumor of the testis in prepubertal children is rare and only 38 cases have been reported in the world literature. While all cases have demonstrated precocious virilism, only 4 have shown concomitant gynecomastia.

The author reports a fifth case, a 5 year old African male with combined precocious virilism and gynecomastia secondary to interstitial-cell tumor of the testis.

The patient had clinical features attributable to oversecretion of both androgens and estrogens. The appearance of gynecomastia has been the result of an excess of estrogens. The estrogen excess may alter the psycho-sexual effects of this tumor, but not the physical virilizing effects.

The child also had elevated 17-ketosteroid levels. There appears to be, however, no absolute relationship between the level of 17-ketosteroid excretion and the degree of sexual precocity.

It is noteworthy that the bone age is usually advanced in patients with interstitial-cell tumor of the testis. This finding was also noted in the patients with gynecomastia.

The hormonal and physical findings in 18 other cases of interstitial-cell tumor are listed, including the other 4 cases showing gynecomastia. No cases of malignancy have been observed so far in these prepubertal tumors; however, this possibility could only be excluded after prolonged observation.—*Captain Edward Savolaine, MC*

RILEY, LEE H., JR., HARTMANN, WILLIAM H., and ROBINSON, ROBERT A. Soft-tissue recurrence of giant-cell tumor of bone after irradiation and excision. *J. Bone & Joint Surg.*, March, 1967, *49A*, 365–368. (Address: Lee H. Riley, Jr., M.D., The Johns Hopkins Hospital, Baltimore, Md. 21205.)

Two cases of soft tissue recurrence of giant cell tumor of bone after irradiation and excision are reported

One case, a 36 year old female, was seen for recurrence 2 years after an unknown amount of radiation was given for a giant cell tumor of the innominate bone. Total excision of the lesion was performed. Approximately 6 months later, submucosal nodules were removed from the vagina along the line of resection. Additional soft tissue nodules were removed 1 and 2 years later. Ten years after primary resection, the patient was free of disease.

The second case was a 25 year old male who had resection of a giant cell tumor in the head of a rib at age 13. Postoperative irradiation was given (2,125 r in 2 weeks). Five years later, recurrent tumor was excised from the thoracic epidural space and 4,000 r $Co^{60}$ radiation was given. Two years later, metastases

to the vertebral bodies were found and a posterior spinal fusion was performed. Subcutaneous nodules were noted in the old incision. The patient died 2 years later with extensive disseminated tumor.

Since there is a definite possibility of soft tissue implantation of the tumor cells, the authors suggest measures to prevent contamination. En bloc excision should be performed when possible. With curettage, one should protect the soft tissue, irrigate thoroughly and change towels, gowns, and gloves prior to reconstruction of the wound.

Although substantiating evidence is not available, radiation therapy was mentioned as a possible factor in the development of soft tissue recurrences and malignant change in giant cell tumors.—*Everett E. Smith, M.D.*

TELFORD, W. M., CRAWFORD, J. E., ZWICK, H. H., and STEPHENS-NEWSHAM, L. G. A linear electron accelerator for medical purposes. *J. Canad. A. Radiologists*, March, 1967, *18*, 278–282. (From: Department of Therapeutic Radiology, Royal Victoria Hospital, Montreal, Quebec, Canada.)

This article presents data on the design and operation of an electron accelerator designed and constructed by the authors. These technical considerations are likely of limited interest to most therapeutic radiologists. The linear accelerator was designed to operate at 8 mev. and makes use of an S-band magnetron. Microwave pulses are fed into the accelerator such that electron currents up to 65 microamperes have been obtained.

For medical purposes the electrons bombard a gold target to produce bremsstrahlung. The head of the accelerator is able to rotate and is equipped with collimator jaws made of uranium metal. The problem of neutron production by photoscintigration of uranium is considered.

A statement is made in the section on neutron production that a dose rate of 2 rem/minute may be safely neglected. This likely is a misprint and what is meant is 2 millirem/minute.—*Merle K. Loken, Ph.D., M.D.*

**SQUIBB**

a research concept in contrast visualization

# for better definition of disease: documenting differences between contrast agents

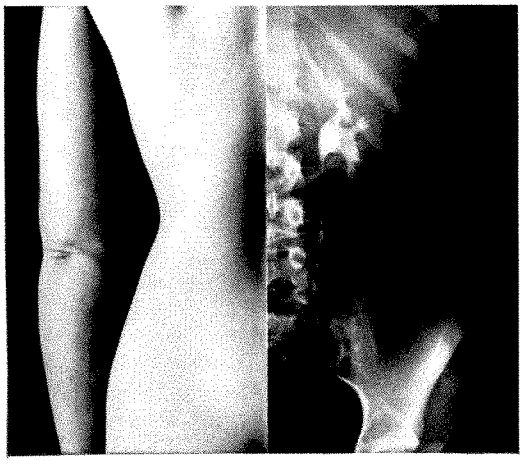
**No. 1 in a series:**

Renografin-60 (meglumine diatrizoate injection) preferred in a study of pyelographies of 2,234 patients.

In a new large scale study,[1] "...to determine which medium would produce adequate visualization of the urinary tract with the fewest toxic effects on the patient," Macht *et al.* have compared Renografin-60 with 2 other contrast agents.

Data were analyzed in 2,234 unselected (consecutive) patients, according to age, sex, and general disease group for the study population as a whole. The first 683 patients received 50% diatrizoate sodium solution, the next 921 patients received Renografin-60, and the final 630 patients received 66.8% sodium iothalamate solution.[1]

**criteria for quality and comparative safety** The contrast agents were evaluated for quality of diagnostic films as follows: films showing a dense concentration of contrast medium with filling and visualization of all major and minor calyces, infundibula, pelves and almost all of each ureter were listed as "good"; films showing less concentration with incomplete visualization of all portions of urinary tract but sufficient to produce diagnostically adequate films were listed as "fair"; films showing unsatisfactory visualization of urinary tract, or films which could not be interpreted, as "poor." The media were also evaluated as to incidence of the following side effects: nausea, vomiting, fainting, shock or severe reaction, hiccups, hives, pain in arm, sneezing, hot flushes, stuffiness of nose or ears.[1]



The following chart* shows comparative results of the 3 media according to age category:

*quality of pyelograms and side effects expected in standard population of 1,000 patients by age, according to type of medium used*

| medium used and type of patient | standard population | quality of pyelograms good | fair | poor | with side effects |
|---|---|---|---|---|---|
| meglumine diatrizoate | | | | | |
| all ages, | 1,000 | 827 | 123 | 50 | 41 |
| 0-19† | 126 | 122 | 3 | 1 | 3 |
| 20-49 | 415 | 374 | 35 | 6 | 27 |
| 50-69 | 315 | 240 | 51 | 24 | 9 |
| 70 or older | 144 | 91 | 34 | 19 | 2 |
| diatrizoate sodium | | | | | |
| all ages, | 1,000 | 782 | 134 | 84 | 72 |
| 0-19 | 126 | 115 | 6 | 5 | 7 |
| 20-49 | 415 | 378 | 26 | 11 | 40 |
| 50-69 | 315 | 227 | 64 | 24 | 19 |
| 70 or older | 144 | 62 | 38 | 44 | 6 |
| sodium iothalamate | | | | | |
| all ages, | 1,000 | 883 | 81 | 36 | 54 |
| 0-19 | 126 | 122 | 4 | 0 | 7 |
| 20-49 | 415 | 393 | 20 | 2 | 29 |
| 50-69 | 315 | 269 | 32 | 14 | 13 |
| 70 or older | 144 | 99 | 25 | 20 | 5 |

*Adapted from Macht[1] †In some patients in this age group, higher than recommended doses were used.

**In theory:** "The choice of contrast agent should ideally be individualized according to the age, sex, and disease group of the patient in order to obtain a high probability of complete visualization of the urinary tract with low probability of adverse side effects."[1]

**In practice:** "The choice is to be made...on the basis of the agent which gives the best concentration and the fewest side effects in the greatest number of patients regardless of age, sex, or disease category."[1]

**In order of preference:** Although Renografin-60 was not rated highest in all categories, the authors feel that their preference of the contrast media for intravenous pyelography would be: first, Renografin-60; second, sodium iothalamate; third, diatrizoate sodium.[1]

For brief summary of prescribing information, please refer to the end of this advertisement.

# definition of safety...

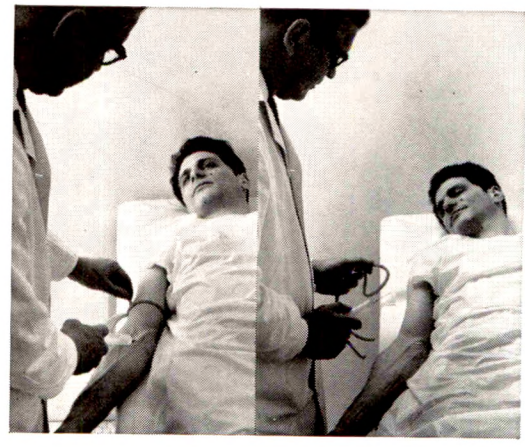
**demonstrated in animals...** Since meglumine diatrizoate is also used for cerebral angiography, toxicity studies of administration via the carotid artery are therefore of interest. Fischer and Eckstein[2] designed angiographic studies in animals in which procedures were very similar to clinical angiography, and which yielded data that was quantitative, graphic and nonsubjective.[2] According to the authors: "We selected the alterations in arterial blood pressure, venous pressure, heart rate and rhythm, the electrocardiogram and end-expiratory $CO_2$ concentration resulting from experimental cerebral angiography as refined, sensitive indications of the toxicity of a particular contrast material."[2] Their results of measured cardiovascular functions in dogs indicated that meglumine diatrizoate was far less toxic than four other contrast media.[2] As Fischer and Cornell reported in a later study: "Despite the testing of more highly concentrated solutions and larger doses, the cardiovascular responses [in dogs] from methylglucamine [meglumine] salts were much less than from sodium salts, an observation consistent with previous experiments."[3]

Other investigators have documented the comparative safety of meglumine salts in experimental studies. In order to determine reaction and tolerance of the intestines, Cooley[4] injected meglumine diatrizoate into mesenteric arteries of dogs and found no damage. Gensini *et al.*[5] reported that cardiovascular responses with it were almost identical to blood transfusions.

**theory of lower toxicity with meglumine...** Gensini and DiGiorgi have offered a hypothesis to explain their findings of lesser toxicity with experimental intravascular injections of methylglucamine (meglumine) salts. "When a relatively undiluted amount of sodium salts of a contrast agent is injected in an artery and carried by the blood stream toward the capillary bed, its molecules rapidly dissociate and readily diffuse through the capillary membrane and into the tissue. There, both the toxic effect of the iodine-containing organic radical and the increased concentration of sodium will readily manifest themselves. At equal concentrations of sodium, the end results will closely reflect the intrinsic toxicity of the iodine-containing organic radical on the tissues...."[5]

"In the case of the methylglucamine compounds, the same dissociation takes place. However the larger methylglucamine molecule, rich in hydrogen bonds, apparently either limits the migration of the organic radicals outside the vessel or at least minimizes their effects on the cellular metabolism."[5]

**proved in practice...** Paralleling similar findings in animals, clinicians have reported a generally lower



incidence of untoward reactions with Renografin-60 in urologic and cerebrovascular use. However, as with all intravascularly injected contrast agents, the possibility of severe reactions should be kept in mind (see Contraindications, Precautions and Side Effects below). In one study of over 600 urologic patients, the investigators reported that Renografin-60 produced urograms of diagnostic quality with a 6% incidence of side effects. The authors concluded: "It is hard to believe that any drug introduced intravenously could be so well borne by so many patients...."[6]

In a 74-patient study,[7] comparing Renografin-60 with diatrizoate sodium in carotid arteriography, Shealy commented: "With confused patients who are to have arteriography under local anesthesia, it is particularly desirable to have an agent that causes little pain." In this study, since "...60 per cent Renografin has resulted in considerably less pain than 50 per cent or 45 per cent [diatrizoate sodium]...we have converted to the routine use of 60 per cent Renografin for carotid arteriography; an additional 1,500 arteriograms done with 60 per cent Renografin have been quite satisfactory."[7]

**better tolerated even in pediatrics...** Citing some difficulties in administering contrast agents intravenously to children, Strasser *et al.*[8] selected Renografin-60 for intramuscular use in excretion urography in 16 pediatric patients because of the mild and relatively few reactions consistently associated with its use. The authors concluded: "The almost complete absence of any kind of local effect from its injection into the gluteal muscle and the absence of any serious reactions, local or systemic, indicate the safety of the medium."[8]

For brief summary of prescribing information please refer to the end of this advertisement.

# definition of efficacy...

**a thoroughly investigated meglumine salt** Extensively evaluated for over a decade, Renografin has been consistently shown to yield a high percentage of diagnostic quality films in many phases of contrast visualization.

Upon intravenous injection, Renografin is rapidly carried to the kidneys and is so well concentrated that renal passages—including renal pelvis, ureters and bladder—may be clearly visualized. This medium also provides high contrast vasography in visualization of the cerebral vessels and the peripheral arteries and veins.

**proved diagnostic excellence...** In a comparative study[9] of 3 contrast agents used in cerebral angiography by Doehner (comprising a cross section of an average neurosurgical practice), Renografin-60 was equal in the arterial phase and slightly superior in the venous phase of the examination.

Findings of a previously cited study by Orr *et al.*,[6] in intravenous pyelography, also attest to the diagnostic excellence of Renografin-60. "Satisfactory roentgenograms of the kidneys were obtained in 636 (97%) of the cases, demonstrating the great diagnostic value of this procedure." And, as noted previously, Shealy found the contrast agent to be "quite satisfactory" in 1,500 carotid arteriograms.[7]

**References: 1.** Macht, S. H.; Williams, R. H., and Lawrence, P. S.: Amer. J. Roentgen. *98*:79 (Sept.) 1966. **2.** Fischer, H. W., and Eckstein, J. W.: Amer. J. Roentgen. *86*:166 (July) 1961. **3.** Fischer, H. W., and Cornell, S. H.: Radiology *85*:1013 (Dec.) 1965. **4.** Cooley, R. N., *et al.*: Angiology *15*:107 (Mar.) 1964. **5.** Gensini, G. G., and DiGiorgi, S.: Radiology *82*:24 (Jan.) 1964. **6.** Orr, L. M.; Campbell, J. L., and Thomley, M. W.: J.A.M.A. *169*:1156 (Mar.) 1959. **7.** Shealy, C. N.: J. Neurosurg. *20*:137 (Feb.) 1963. **8.** Strasser, N. F., *et al.*: Radiology *79*:408 (Sept.) 1962. **9.** Doehner, G. A., and Brugger, G. E.: New York J. Med. *60*:4022 (Dec.) 1960.

**Contraindication**

A history of sensitivity to iodine per se or to other contrast media is not an absolute contraindication to the use of meglumine diatrizoate.

**Precautions and Side Effects**

Severe, life-threatening reactions are rare; when they occur they suggest hypersensitivity. A personal or family history of asthma or allergy warrants special attention and may predict, more accurately than pretesting, the likelihood of a reaction, although not the type nor severity of the reaction in the individual.

The value of any pretest is questionable. The pretest most performed is the slow injection of 0.5-1.0 cc. of the preparation into a peripheral vein. An impending reaction is often indicated by transient burning and flushing, pain, "jump-like" reactions, respiratory difficulty, faintness, sneezing, itching, nausea, vomiting or urticaria. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. Antiallergic drugs may be used to advantage. In a few cases, the reactions to the test dose have been extremely severe.

The more serious anaphylactoid reaction requires immediate treatment and may occur despite a negative sensitivity test. An emergency tray consisting of vasopressor drugs, epinephrine hydrochloride 1:1000, methoxamine (Vasoxyl) or metaraminol bitartrate (Aramine), and glucose and saline is recommended. Oxygen and instruments to guarantee a clear airway must be readily available.

Caution must be exercised, especially in cerebral angiography in extreme age, in severely debilitated patients and in those with marked or severe hypertension, advanced arteriosclerosis, cardiac decompensation, recent cerebral embolism, or thrombosis, chronic pulmonary emphysema and in cyanotic infants.

For full details the Package Insert should be read.

**Supply:** Renografin-60 is supplied in vials and ampuls as a sterile aqueous solution providing 60% meglumine diatrizoate containing approximately 29% firmly bound iodine.

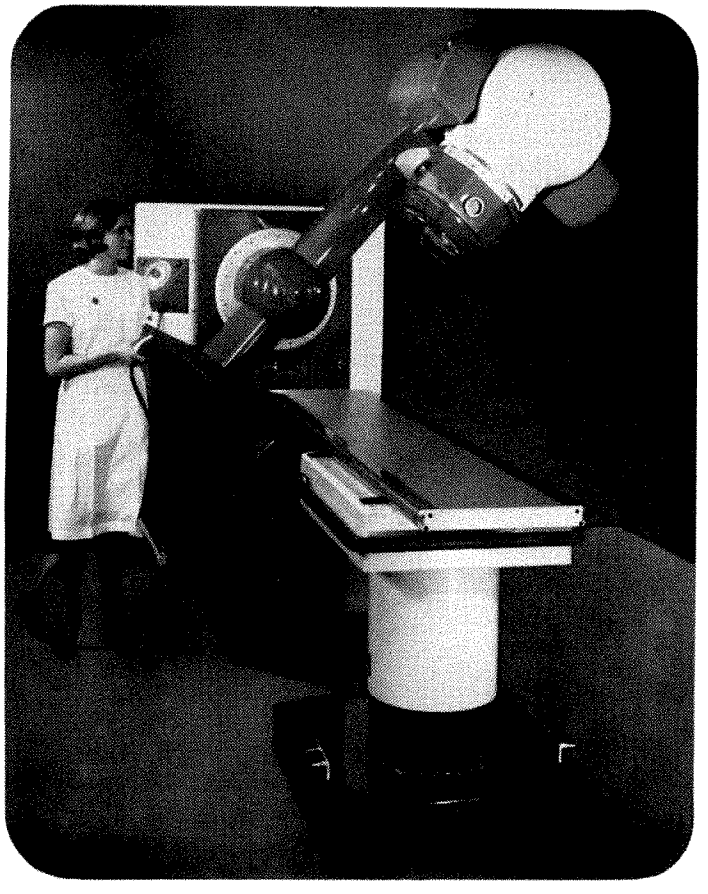
Available in 30 cc. vials or 25 cc. ampuls.

**in excretion urography**
**cerebral angiography**
**peripheral arteriography**
**and venography**

# Renografin®-60

## Squibb Meglumine Diatrizoate Injection U.S.P.

SQUIBB 'The Priceless Ingredient' of every product is the honor and integrity of its maker.



# THIS UNIT MAY NOT MEET YOUR REQUIREMENTS,

## BUT WE HAVE ONE THAT WILL!

422

This is the most sophisticated of the Cobalt 60 units derived from our basic model.
It was installed with no brickwork or cementing whatsoever.
Some of its specific characteristics:
irradiation head accommodates a source of 4500 RHM capacity - the head swivels side-ways and tilts back and forward, both movements power driven with automatic stops at the zero positions, continuous rotation without end-stop for full flexibility in operation.
The counterweight carries a light-pointer.
These features, important as they are cannot, of course, possibly cover all the widely divergent requirements of modern radiotherapy. That is why we offer a range of sixteen versions extending from the simple, basic model to the top version shown here.

**The elements of choice:**

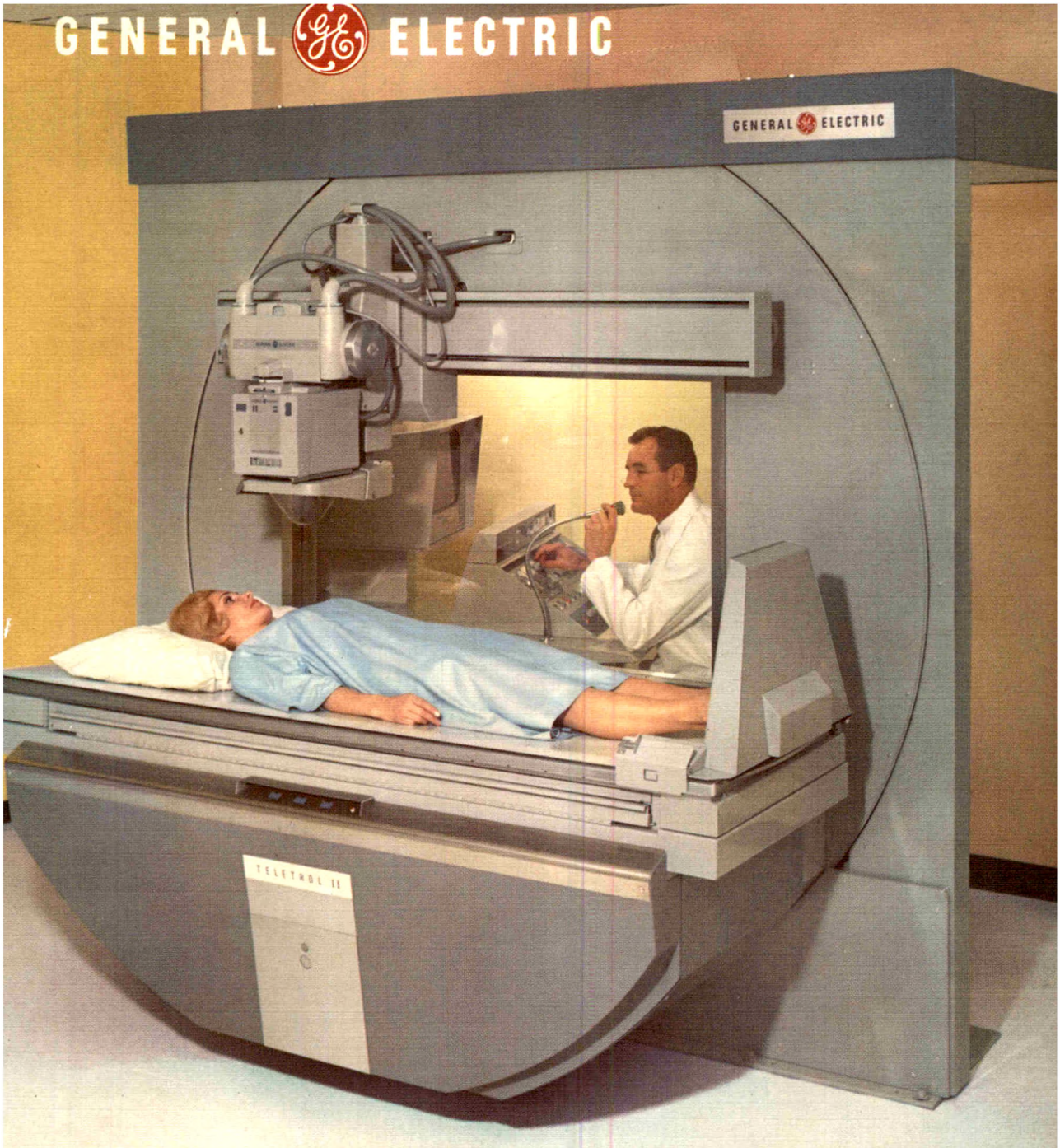
- maximum source capacity of 3000 RHM or 4500 RHM
- beam absorber or counterweight
- source-rotation axis distance 60 or 75 cm
- side-ways head-swivel only - or combined with forward and backward tilt
- motor-drive or manual operation for the combined head movements.

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## DIAGNOSTIC X-RAY SYSTEM

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- Remote Patient Positioning
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- Remote Bucky/Spot-Filming
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- Remote Compression Control

Here's the diagnostic x-ray system that speeds patient handling, eliminates cumbersome lead clothing, while maintaining close patient/radiologist

## The Completely Automated System for High Volume Work Loads

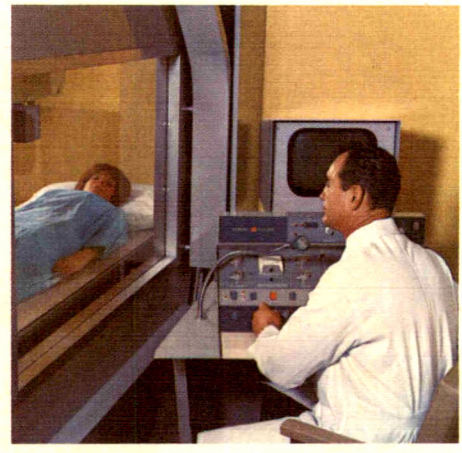
# TELETROL II

DIAGNOSTIC X-RAY SYSTEM

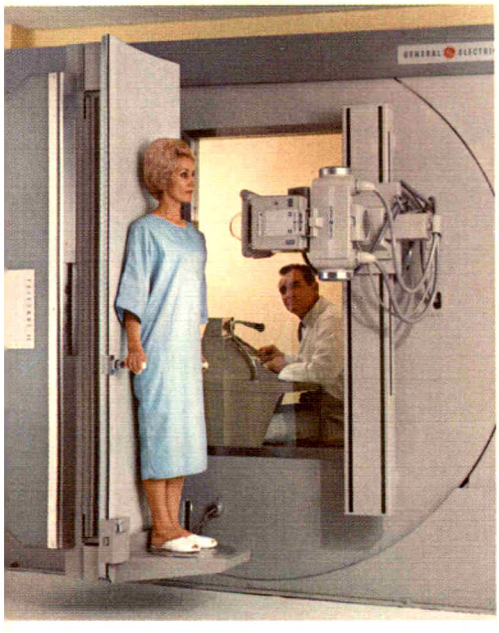
**Speeds Up Procedures as much as 50%**—New diagnostic technics and rapidly increasing patient loads place heavy demands on radiology departments. Unique integration of table, generator and image presentation components into one compact system allows for easy accommodation into most existing rooms without major alteration. One radiologist can perform a variety of procedures—brightly intensified fluoroscopy, 16-mm cine-filming, 70-mm spot-filming and Bucky radiography—*without ever moving from the convenient, radiation shielded control console.* Patients move through rapidly with consistently efficient results. The generator provides 150 kVp for all radiographic technics with independent fluoroscopic kVp.

The Teletrol II is provided with high performance operational features. New transverse table top movement is independent of Bucky. The radiographic tube is automatically aligned for precision recording because it's coupled to the intensifier. A new 4-on-1 serial cassette tray speeds up filming; also accepts conventional standard cassette tray. Rotating foot rest, shoulder rest, grasp handles and safety harness are furnished as standard features.

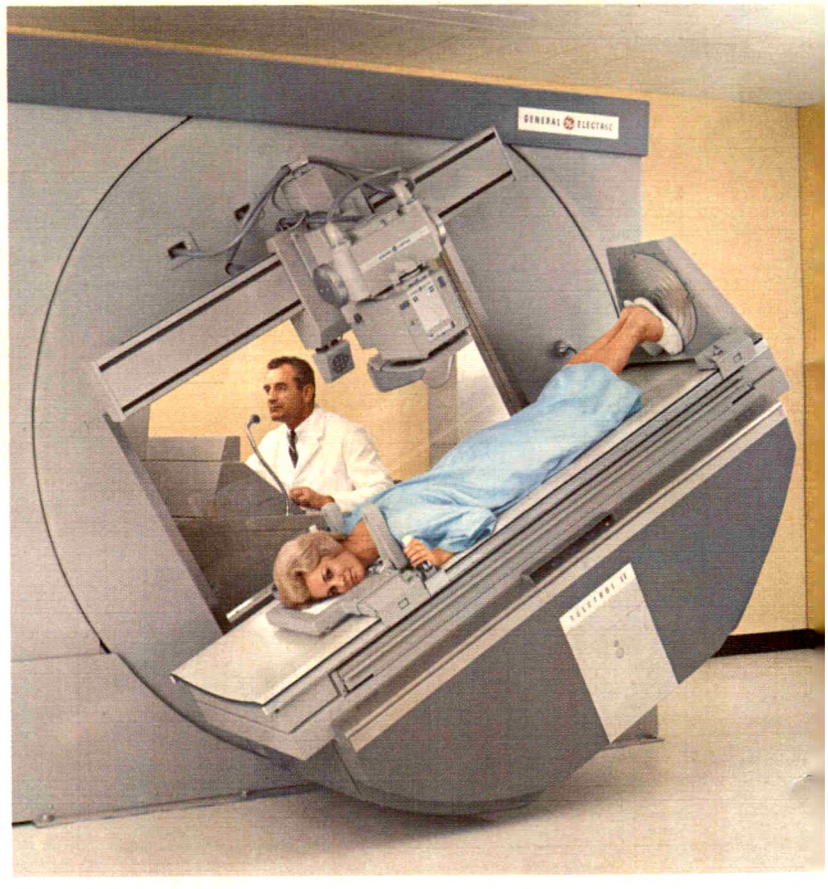
**New Relicon Television System**—Teletrol II is equipped with a new high resolution separate mesh vidicon TV camera, available in 525 or 875 line scan rate, featuring 15 megacycle band width and contrast-improving circuitry. Relicon camera and read-out system are designed specifically for image intensification. The monitor has a full 14-inch screen.



**Instant Command Control Console**—The ultimate in convenience and equipment response is provided by the Teletrol II control console. Operator maintains close patient contact visually through 30¾-inch x 54¾-inch radiation-shielded window and audibly through intercom system.

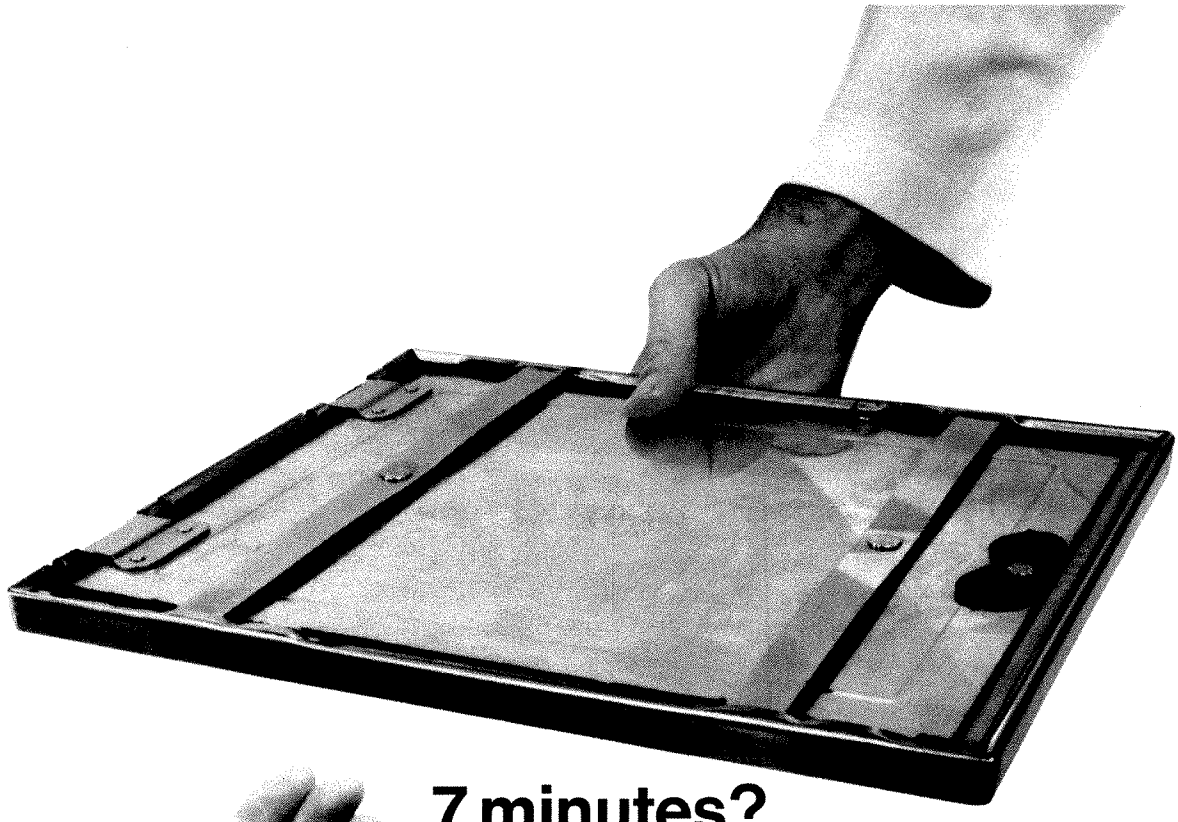


**Smooth 90-90° Angulation**—Teletrol II provides smooth, quiet powered angulation and table-top movement. Table angulates at choice of 2.2 or 4.5 degrees per second with automatic stops at horizontal, Trendelenburg and vertical. Moving top measures 84-inch x 29-inch, easily supports 300-pound patient. Top extends 30 inches off either end and 10 inches transversely for complete patient coverage. New top design is contoured to provide minimum patient-to-film distance for improved detail and sharpness. All positioning of table and patient easily controlled from operator's console.



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Liquamat-matched solutions deliver top quality images... consistently...with no contrast drop-off and no sludge build-up.
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When seeing NOTHING tells you a lot

# "Silent" gallbladders speak more clearly with Telepaque

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Telepaque actually provides a *dynamic picture of gallbladder function*—not just merely a *static* or *passive* filling effect. This is why Telepaque, with its *unexcelled record of diagnostic accuracy*, 98.3% to 100% in large-scale studies, has long been the contrast agent of choice in oral cholecystography and cholangiography.

**Contraindications:** Contraindicated in advanced hepatorenal disease or severe impairment of renal function, severe gastrointestinal disorders that prevent absorption, and in patients sensitive to iodine compounds.

**Precautions:** Severe, advanced liver disease may interfere with metabolism of Telepaque, thus increasing the excretory load on the kidneys. Although renal difficulty has rarely been attributed to Telepaque, renal function should be assessed before cholecystography in severe, advanced liver disease, and renal output and hepatic function should be observed for a few days after the procedure. Patients with preexisting renal disease should not receive high doses of cholecystographic media. Possible renal irritation in susceptible individuals could result in reflex vascular spasm with partial or complete renal shutdown. Caution is advised in patients with coronary disorders, especially those with recent symptoms of coronary artery disease. Blood pressure should be observed after administration of cholecystographic media to these patients. Elevation of protein-bound iodine for several months and false positive urine albumin tests (for three days) may occur after ingestion of iodine-containing cholecystographic media.

**Adverse Reactions:** Most reactions are mild and transitory; serious side effects are very rare. Gastrointestinal effects (diarrhea, cramps, nausea, vomiting) are the most common. Usually the diarrhea consists merely of a few loose stools, although in isolated cases it may be severe. A mild stinging sensation during urination may occur, and rarely, skin rash, urticaria, pruritus, and flushing. One case of thrombocytopenia has been reported in a patient with a history of conjunctival hemorrhages. Subjective complaints have been: dryness of throat, burning on swallowing, heartburn, sore throat, dizziness, and headache.

**Usual Dosage:** 3 Gm. (6 tablets) at night after a light supper.

**Supplied:** Tablets of 500 mg., envelopes of 6 tablets, boxes of 5 and 25 envelopes; also bottles of 500.

*However other unusual causes of nonvisualization are occasionally encountered.

**Telepaque®** for precise oral cholecystography and cholangiography

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**CASE OF THE DAY** *by* **Benjamin Felson** *and* **Jerome F. Wiot,** *both of Univ. of Cincinnati, Ohio.* Discussion of each case presented includes diagnosis, roentgen findings, general discussion of subject illustrated—of roentgen principles involved in diagnosis, and key references. Principles are emphasized—applicable not only to the cases illustrated but also to more common conditions. **Discussions are directed toward areas not usually covered in roentgen texts.** *Sept. '67, 96 pp. (6¾ × 9¾), 91 il., (Amer. Lec. Roentgen Diagnosis edited by Lewis E. Etter), $8.50*

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**RADIOLOGICAL PHYSICS (2nd Ed.)** *by* **M. E. J. Young,** *British Columbia Cancer Institute, Vancouver.* This Second Edition retains the primary objectives of the first edition, and has been enlarged and updated to include those changes which have taken place in radiological practice in the last ten years. *Partial list of Contents:* The Production of X-rays up to about 400 keV in Energy; The Production of High Energy X-rays; The Interaction of X- and γ-radiation with Matter; The Measurement of Ionizing Radiations; etc. *May '67, 616 pp., 244 il., $16.50*

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- **TREATMENT OF HYPERTHYROIDISM WITH RADIOACTIVE IODINE: A Twenty-year Review** *by* **John E. Kearns,** *Northwestern Univ., Chicago.* May '67, 88 pp., 17 il., $6.50

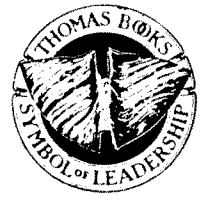
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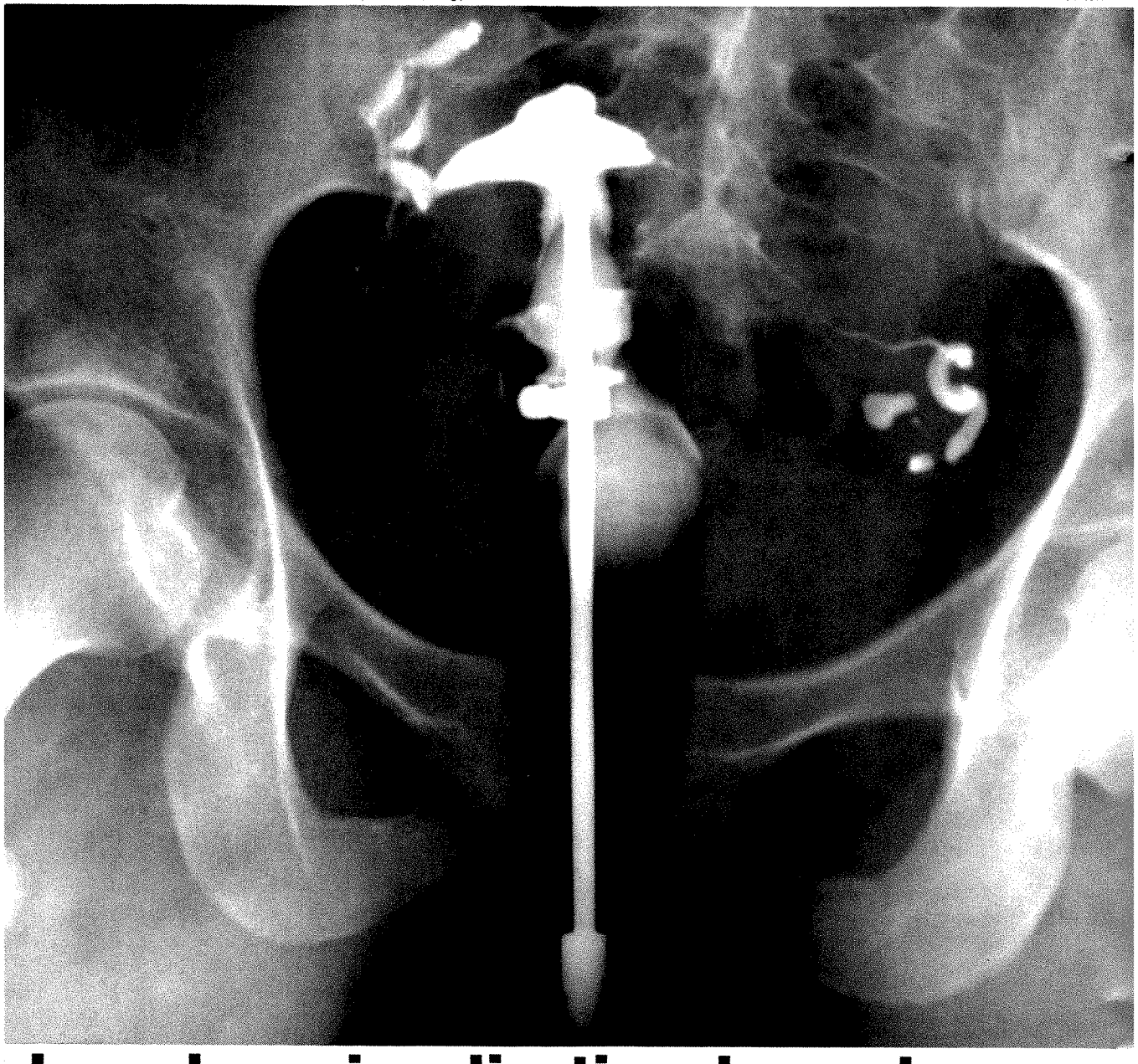
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ord of this hysterosalpingogram on file in Medical Department, Geigy Pharmaceuticals.

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# hen clear visualization depends on clear bowel, depend on Dulcolax®

merous reports in the radiology literature scribe the superiority of Dulcolax over castor and enema in prepping patients for abdomi- l X-ray examinations.

klesaria and Antia[1] reported that with Dulcolax of 40 patients showed no gas shadows, showed moderate shadows, and 5 showed arked shadows. With a similar group prepped th castor oil and enema, 11 showed no gas shadows, 14 showed moderate shadows, and 15 showed marked shadows.

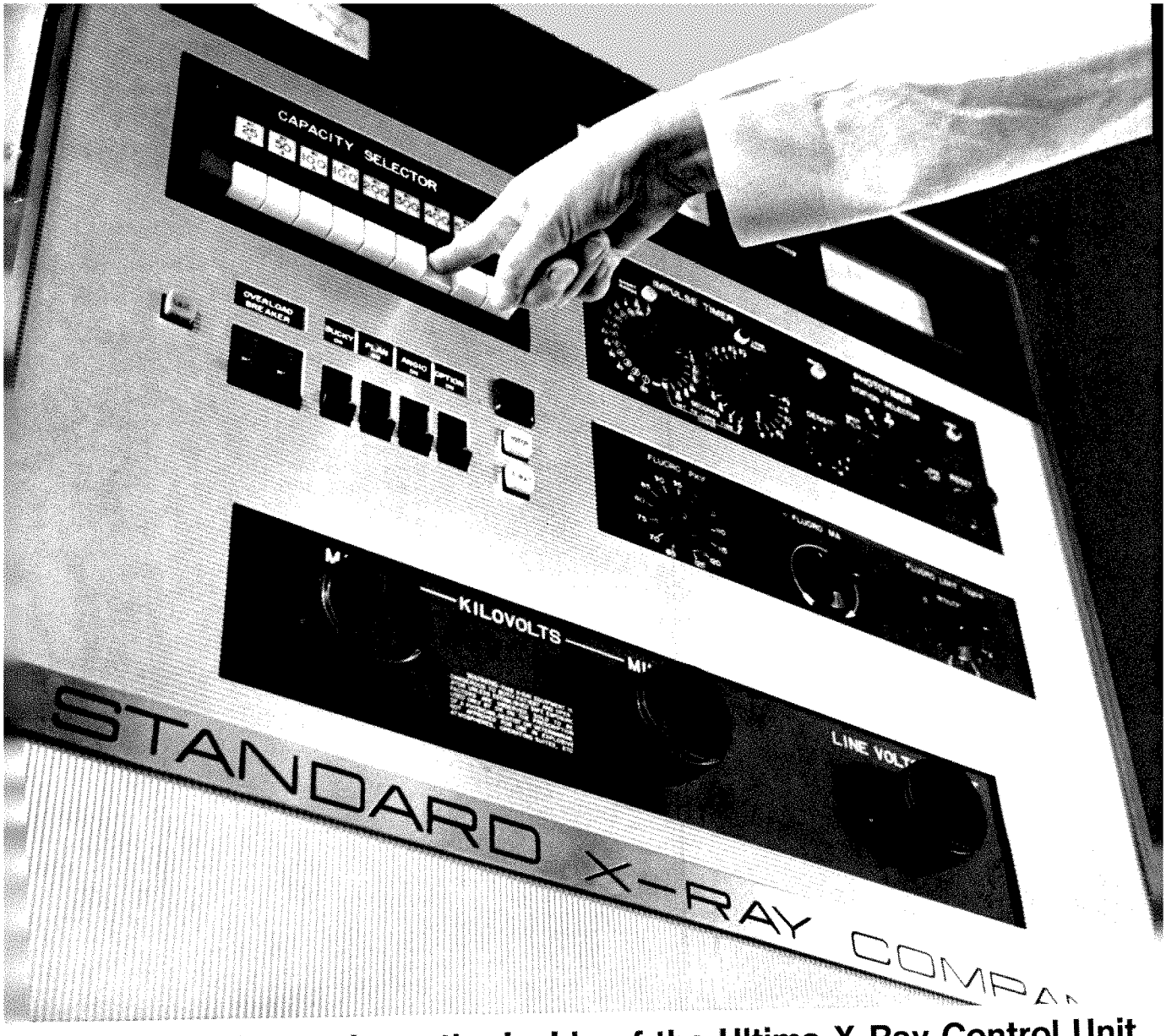
Poppel and Bangappa[2] likewise reported "a more reliable cleansing effect" with Dulcolax. These investigators felt that 3 tablets at night followed by a suppository the morning of the examination "...should result in satisfactory visualization even in procedures presenting as much difficulty in preparation as pyelography."

1. Curr. Med. Pract. 7:145, 1963. 2. Am. J. Roentgenol. 81:696, 1959.

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The story is modular unit construction. That's what makes the Ultima X-Ray Control Unit unique. It means we can build it to your exact requirements without the cost or waiting normally associated with custom construction. You can actually choose from 1056 different possible combinations of controls, timer modes, and special features. You can also choose from either 400 or 600 MA models with solid state or thermionic valve high-voltage rectified transformers.

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You get more than just choice with the Ultima X-Ray Control Unit. You get flexibility too. At any time, simply by changing modules, you can modify or up-grade the Ultima to meet new requirements. For example, if you wish to convert from the control of conventional 60-cycle x-ray tubes to control of high-speed 180-cycle x-ray tubes, the Ultima can be modified quickly, on the-spot, and with minimum cost.

Servicing is simplified too. Modules and componen can be serviced or replaced individually as require without dismantling the entire unit. Careful design ha kept all parts readily accessible and easy to reac

Most important, you benefit from Standard's 53 yea of experience in producing durable, dependable, ar quality x-ray equipment. Every bit of this experienc has gone into the design and production of the Ultim X-Ray Control Unit.

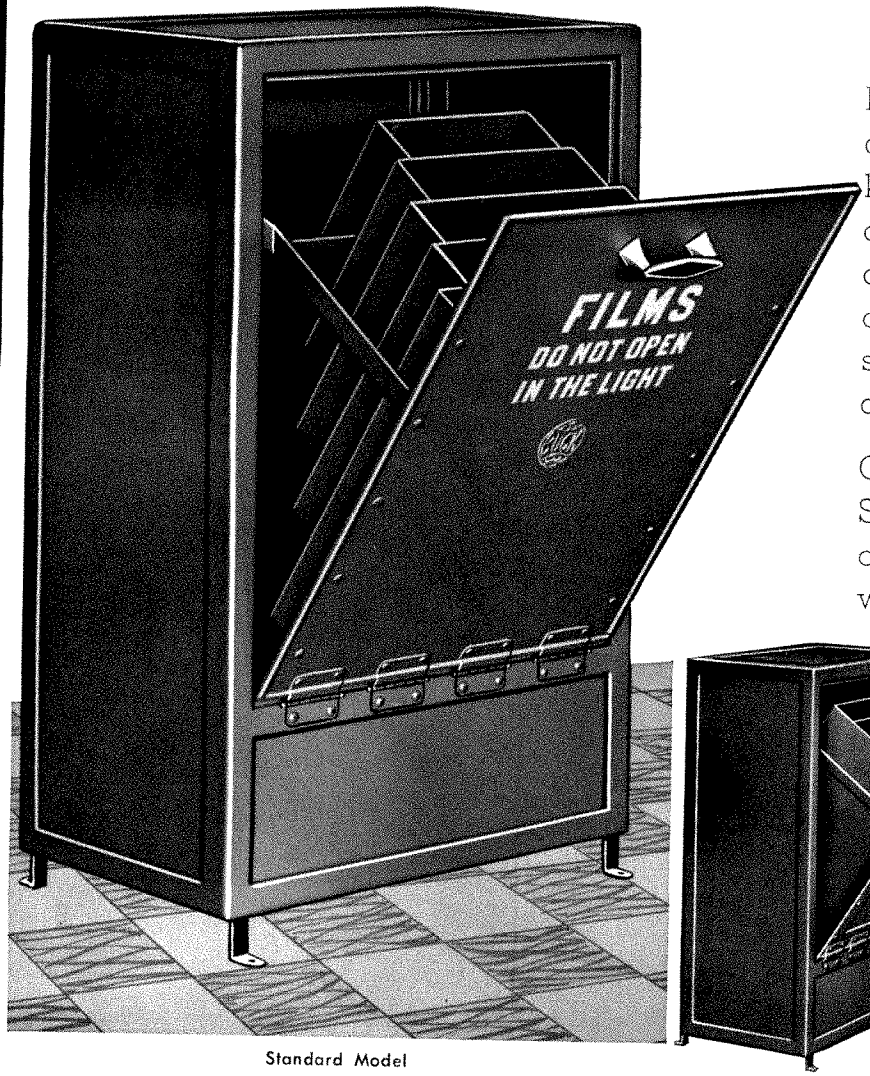
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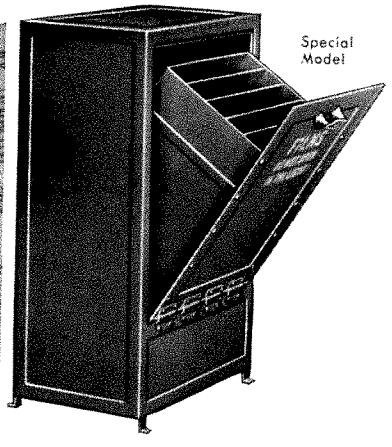
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Can be supplied with Limit Switch to automatically turn off darkroom white light when bin is opened.



Special Model

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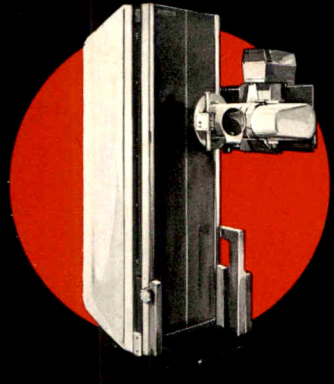
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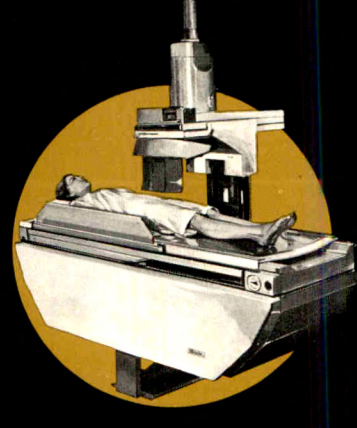
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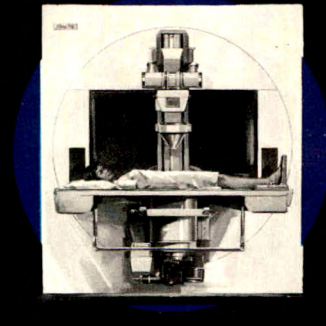
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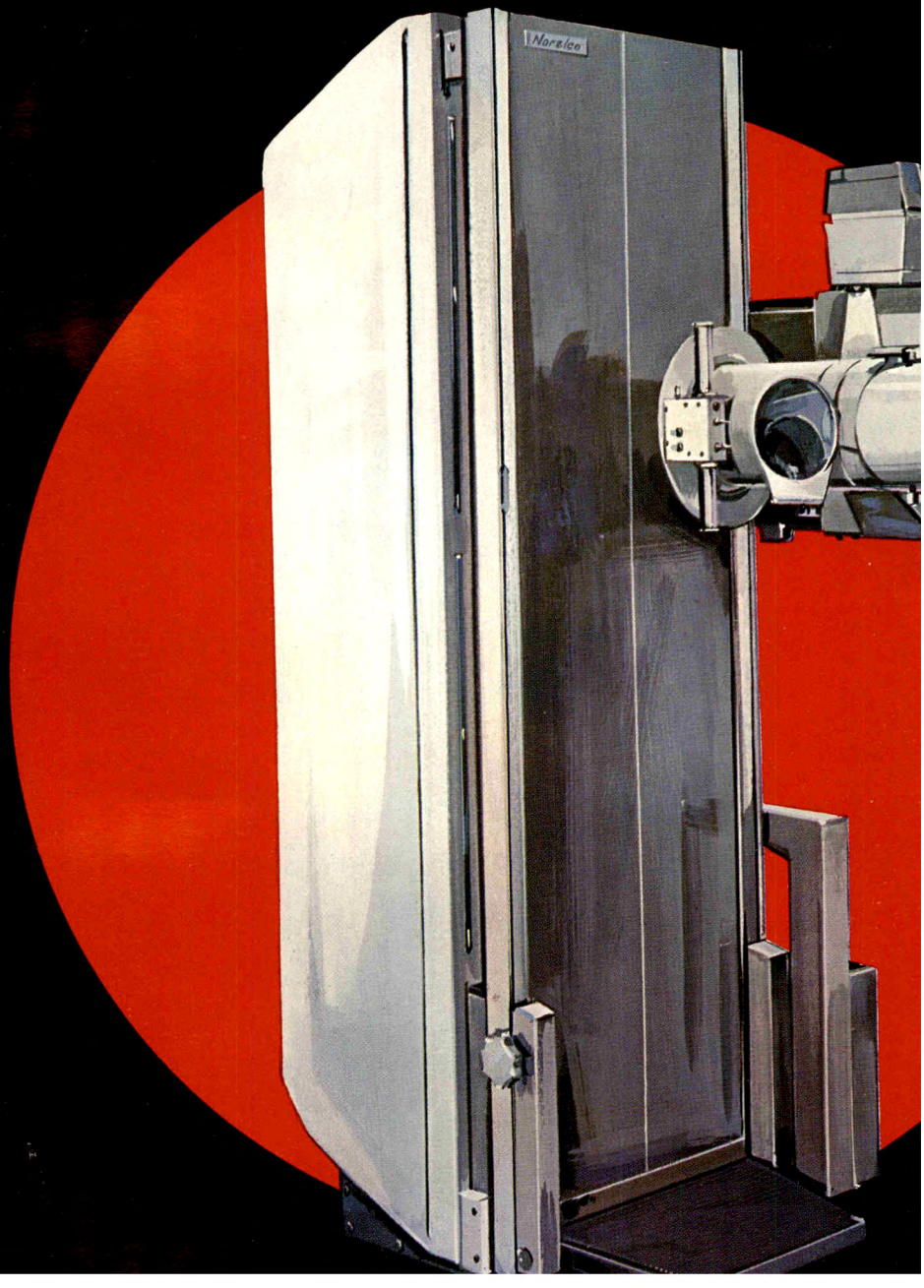




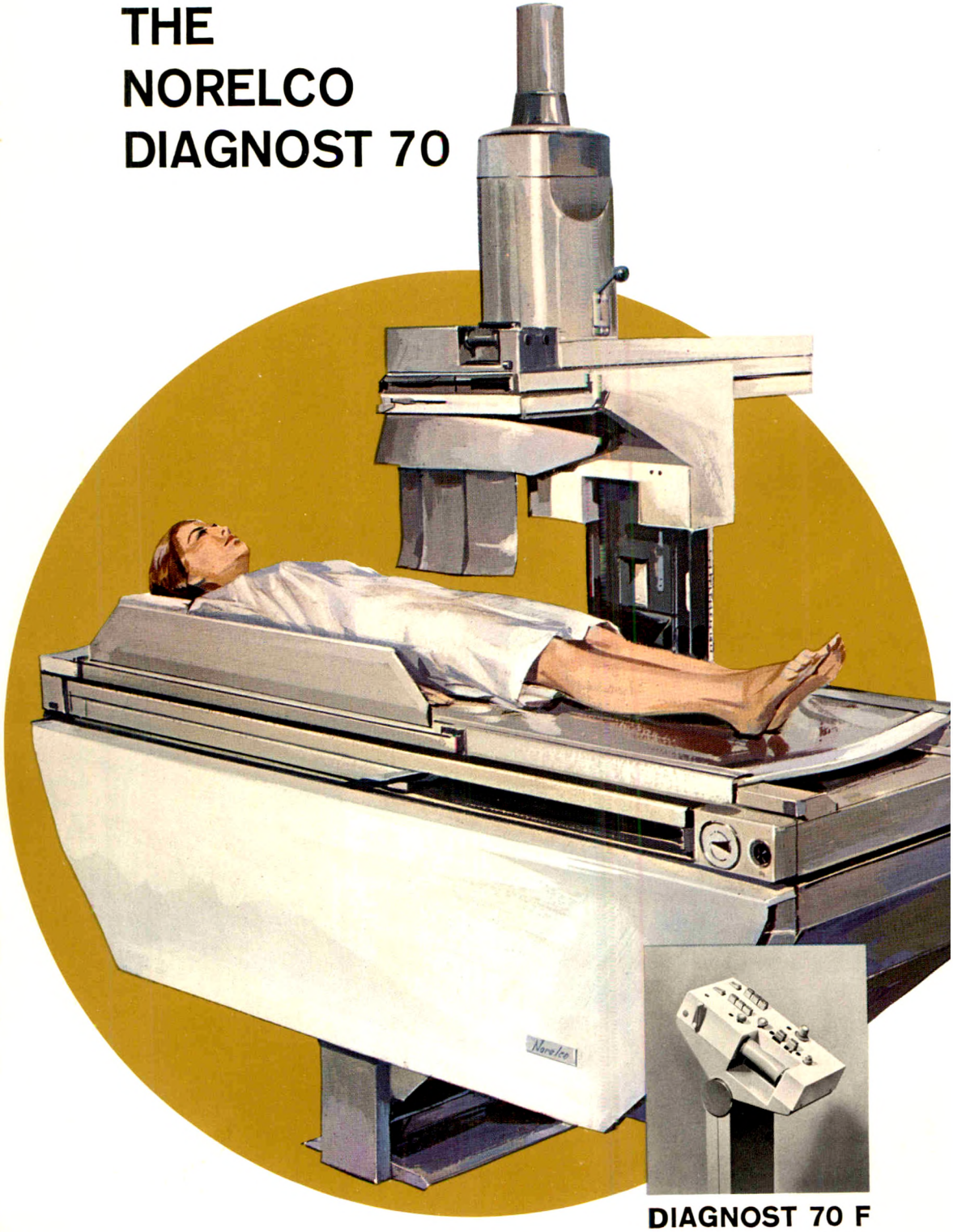
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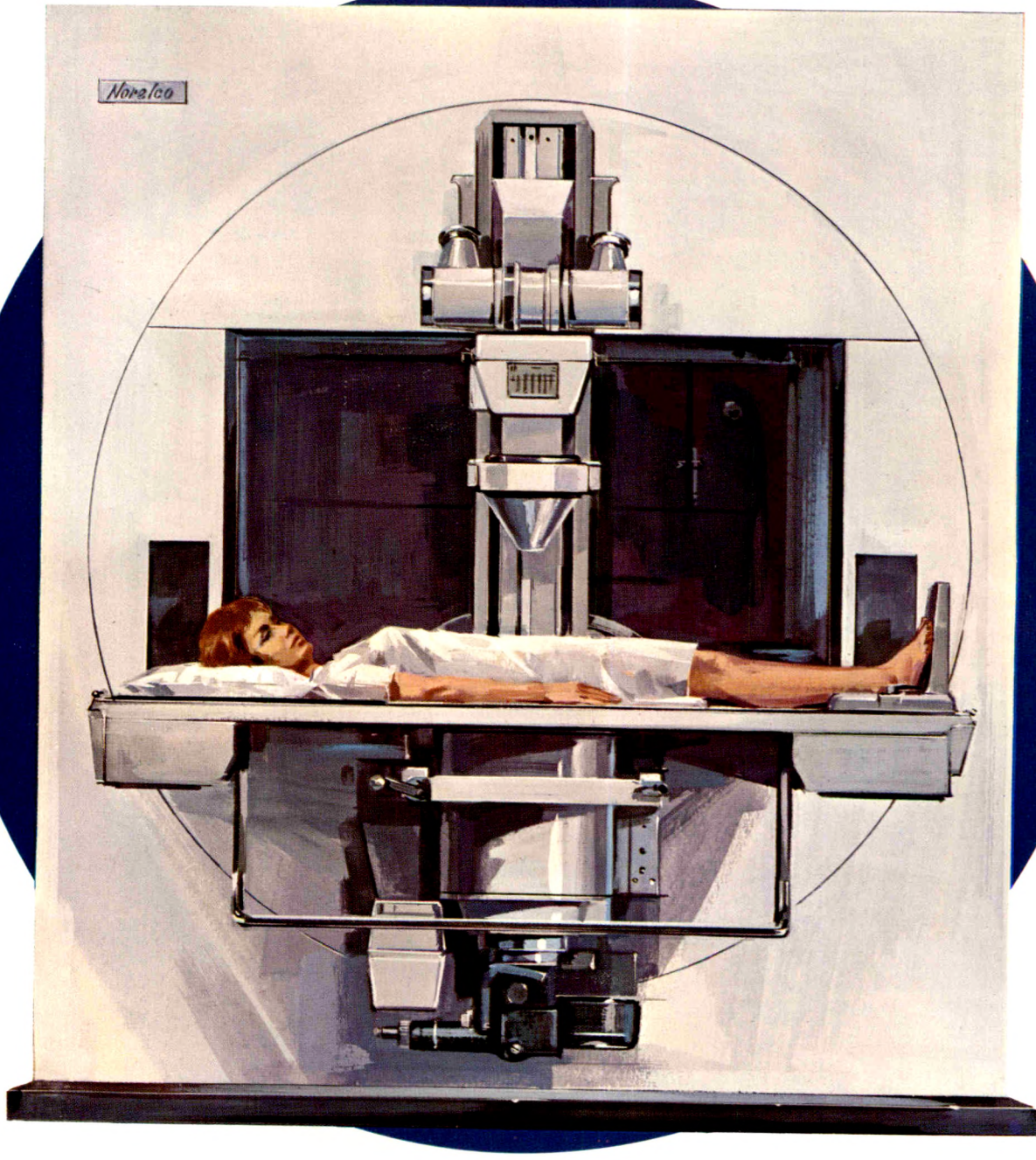
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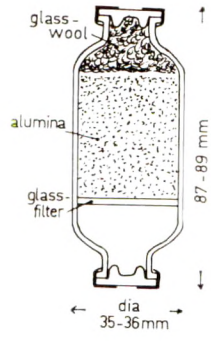
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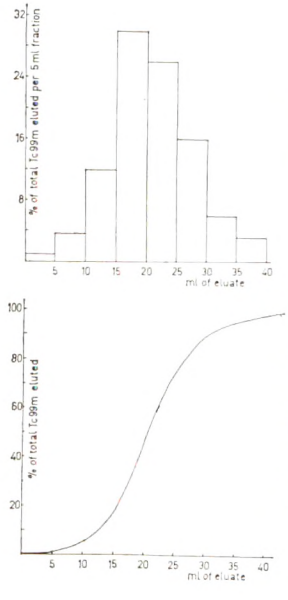
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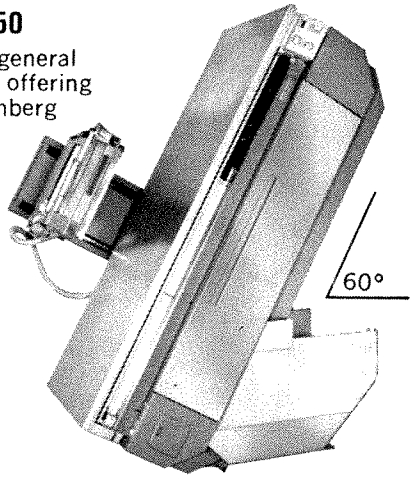
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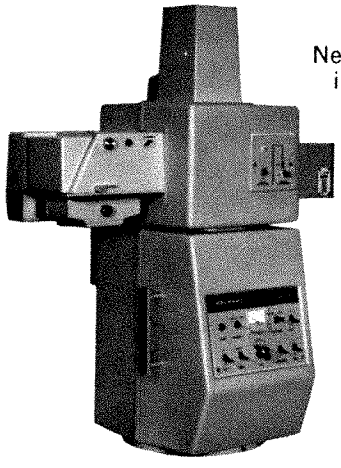
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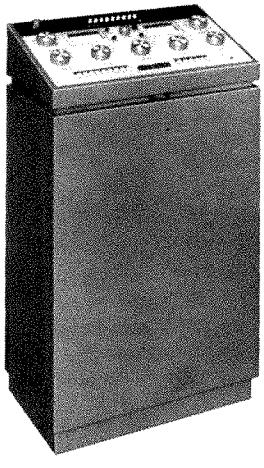
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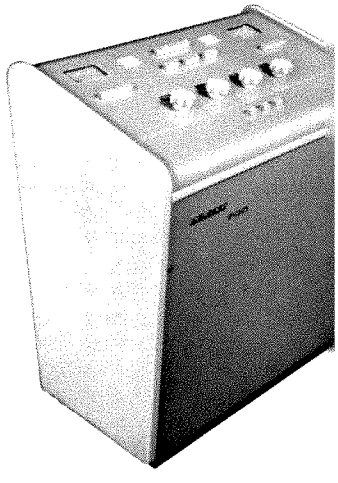

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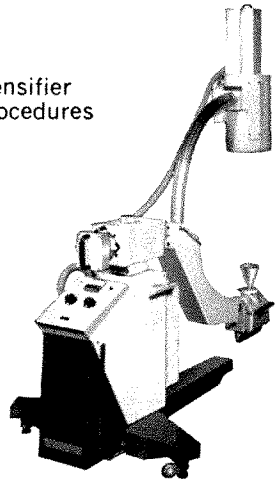


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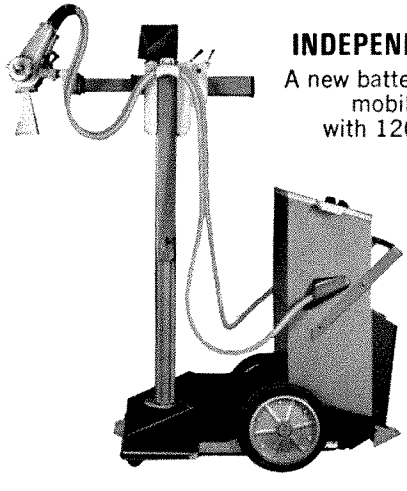


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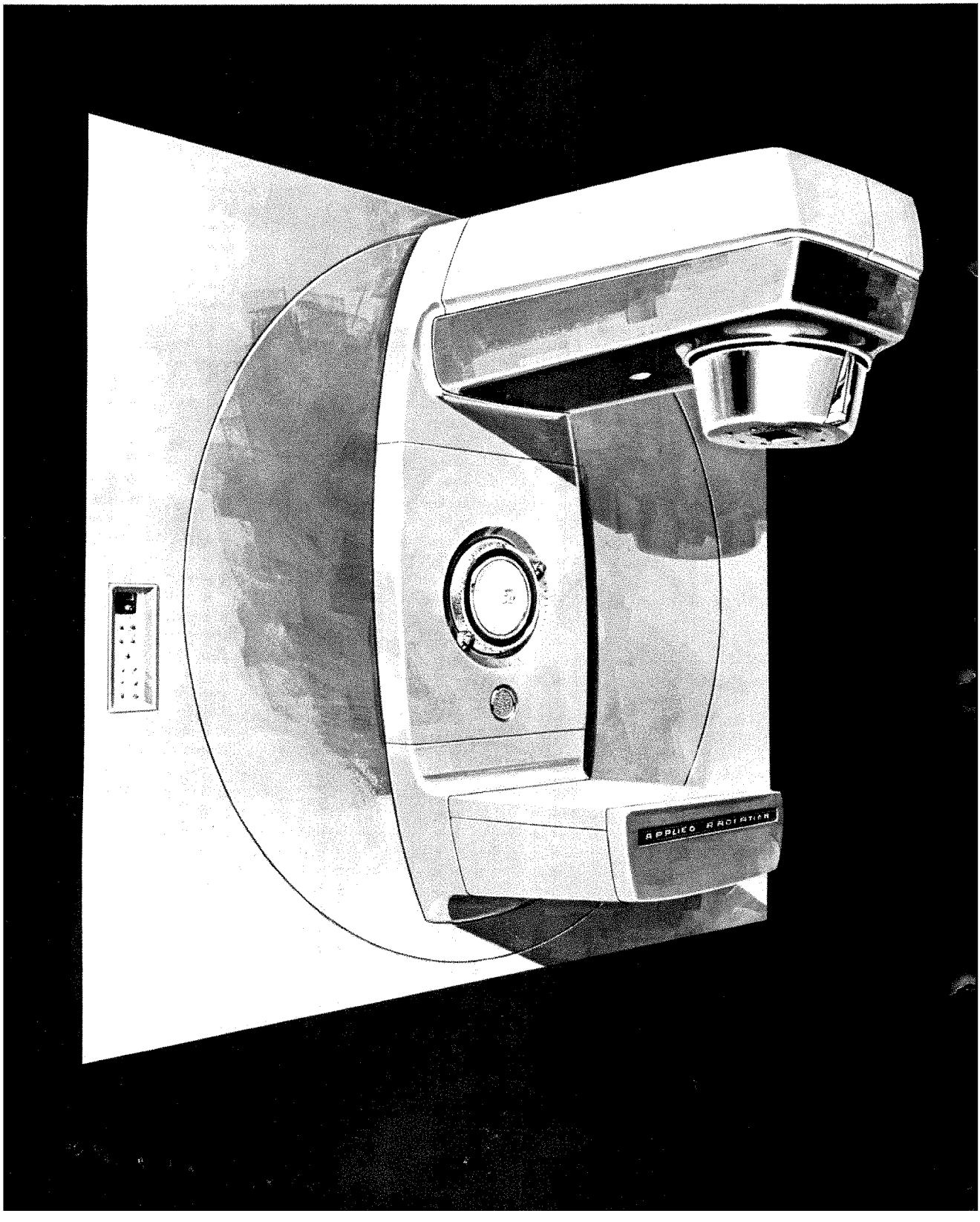


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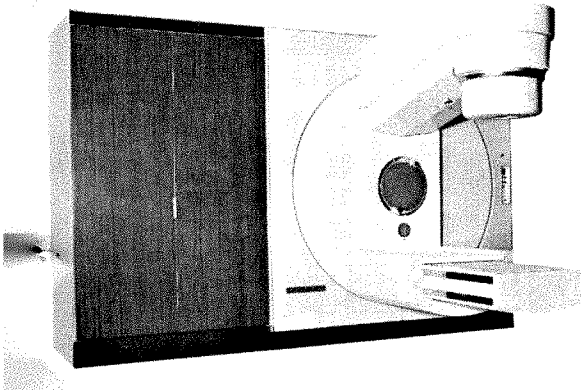
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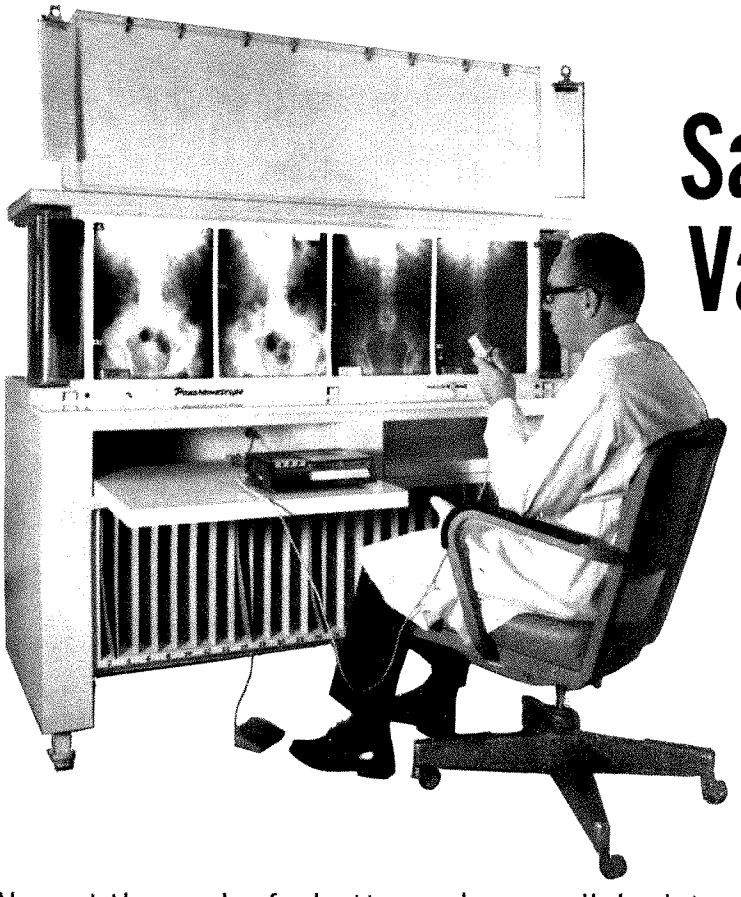


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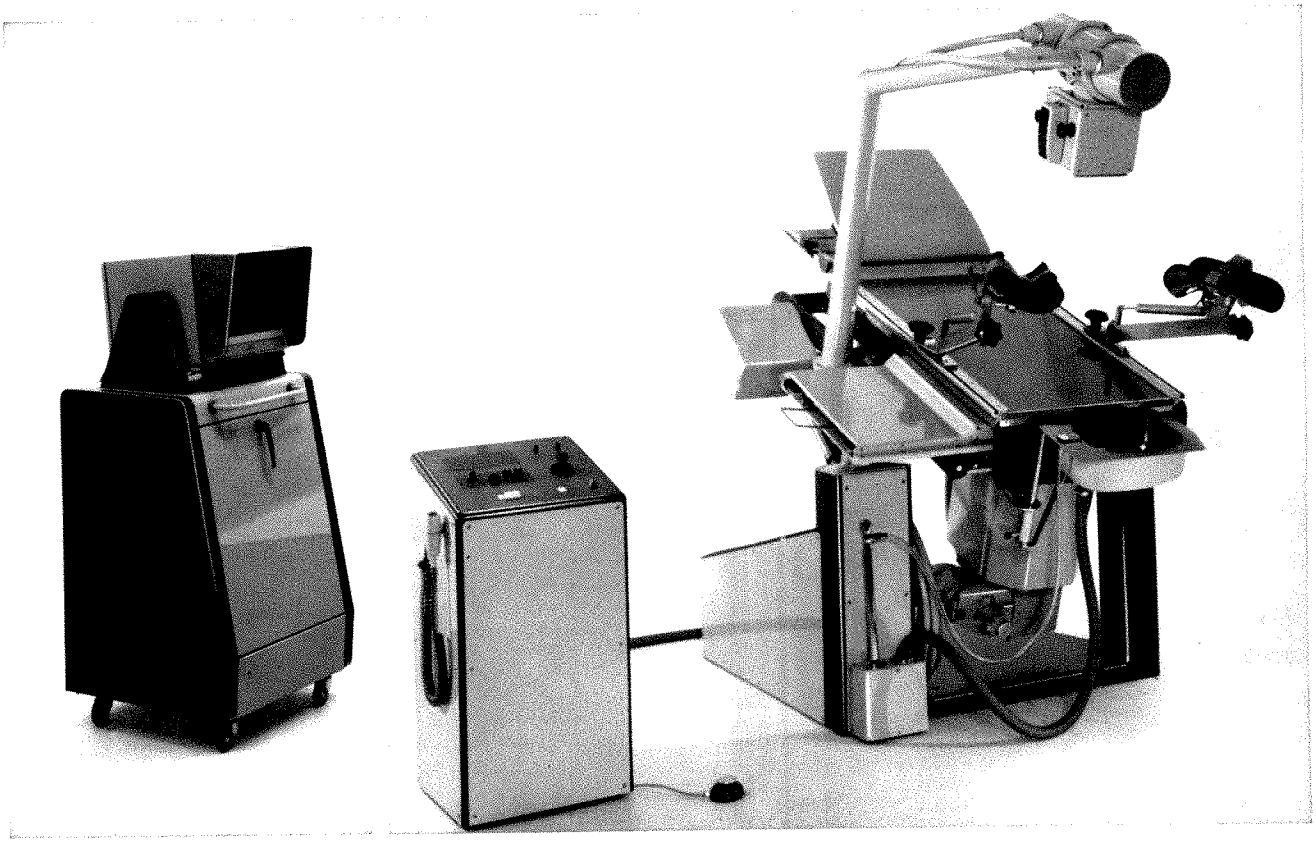
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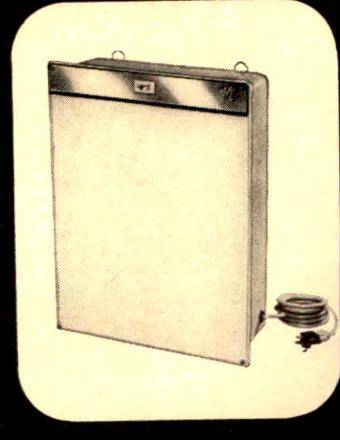
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VOLUME 101 DECEMBER, 1967 NUMBER 4

INDEX NUMBER

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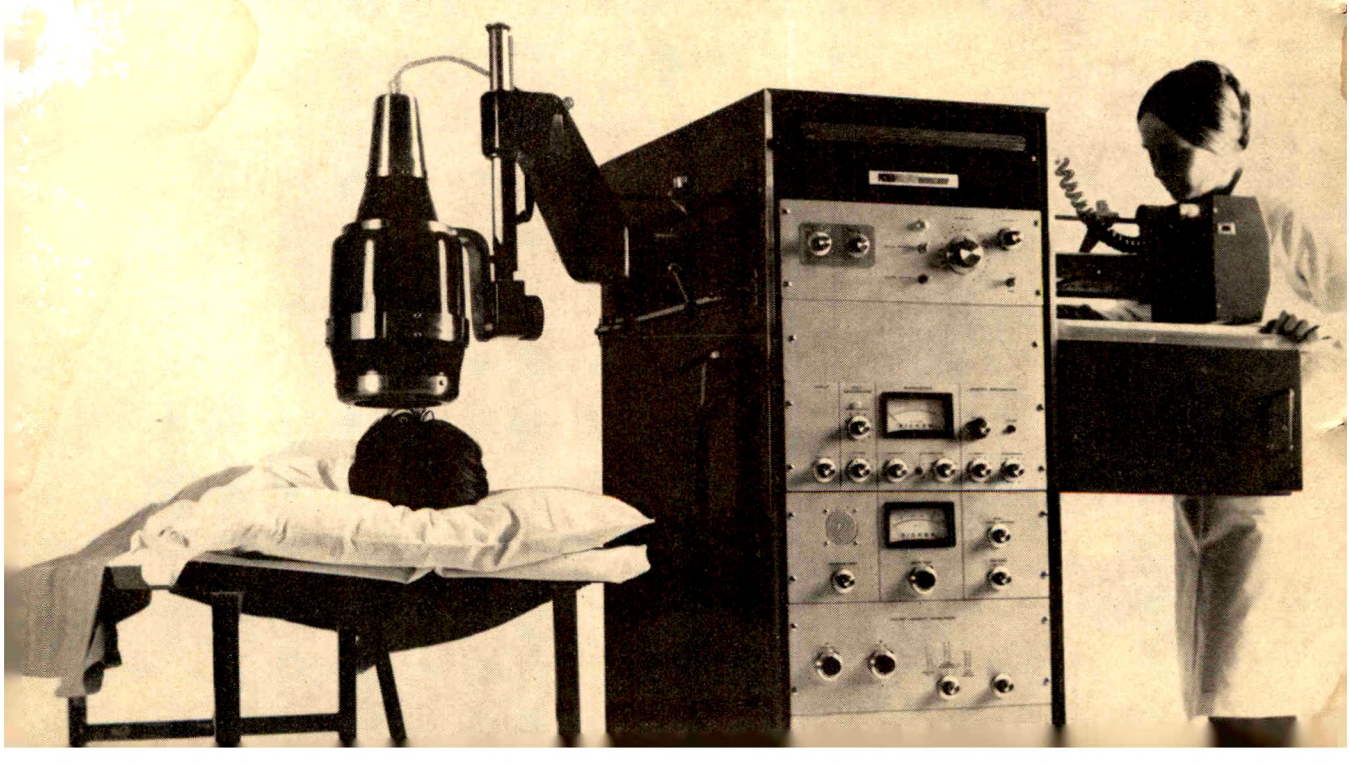
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# New help in diagnosing pulmonary problems

## New radioisotope scanning procedure can help detect the vascular changes of pulmonary disease before they show on chest films

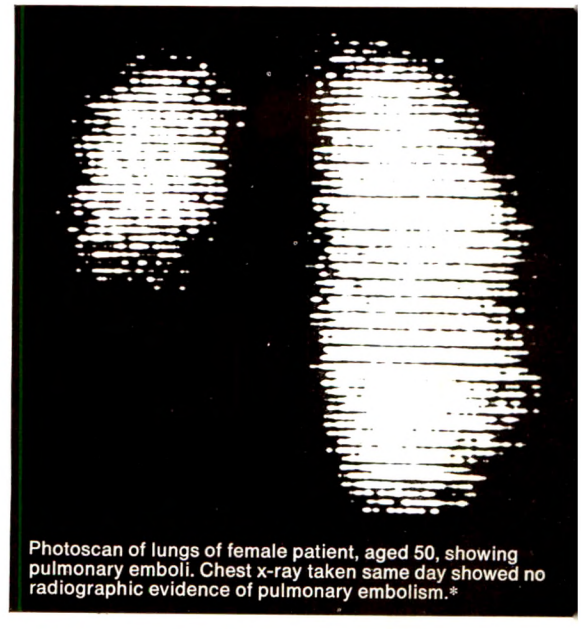
Scintiscanning of the lungs now offers a new approach to the diagnosis of pulmonary disease.[1] With use of macroaggregated radio-iodinated $I^{131}$ albumin, lung scanning has been found to be simple, rapid and relatively safe,[2,3] and is invaluable as an adjunct to other diagnostic procedures whenever information about pulmonary vasculature is desired.

Perhaps the most useful application of the lung scan has been for the early detection of pulmonary embolism where "... it appears that the lung scan can point to the site of embolic lesions before signs of lung infarction are recognizable on plain chest films."[3] This is important, for with the development of new means of treating pulmonary embolism, the need for improved diagnostic ability has increased. For example, the availability of anticoagulant drugs to prevent further thrombosis and of proteolytic agents to dissolve thrombi already formed, the use of surgical therapy (such as ligation or plication of the inferior vena cava and even pulmonary embolectomy) — all require more accurate diagnostic procedures.[4,5]

Of course, pulmonary arteriography can give an immediate positive demonstration of an obstruction in the pulmonary circulation as soon as it occurs, but this procedure is time consuming and technically difficult to perform. It necessitates injection of large quantities of high density contrast medium directly into the pulmonary artery, and it also requires cardiac catheterization (with some risk of dislodgement of venous thrombi). Moreover, experience has shown that patients with pulmonary hypertension may tolerate injections of contrast material poorly. Other examinations, such as x-ray study of the chest and electrocardiography, are rarely definitive.[4]

In contrast, lung scanning with Albumotope-LS is a simple and direct adjunctive measure; reliable and virtually without risk of morbidity to the patient. And unlike pulmonary arteriography it does not require cardiac catheterization and involves only minimal inconvenience to the patient. All that is required is the i.v. administration of a relatively small amount of the isotope. And the test may be supplemented with other procedures when necessary.

Although the lung scan has been used most frequently for the detection of pulmonary emboli, it can provide useful information in the diagnosis and evaluation of other pulmonary problems. For example, a recent report[6] in the September, 1966, issue of *Circulation* discusses the potential applicability of the technique in the detection and assessment of mitral valve disease. According to the authors, the technique has been found useful in screening patients with clinical findings of mitral valve disease who were not considered symptomatic enough to warrant cardiac catheterization... in the preoperative study of patients so ill that left heart catheterization was unusually hazardous... and in determining whether the pulmonary venous pressure is elevated in patients with known severe pulmonary arterial hypertension. In these latter patients it is often difficult to measure pulmonary arterial wedge pressure reliably and the more extensive manipulations necessary for left heart catheterization may be poorly tolerated. Thus, assessment of the distribution of pulmonary arterial blood flow by lung scanning affords a means for determining the existence of pulmonary venous hypertension, which suggests the presence of potentially correctable lesions, such as mitral stenosis or cor triatriatum.[6]



Photoscan of lungs of female patient, aged 50, showing pulmonary emboli. Chest x-ray taken same day showed no radiographic evidence of pulmonary embolism.*

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**References:**

(1) Quinn, J. L., III; Whitley, J. E.; Hudspeth, A. S., and Prichard, R. W.: Radiology *82*:315 (Feb.) 1964. (2) Sabiston, D. C., Jr., and Wagner, H. N., Jr.: Ann. Surg. *160*:575 (Oct.) 1964. (3) Haynie, T. P.; Hendrick, C. K., and Schreib M. H.: J. Nucl. Med. *6*:613, 1965. (4) Wagner, H. N., Jr., *et al.*: New Eng. J. Med. *271*:377 (Aug. 20) 1964. (5) Quinn, J. L., III; Whitley, J. E.; Hudspeth, A. S., and Watts, F. C.: J. Nucl. Med. *5*:1 (Jan.) 1964. (6) Friedman, W. F., and Braunwald, E.: Circulation *34*:363 (Sept.) 1966.


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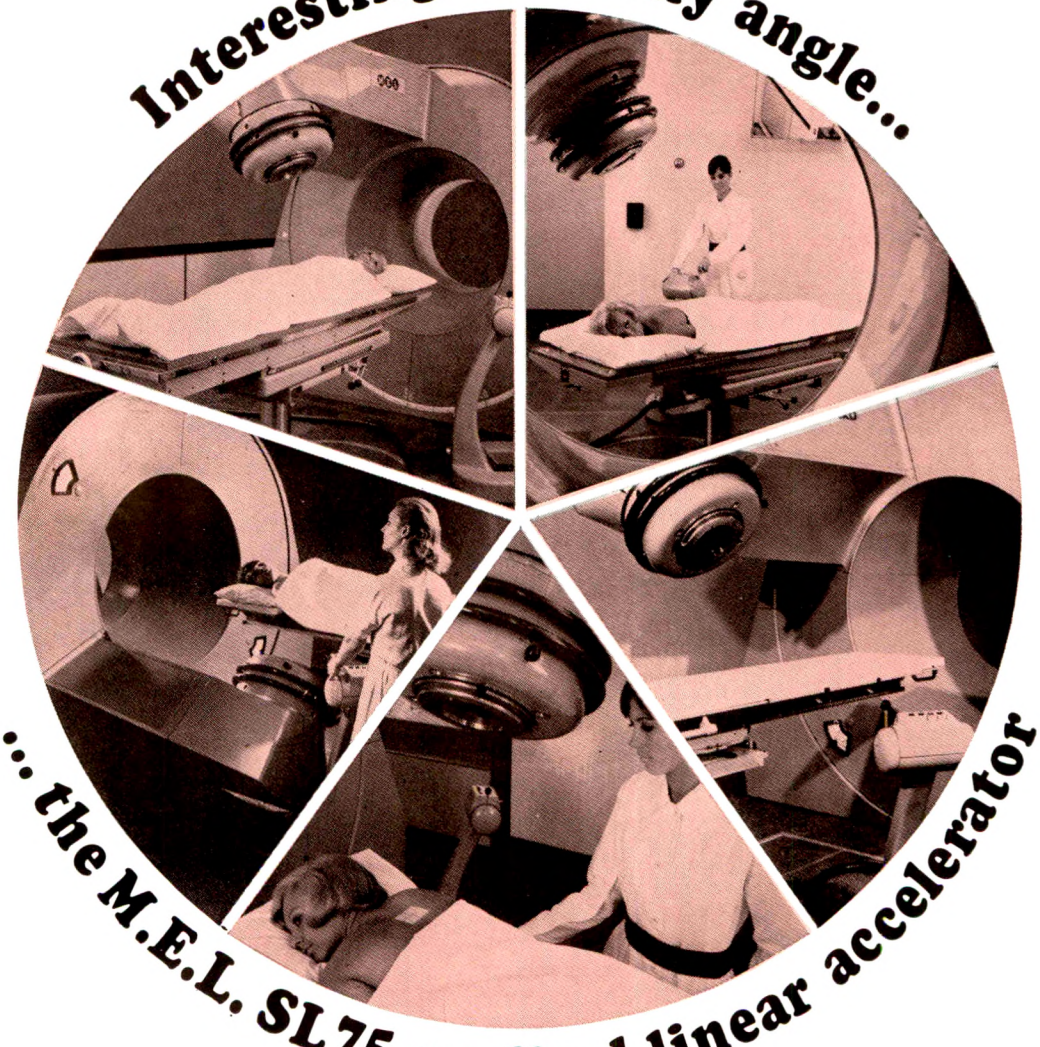
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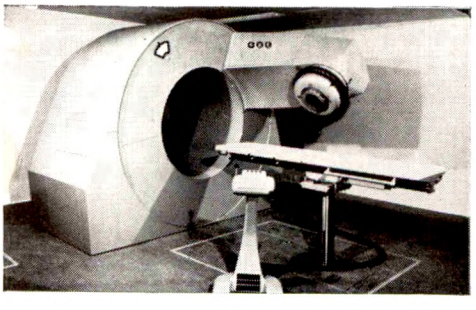
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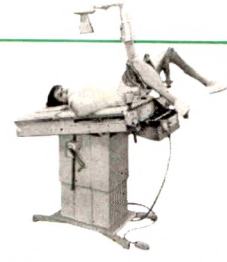
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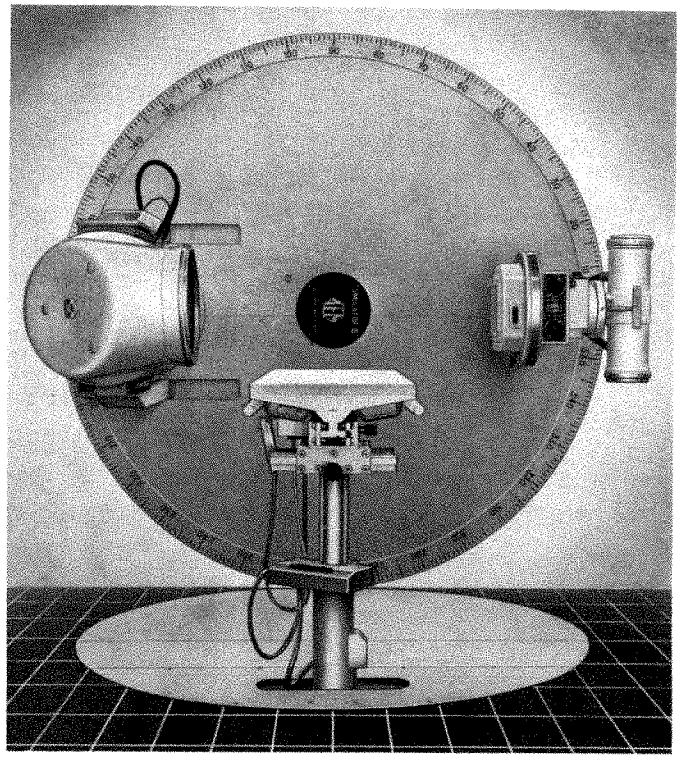
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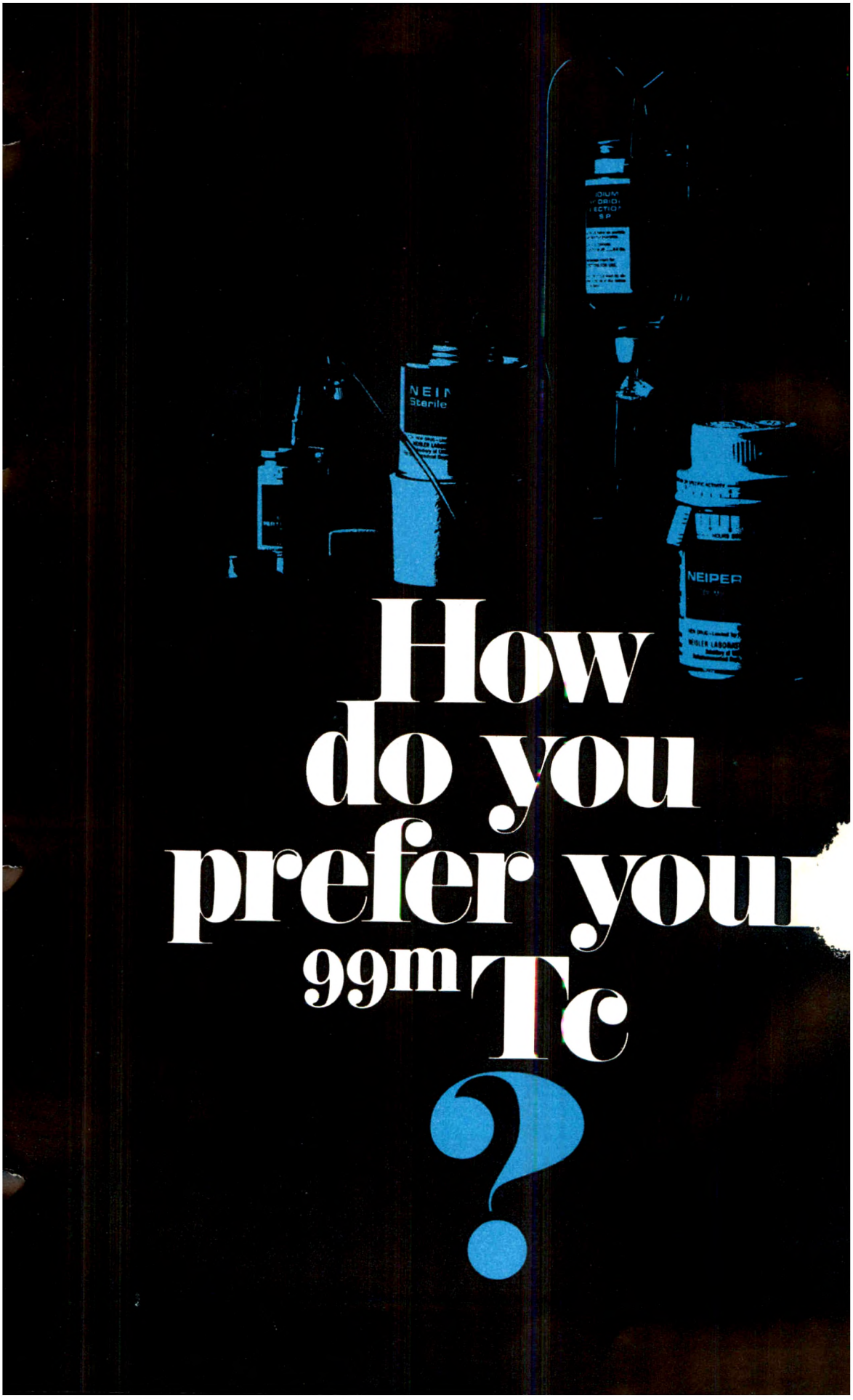
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# How do you prefer your $^{99m}Tc$ ?

# now from Neisler...

## STERILE, PYROGEN-FREE SODIUM PERTECHNETATE Tc 99m AS YOU NEED IT, ALL WEEK LONG

# NEIMOTEC® Sterile Generator

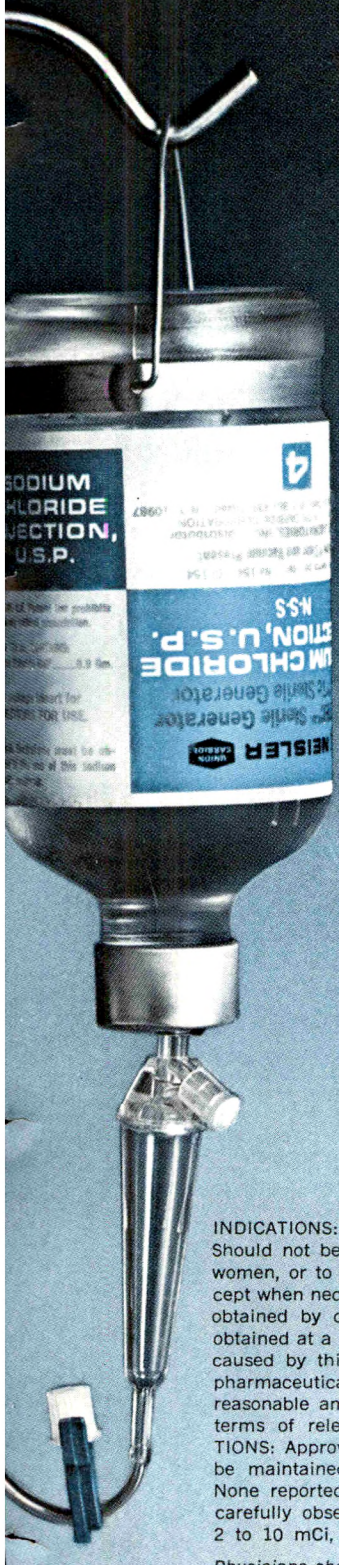
$^{99}Mo/^{99m}Tc$ Sterile Generator

**FAST... EASY... ECONOMICAL**

- simple vacuum elution system for maximum dependability
- entire system sterile...one-time entry to easily accessible septa
- high-yield...high chemical purity
- multiple daily elutions possible

SUPPLIED: 100, 200 or 300 mCi at noon, New York time, on Mondays following shipment; in nonreturnable lead container, with complete eluting accessories.






# PRECALIBRATED READY-TO-USE

## NEIPERTEC™ 99m

sodium pertechnetate Tc 99m

INDICATIONS: Brain scanning. CONTRAINDICATIONS: Should not be administered to pregnant or lactating women, or to patients under the age of 18 years, except when necessary diagnostic information cannot be obtained by other types of studies or can only be obtained at a risk greater than the radiation exposure caused by this drug. WARNINGS: As with all radiopharmaceuticals, dose should be limited to smallest reasonable amount consistent with greatest value in terms of relevant diagnostic information. PRECAUTIONS: Approved radiation safety precautions should be maintained at all times. ADVERSE REACTIONS: None reported to date; however, patients should be carefully observed. DOSAGE AND ADMINISTRATION: 2 to 10 mCi, administered by intravenous injection.

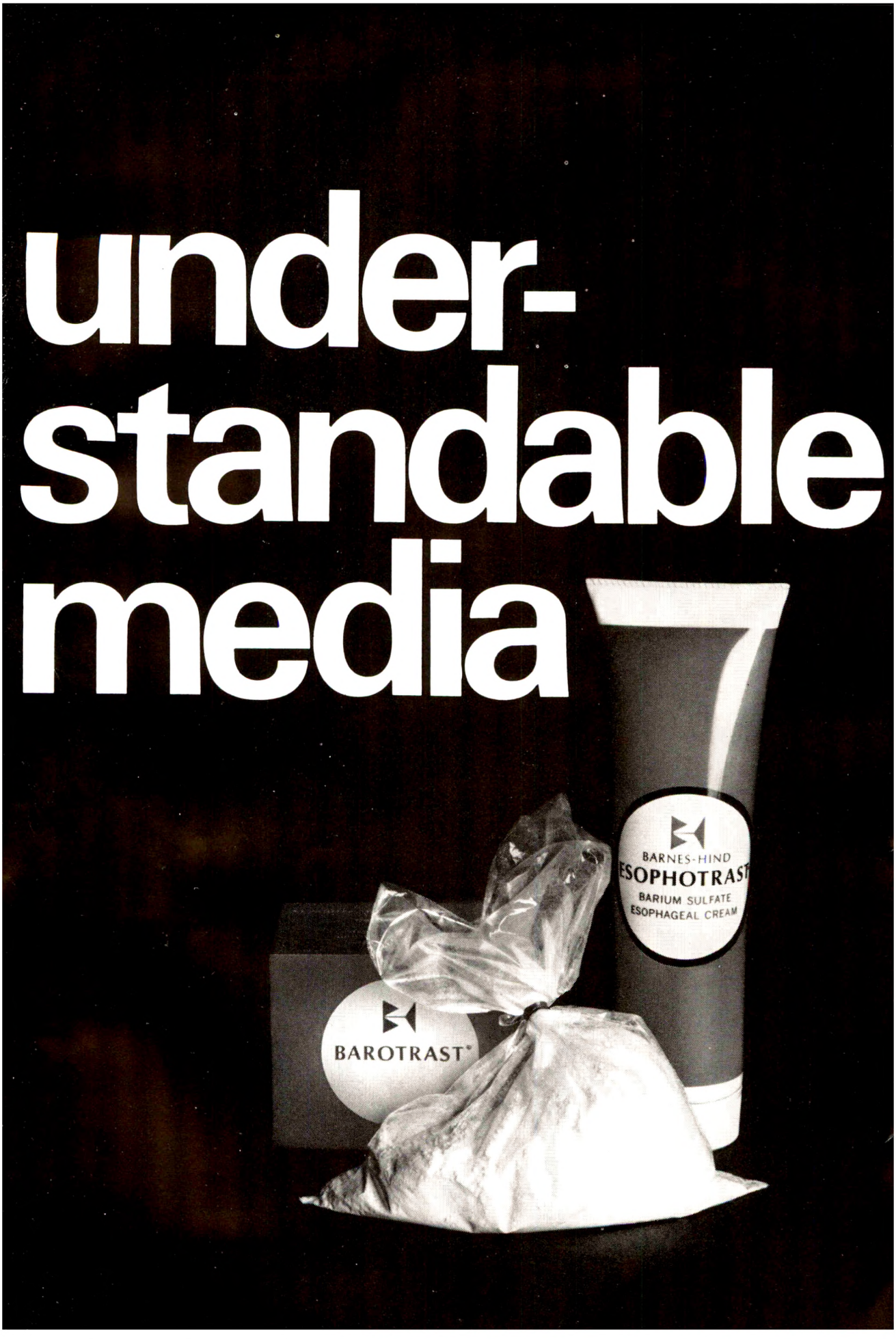
*Physicians should consult product package insert before administering.*

SUPPLIED: In lead-shielded vials in convenient COMPUTERCAP™ packaging; 10 or 15 mCi at the time of calibration.




**NEISLER LABORATORIES, INC.**
Subsidiary of
UNION CARBIDE CORPORATION
Radiopharmaceutical Dept.
P.O. Box 433, Tuxedo, New York 10987

UNION CARBIDE


under-
standable
media
BARNES-HIND
ESOPHOTRAST
BARIUM SULFATE
ESOPHAGEAL CREAM
BAROTRAST

If the barium preparations you've used up to now have all seemed pretty much alike, then it's time to compare the contrasts with ESOPHOTRAST® (the ready-to-use esophageal cream) and BAROTRAST® (a micronized form of barium sulfate).

One of the prominent problems with most contrast media is no small matter. Their large particles have a tendency to precipitate and to flocculate out of suspension with the slightest change in pH, mucin concentration, or enzyme activity. Not so with ESOPHOTRAST and BAROTRAST, both of which are formulated to ensure an easy suspension that remains in suspension even during routine storage.

Then too, a common feature of many other barium sulfates suggests a sticky problem. The varying size of the particles reduces their adhesive properties since not enough particles come into contact with the esophageal and/or intestinal lining to provide complete coverage. With micronized ESOPHOTRAST and BAROTRAST, a maximum number of particles are available to produce a tenacious elastic coating that lasts for the time it takes to "get the picture."

ESOPHOTRAST, because of its specially formulated viscosity, provides a consistent, prolonged, residual coat that permits an excellent outline of the esophagus as well as an evaluation of cardiac size. Since it is premixed, ESOPHOTRAST is ready for instant use, which means longer visualization time and shorter preparation time. BAROTRAST, because of its natural viscosity, flows in a steady consistent column at a speed that can cut the upper G.I. series and small bowel examination time almost in half.

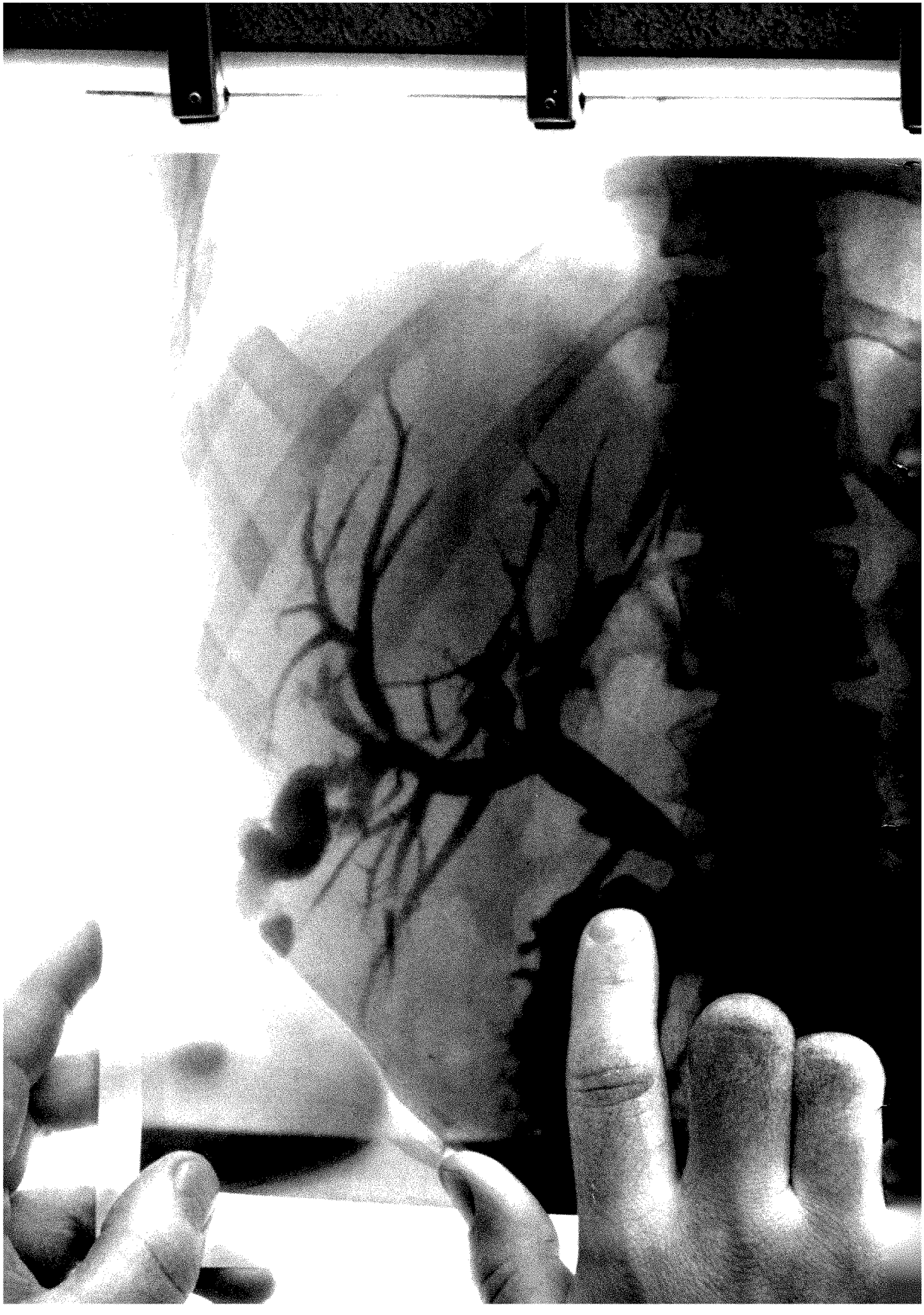
Both formulations are creamy, nongritty and pleasantly flavored to help your patient help you make the examination that much easier.

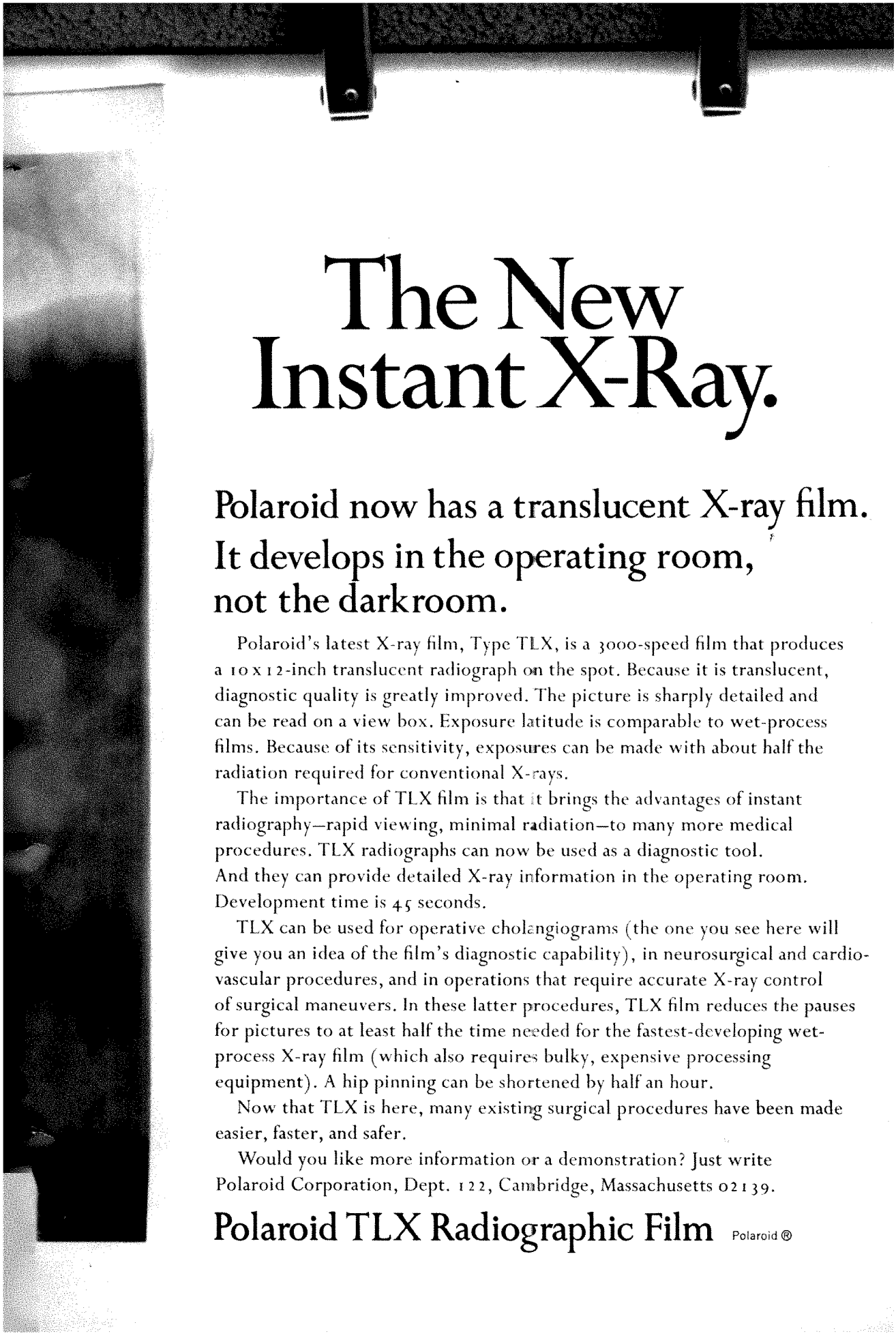
Considering the barium sulfates in the light of this inside information, it would seem that there is a marked contrast. And all it takes is a little understanding to get the clear picture.

# Esophotrast® · Barotrast®

BARNES-HIND BARIUM PRODUCTS

Subsidiary of Barnes-Hind Pharmaceuticals, Inc.
Sunnyvale, Calif. 94086





# The New Instant X-Ray.

## Polaroid now has a translucent X-ray film. It develops in the operating room, not the darkroom.

Polaroid's latest X-ray film, Type TLX, is a 3000-speed film that produces a 10 x 12-inch translucent radiograph on the spot. Because it is translucent, diagnostic quality is greatly improved. The picture is sharply detailed and can be read on a view box. Exposure latitude is comparable to wet-process films. Because of its sensitivity, exposures can be made with about half the radiation required for conventional X-rays.

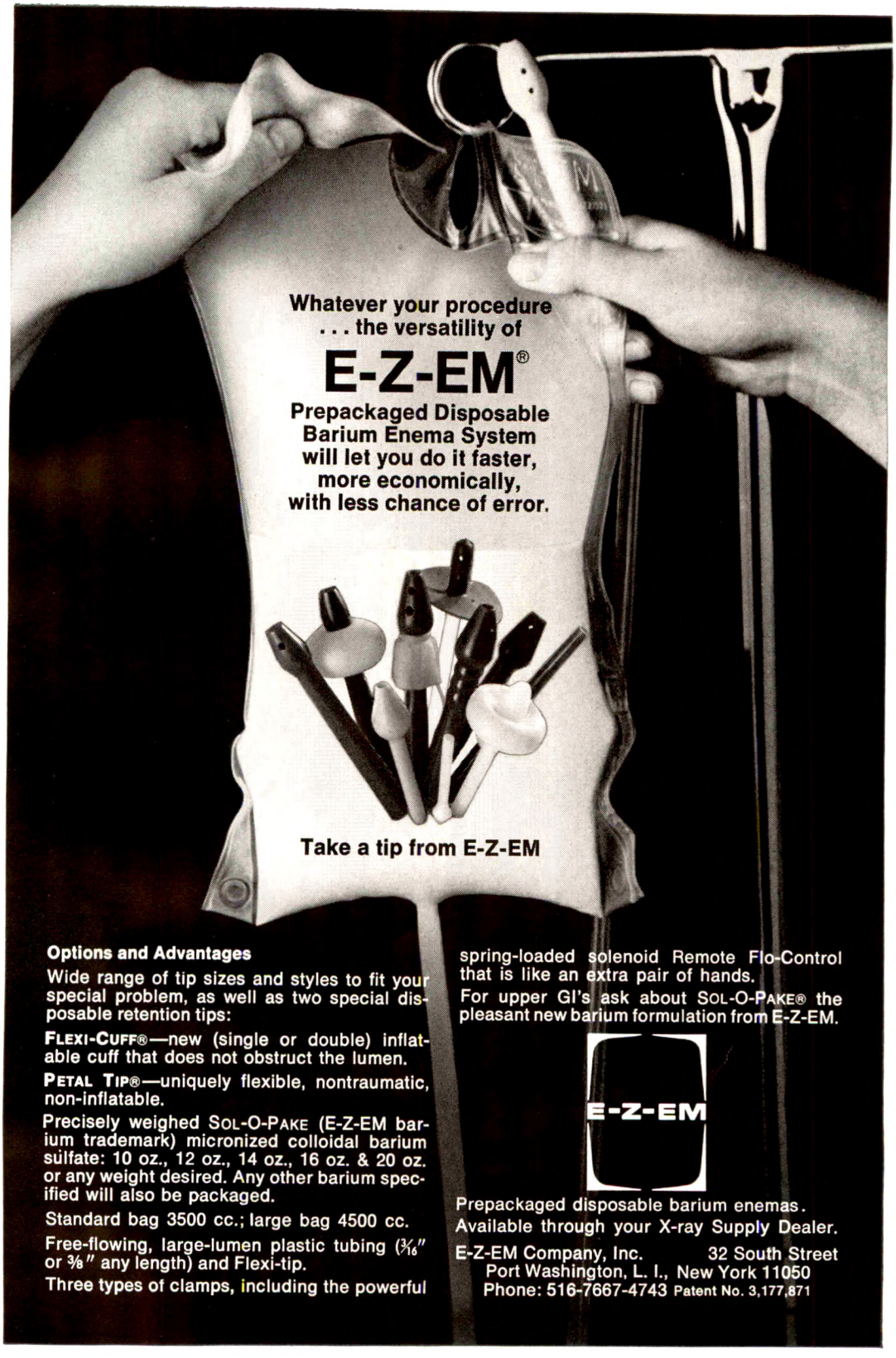
The importance of TLX film is that it brings the advantages of instant radiography—rapid viewing, minimal radiation—to many more medical procedures. TLX radiographs can now be used as a diagnostic tool. And they can provide detailed X-ray information in the operating room. Development time is 45 seconds.

TLX can be used for operative cholangiograms (the one you see here will give you an idea of the film's diagnostic capability), in neurosurgical and cardiovascular procedures, and in operations that require accurate X-ray control of surgical maneuvers. In these latter procedures, TLX film reduces the pauses for pictures to at least half the time needed for the fastest-developing wet-process X-ray film (which also requires bulky, expensive processing equipment). A hip pinning can be shortened by half an hour.

Now that TLX is here, many existing surgical procedures have been made easier, faster, and safer.

Would you like more information or a demonstration? Just write Polaroid Corporation, Dept. 122, Cambridge, Massachusetts 02139.

**Polaroid TLX Radiographic Film** Polaroid®




**Options and Advantages**

Wide range of tip sizes and styles to fit your special problem, as well as two special disposable retention tips:

**FLEXI-CUFF®**—new (single or double) inflatable cuff that does not obstruct the lumen.

**PETAL TIP®**—uniquely flexible, nontraumatic, non-inflatable.

Precisely weighed SOL-O-PAKE (E-Z-EM barium trademark) micronized colloidal barium sulfate: 10 oz., 12 oz., 14 oz., 16 oz. & 20 oz. or any weight desired. Any other barium specified will also be packaged.

Standard bag 3500 cc.; large bag 4500 cc.

Free-flowing, large-lumen plastic tubing (3/16" or 3/8" any length) and Flexi-tip.

Three types of clamps, including the powerful spring-loaded solenoid Remote Flo-Control that is like an extra pair of hands.

For upper GI's ask about SOL-O-PAKE® the pleasant new barium formulation from E-Z-EM.

E-Z-EM

Prepackaged disposable barium enemas.
Available through your X-ray Supply Dealer.

E-Z-EM Company, Inc. 32 South Street
Port Washington, L. I., New York 11050
Phone: 516-7667-4743 Patent No. 3,177,871

THE NORELCO PROGRAMMED SYSTEM:

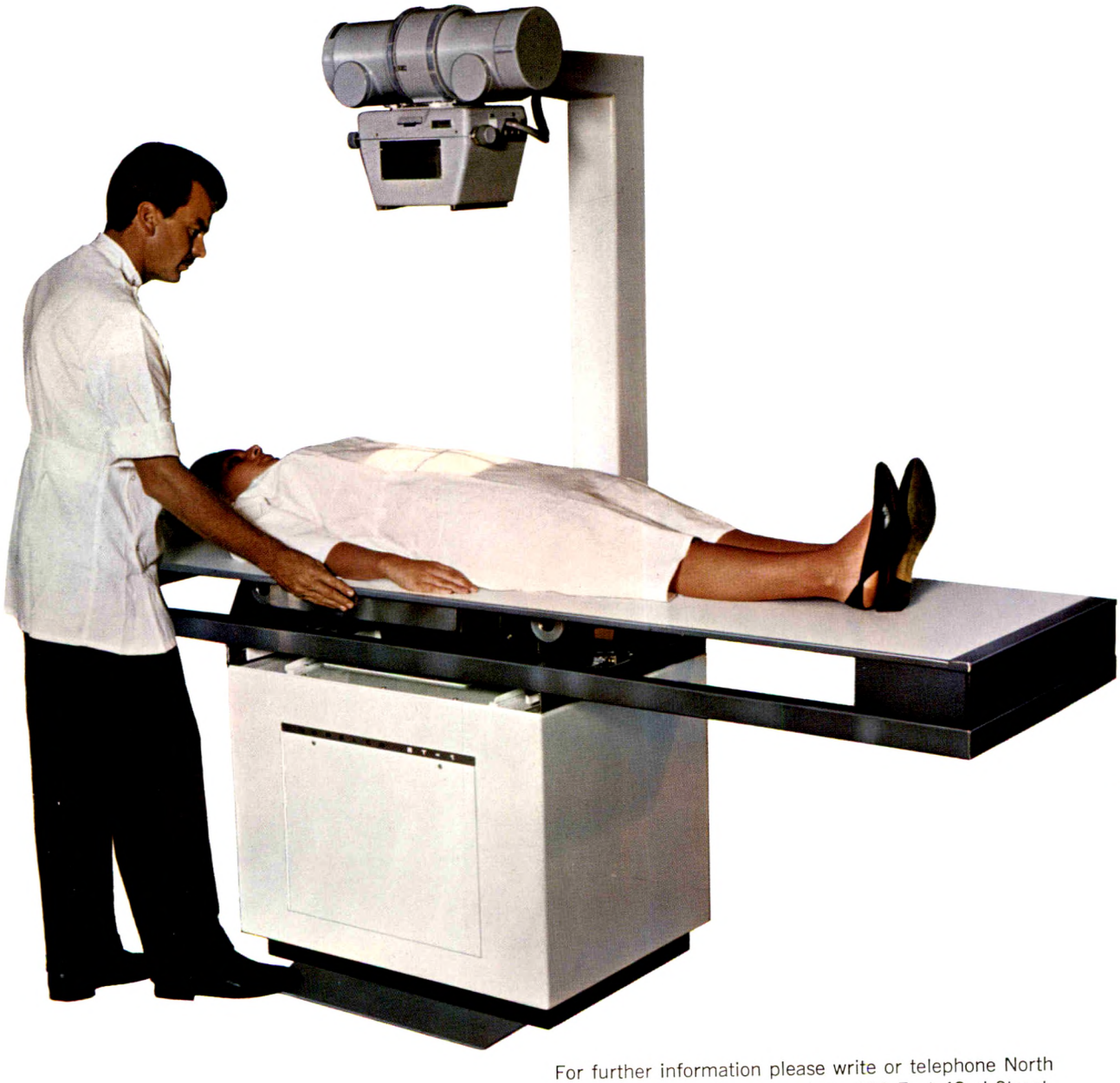
# MORE EFFICIENT USE OF FACILITIES, PERSONNEL and TIME




This automated laboratory for routine radiographic procedures eliminates facto selection, technique charts, tube centering, collimator adjustment and FFD adjust ment. It enables technologists to concentrate on patient care and positioning, while producing consistently excellent radiographs – in the shortest possible times.

Under your guidance, exposure data are programmed and set into the Norelco System Control – in place of printed exposure charts. Once the control and installation are set. The System does everything except choose the examination, position the patient

# ALL YOU DO IS
# CHOOSE THE EXAMINATION, POSITION THE PATIENT, AND INITIATE THE EXPOSURE.



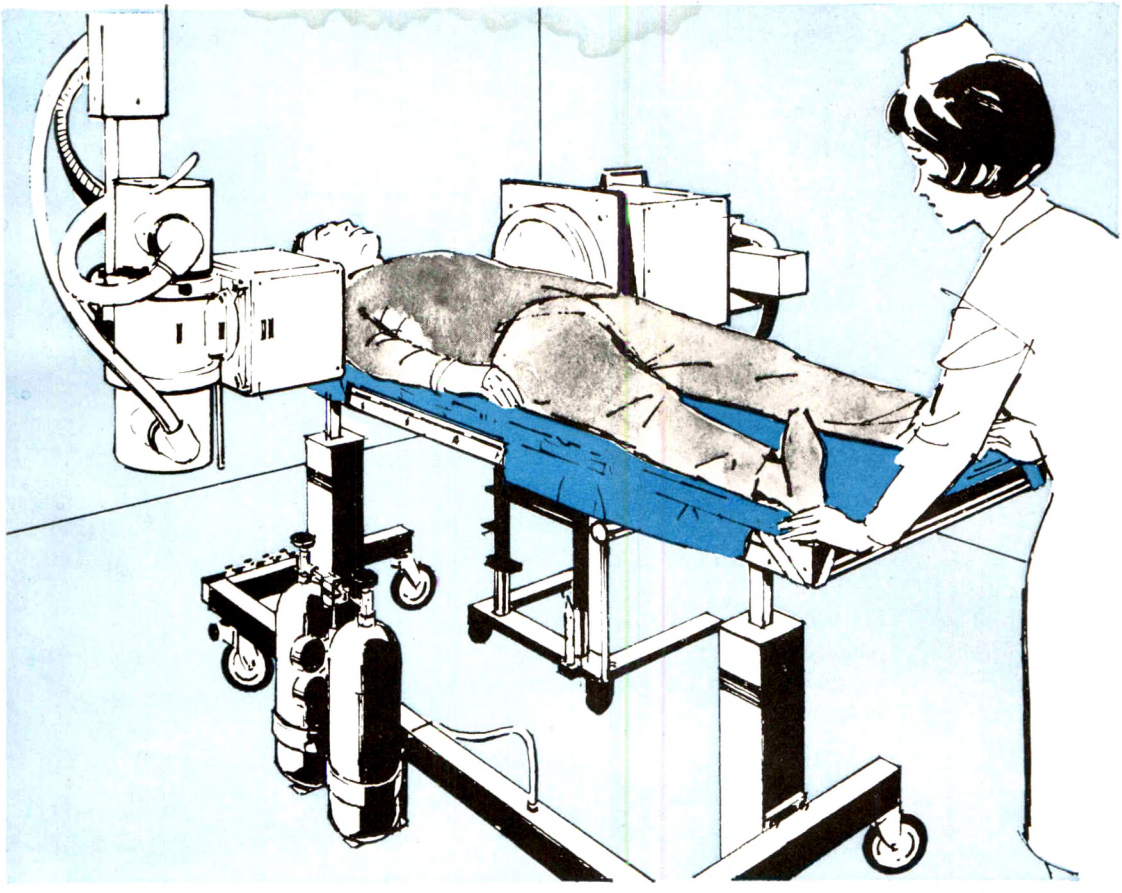
For further information please write or telephone North American Philips Company, Inc., 100 East 42nd Street, New York, N.Y. 10017 (212-697-3600), contact your Norelco Branch Office or your local Norelco Medical Equipment Dealer.

M-47

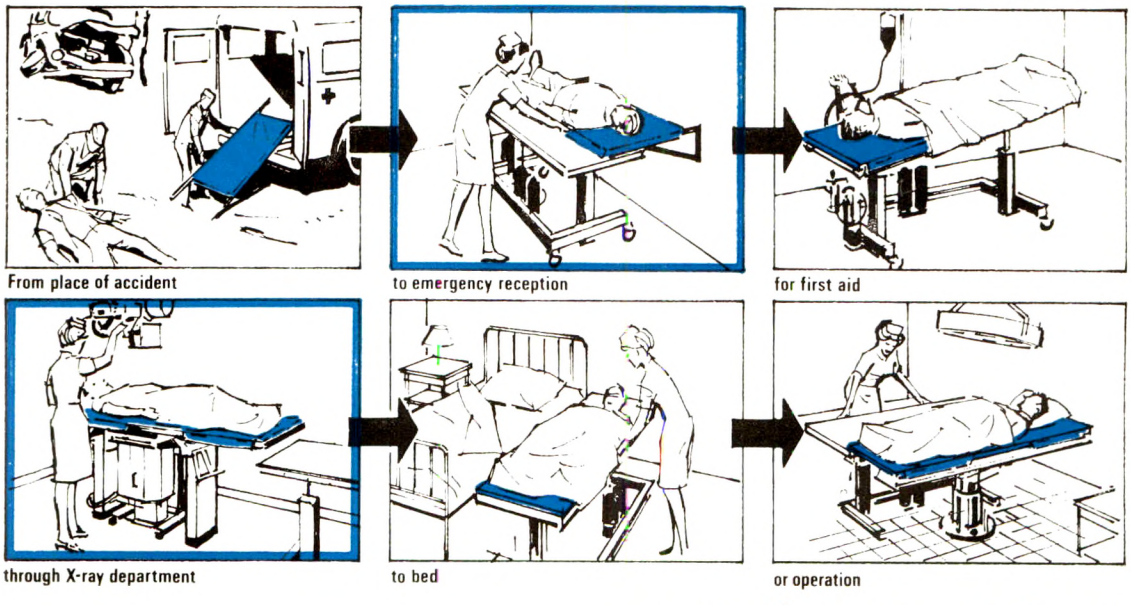
Norelco® MEDICAL EQUIPMENT

NORTH AMERICAN PHILIPS COMPANY, INC.
Professional Products Division, 100 East 42nd St., New York, N.Y. 10017

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Write for details of the E-S system for transport and care of casualty patients.

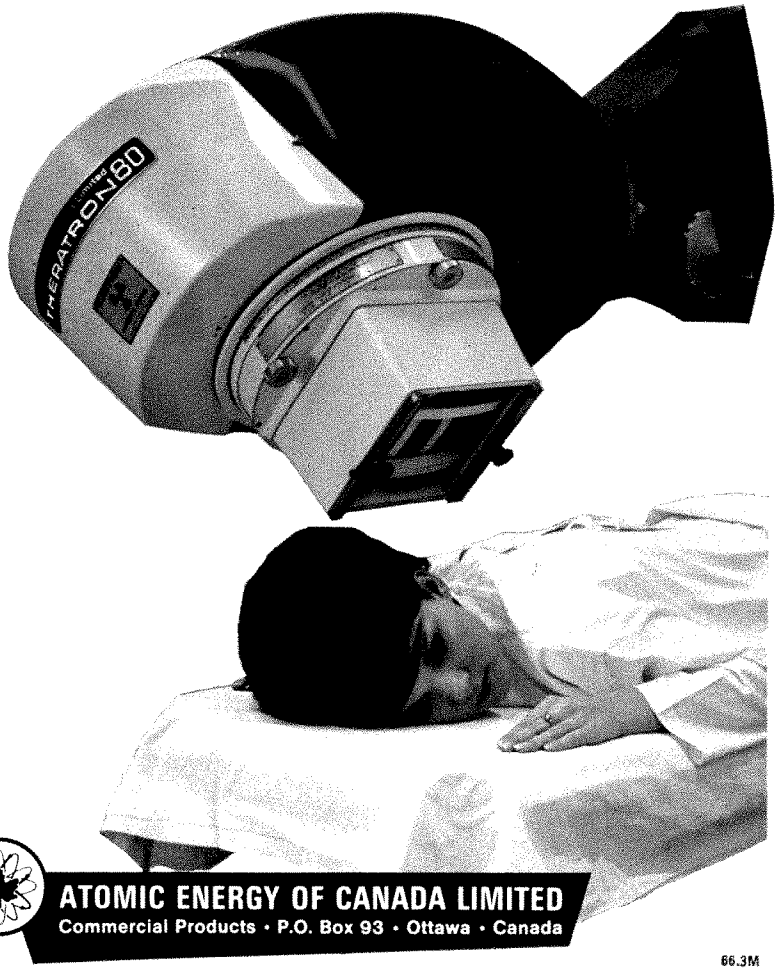
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**Telephone 312 259-7206**

# PROVEN PERFORMANCE!

## ELDORADO AND THERATRON

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We would like to tell you more — we would like to give you facts, figures and specifications. We have representatives all over the world. Write for full information.




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**SQUIBB**

a research concept in contrast visualization

# for better definition of disease: documenting differences between contrast agents

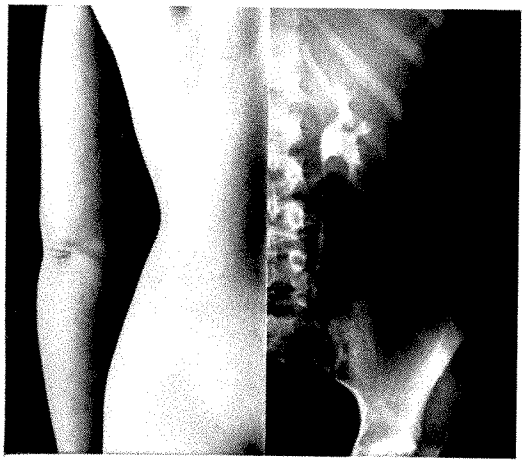
**No. 1 in a series:**

Renografin-60 (meglumine diatrizoate injection) preferred in a study of pyelographies of 2,234 patients.

In a new large scale study,[1] "...to determine which medium would produce adequate visualization of the urinary tract with the fewest toxic effects on the patient," Macht *et al.* have compared Renografin-60 with 2 other contrast agents.

Data were analyzed in 2,234 unselected (consecutive) patients, according to age, sex, and general disease group for the study population as a whole. The first 683 patients received 50% diatrizoate sodium solution, the next 921 patients received Renografin-60, and the final 630 patients received 66.8% sodium iothalamate solution.[1]

**criteria for quality and comparative safety** The contrast agents were evaluated for quality of diagnostic films as follows: films showing a dense concentration of contrast medium with filling and visualization of all major and minor calyces, infundibula, pelves and almost all of each ureter were listed as "good"; films showing less concentration with incomplete visualization of all portions of urinary tract but sufficient to produce diagnostically adequate films were listed as "fair"; films showing unsatisfactory visualization of urinary tract, or films which could not be interpreted, as "poor." The media were also evaluated as to incidence of the following side effects: nausea, vomiting, fainting, shock or severe reaction, hiccups, hives, pain in arm, sneezing, hot flushes, stuffiness of nose or ears.[1]



The following chart* shows comparative results of the 3 media according to age category:

*quality of pyelograms and side effects expected in standard population of 1,000 patients by age, according to type of medium used*

| medium used and type of patient | standard population | quality of pyelograms good | fair | poor | with side effects |
|---|---|---|---|---|---|
| meglumine diatrizoate | | | | | |
| all ages, | 1,000 | 827 | 123 | 50 | 41 |
| 0-19† | 126 | 122 | 3 | 1 | 3 |
| 20-49 | 415 | 374 | 35 | 6 | 27 |
| 50-69 | 315 | 240 | 51 | 24 | 9 |
| 70 or older | 144 | 91 | 34 | 19 | 2 |
| diatrizoate sodium | | | | | |
| all ages, | 1,000 | 782 | 134 | 84 | 72 |
| 0-19 | 126 | 115 | 6 | 5 | 7 |
| 20-49 | 415 | 378 | 26 | 11 | 40 |
| 50-69 | 315 | 227 | 64 | 24 | 19 |
| 70 or older | 144 | 62 | 38 | 44 | 6 |
| sodium iothalamate | | | | | |
| all ages, | 1,000 | 883 | 81 | 36 | 54 |
| 0-19 | 126 | 122 | 4 | 0 | 7 |
| 20-49 | 415 | 393 | 20 | 2 | 29 |
| 50-69 | 315 | 269 | 32 | 14 | 13 |
| 70 or older | 144 | 99 | 25 | 20 | 5 |

*Adapted from Macht[1] †In some patients in this age group, higher than recommended doses were used.

**In theory:** "The choice of contrast agent should ideally be individualized according to the age, sex, and disease group of the patient in order to obtain a high probability of complete visualization of the urinary tract with low probability of adverse side effects."[1]

**In practice:** "The choice is to be made...on the basis of the agent which gives the best concentration and the fewest side effects in the greatest number of patients regardless of age, sex, or disease category."[1]

**In order of preference:** Although Renografin-60 was not rated highest in all categories, the authors feel that their preference of the contrast media for intravenous pyelography would be: first, Renografin-60; second, sodium iothalamate; third, diatrizoate sodium.[1]

For brief summary of prescribing information, please refer to the end of this advertisement.

# definition of safety...

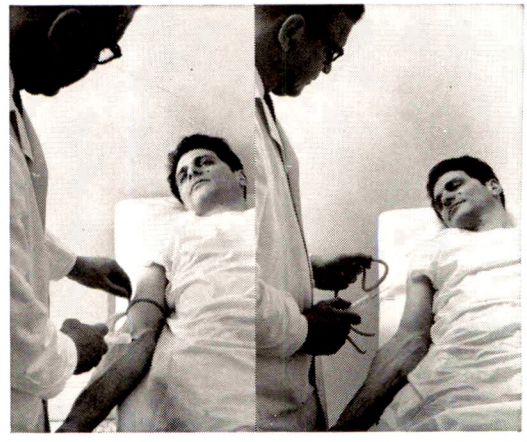
**demonstrated in animals...** Since meglumine diatrizoate is also used for cerebral angiography, toxicity studies of administration via the carotid artery are therefore of interest. Fischer and Eckstein[2] designed angiographic studies in animals in which procedures were very similar to clinical angiography, and which yielded data that was quantitative, graphic and nonsubjective.[2] According to the authors: "We selected the alterations in arterial blood pressure, venous pressure, heart rate and rhythm, the electrocardiogram and end-expiratory $CO_2$ concentration resulting from experimental cerebral angiography as refined, sensitive indications of the toxicity of a particular contrast material."[2] Their results of measured cardiovascular functions in dogs indicated that meglumine diatrizoate was far less toxic than four other contrast media.[2] As Fischer and Cornell reported in a later study: "Despite the testing of more highly concentrated solutions and larger doses, the cardiovascular responses [in dogs] from methylglucamine [meglumine] salts were much less than from sodium salts, an observation consistent with previous experiments."[3]

Other investigators have documented the comparative safety of meglumine salts in experimental studies. In order to determine reaction and tolerance of the intestines, Cooley[4] injected meglumine diatrizoate into mesenteric arteries of dogs and found no damage. Gensini *et al.*[5] reported that cardiovascular responses with it were almost identical to blood transfusions.

**theory of lower toxicity with meglumine...** Gensini and DiGiorgi have offered a hypothesis to explain their findings of lesser toxicity with experimental intravascular injections of methylglucamine (meglumine) salts. "When a relatively undiluted amount of sodium salts of a contrast agent is injected in an artery and carried by the blood stream toward the capillary bed, its molecules rapidly dissociate and readily diffuse through the capillary membrane and into the tissue. There, both the toxic effect of the iodine-containing organic radical and the increased concentration of sodium will readily manifest themselves. At equal concentrations of sodium, the end results will closely reflect the intrinsic toxicity of the iodine-containing organic radical on the tissues...."[5]

"In the case of the methylglucamine compounds, the same dissociation takes place. However the larger methylglucamine molecule, rich in hydrogen bonds, apparently either limits the migration of the organic radicals outside the vessel or at least minimizes their effects on the cellular metabolism."[5]

**proved in practice...** Paralleling similar findings in animals, clinicians have reported a generally lower



incidence of untoward reactions with Renografin-60 in urologic and cerebrovascular use. However, as with all intravascularly injected contrast agents, the possibility of severe reactions should be kept in mind (see Contraindications, Precautions and Side Effects below). In one study of over 600 urologic patients, the investigators reported that Renografin-60 produced urograms of diagnostic quality with a 6% incidence of side effects. The authors concluded: "It is hard to believe that any drug introduced intravenously could be so well borne by so many patients...."[6]

In a 74-patient study,[7] comparing Renografin-60 with diatrizoate sodium in carotid arteriography, Shealy commented: "With confused patients who are to have arteriography under local anesthesia, it is particularly desirable to have an agent that causes little pain." In this study, since "...60 per cent Renografin has resulted in considerably less pain than 50 per cent or 45 per cent [diatrizoate sodium]...we have converted to the routine use of 60 per cent Renografin for carotid arteriography; an additional 1,500 arteriograms done with 60 per cent Renografin have been quite satisfactory."[7]

**better tolerated even in pediatrics...** Citing some difficulties in administering contrast agents intravenously to children, Strasser *et al.*[8] selected Renografin-60 for intramuscular use in excretion urography in 16 pediatric patients because of the mild and relatively few reactions consistently associated with its use. The authors concluded: "The almost complete absence of any kind of local effect from its injection into the gluteal muscle and the absence of any serious reactions, local or systemic, indicate the safety of the medium."[8]

For brief summary of prescribing information please refer to the end of this advertisement.

# definition of efficacy...

**a thoroughly investigated meglumine salt** Extensively evaluated for over a decade, Renografin has been consistently shown to yield a high percentage of diagnostic quality films in many phases of contrast visualization.

Upon intravenous injection, Renografin is rapidly carried to the kidneys and is so well concentrated that renal passages—including renal pelvis, ureters and bladder—may be clearly visualized. This medium also provides high contrast vasography in visualization of the cerebral vessels and the peripheral arteries and veins.

**proved diagnostic excellence...** In a comparative study[9] of 3 contrast agents used in cerebral angiography by Doehner (comprising a cross section of an average neurosurgical practice), Renografin-60 was equal in the arterial phase and slightly superior in the venous phase of the examination.

Findings of a previously cited study by Orr *et al.*,[6] in intravenous pyelography, also attest to the diagnostic excellence of Renografin-60. "Satisfactory roentgenograms of the kidneys were obtained in 636 (97%) of the cases, demonstrating the great diagnostic value of this procedure." And, as noted previously, Shealy found the contrast agent to be "quite satisfactory" in 1,500 carotid arteriograms.[7]

**References: 1.** Macht, S. H.; Williams, R. H., and Lawrence, P. S.: Amer. J. Roentgen. *98*:79 (Sept.) 1966. **2.** Fischer, H. W., and Eckstein, J. W.: Amer. J. Roentgen. *86*:166 (July) 1961. **3.** Fischer, H. W., and Cornell, S. H.: Radiology *85*:1013 (Dec.) 1965. **4.** Cooley, R. N., *et al.*: Angiology *15*:107 (Mar.) 1964. **5.** Gensini, G. G., and DiGiorgi, S.: Radiology *82*:24 (Jan.) 1964. **6.** Orr, L. M.; Campbell, J. L., and Thomley, M. W.: J.A.M.A. *169*:1156 (Mar.) 1959. **7.** Shealy, C. N.: J. Neurosurg. *20*:137 (Feb.) 1963. **8.** Strasser, N. F., *et al.*: Radiology *79*:408 (Sept.) 1962. **9.** Doehner, G. A., and Brugger, G. E.: New York J. Med. *60*:4022 (Dec.) 1960.

**Contraindication**

A history of sensitivity to iodine per se or to other contrast media is not an absolute contraindication to the use of meglumine diatrizoate.

**Precautions and Side Effects**

Severe, life-threatening reactions are rare; when they occur they suggest hypersensitivity. A personal or family history of asthma or allergy warrants special attention and may predict, more accurately than pretesting, the likelihood of a reaction, although not the type nor severity of the reaction in the individual.

The value of any pretest is questionable. The pretest most performed is the slow injection of 0.5-1.0 cc. of the preparation into a peripheral vein. An impending reaction is often indicated by transient burning and flushing, pain, "jump-like" reactions, respiratory difficulty, faintness, sneezing, itching, nausea, vomiting or urticaria. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. Antiallergic drugs may be used to advantage. In a few cases, the reactions to the test dose have been extremely severe.

The more serious anaphylactoid reaction requires immediate treatment and may occur despite a negative sensitivity test. An emergency tray consisting of vasopressor drugs, epinephrine hydrochloride 1:1000, methoxamine (Vasoxyl) or metaraminol bitartrate (Aramine), and glucose and saline is recommended. Oxygen and instruments to guarantee a clear airway must be readily available.

Caution must be exercised, especially in cerebral angiography in extreme age, in severely debilitated patients and in those with marked or severe hypertension, advanced arteriosclerosis, cardiac decompensation, recent cerebral embolism, or thrombosis, chronic pulmonary emphysema and in cyanotic infants.

For full details the Package Insert should be read.

**Supply:** Renografin-60 is supplied in vials and ampuls as a sterile aqueous solution providing 60% meglumine diatrizoate containing approximately 29% firmly bound iodine.

Available in 30 cc. vials or 25 cc. ampuls.

**in excretion urography**
**cerebral angiography**
**peripheral arteriography**
**and venography**

## Renografin®-60

**Squibb Meglumine Diatrizoate Injection U.S.P.**

SQUIBB 'The Priceless Ingredient' of every product is the honor and integrity of its maker.

# Du Pont focuses its science and energy on Diagnostic Clarity



# ...latest result: a new CRONEX® screen that eliminates the need for wedge filters.

This bilateral venogram was reproduced from a radiograph taken without wedge filters.

It's a demonstration of our new 14″ x 36″ gradient intensifying screen. The screen speed is adjusted to the range of thickness of the average pelvis and lower extremity.

The result: a radiograph uniform in contrast and detail from end to end—without the use of wedge filters.

This unique screen—like all CRONEX screens—has Du Pont's durable moisture-resistant polyester base; for long life, easy cleaning, and superior antistatic properties.

Your Du Pont Technical Representative can show you a full-size radiograph made with our new screen.

The development of this new screen is today's proof of our total commitment to Diagnostic Clarity. Tomorrow it might be a new film, or a new chemistry, or something else that will improve the radiographic process.

DU PONT

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## CRONEX X-ray Products

On Du Pont CRONEX 2 D.C.: bilateral venogram, 54-year-old female. 300 ma, 3/20 sec., 72 kv, 18 cm. patient, 72″ F.F. Dist. Grid Tunnel. **2 grids butting cause black line.**





# 7 minutes?
# 3½ minutes?
# 90 seconds?

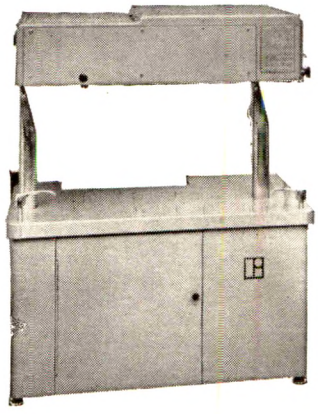
Pick a system... *any* system.
Whatever automatic processing setup you choose, you can count on Liquamat chemicals to bring out the best in your radiographs.
Liquamat-matched solutions deliver top quality images... consistently...with no contrast drop-off and no sludge build-up.
They're adaptable to any automatic processing system.
So *you* make the decisions. The processing time you choose will be a good one – with GAF Liquamat chemistry.

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# Same Old Sanchez-Perez Automatic Seriograph.

There she sits. Our baby. For more than 15 years, she's been our baby. Try as we may, she's a tough one to improve on.

So we're stuck with her. Same old reliable design. Same old simple, trouble-free operation. Same old dependability. (And still the only film changer in its price class.)

She couldn't be too bad, though. Lots of our customers keep as many as three and four Automatic Seriographs busy at a time! Whenever they need an additional film changer, back they come for our Sanchez-Perez. Guess they kind of like it.

Sure, we've made some improvements here, some changes there—and we'll make more in the future. But basically she'll still be our same old Seriograph. We're stuck with her.

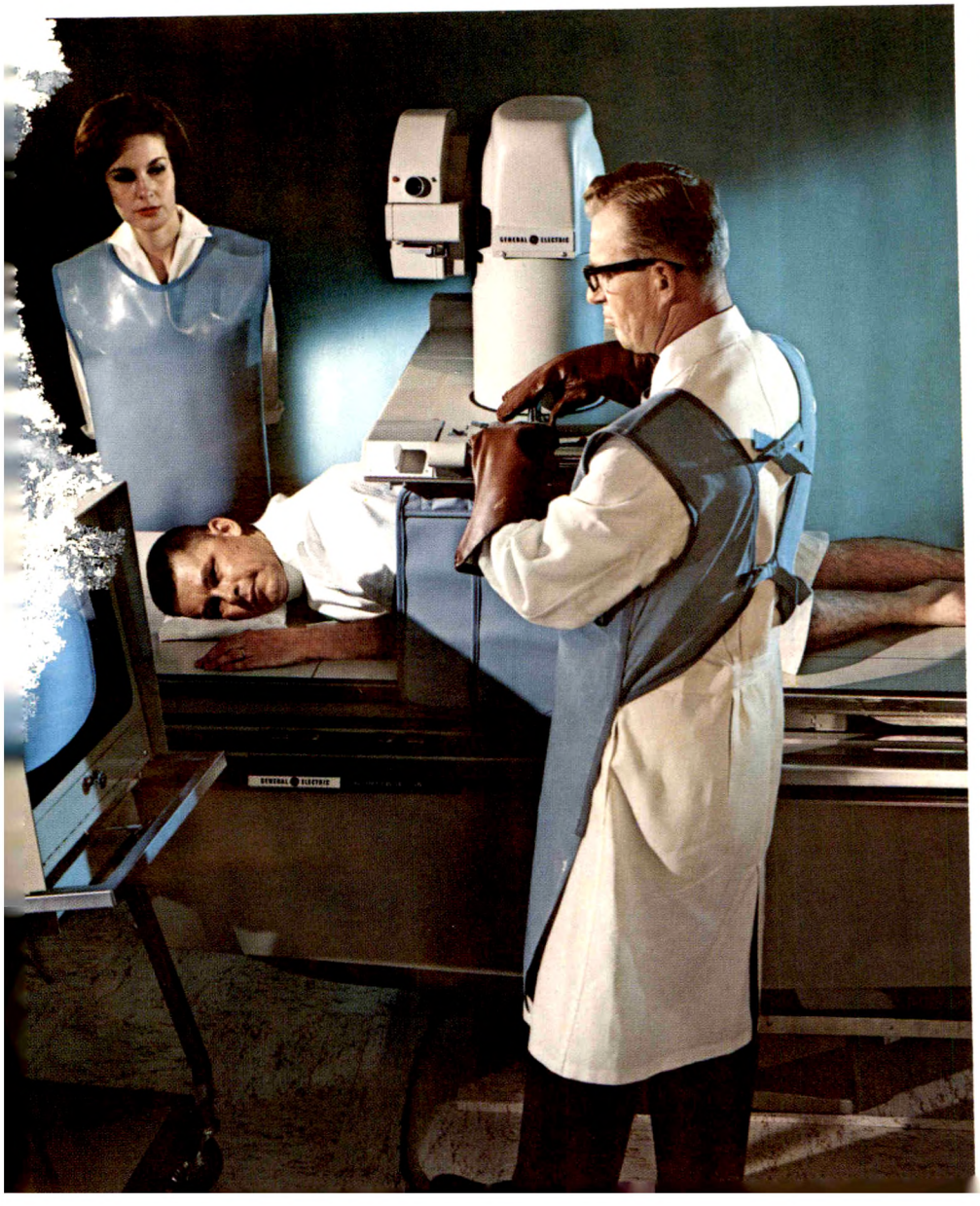
**THE AUTOMATIC SERIOGRAPH CORPORATION**
DIVISION OF LITTON INDUSTRIES
**P.O. Box 5000 • Des Plaines, Illinois**

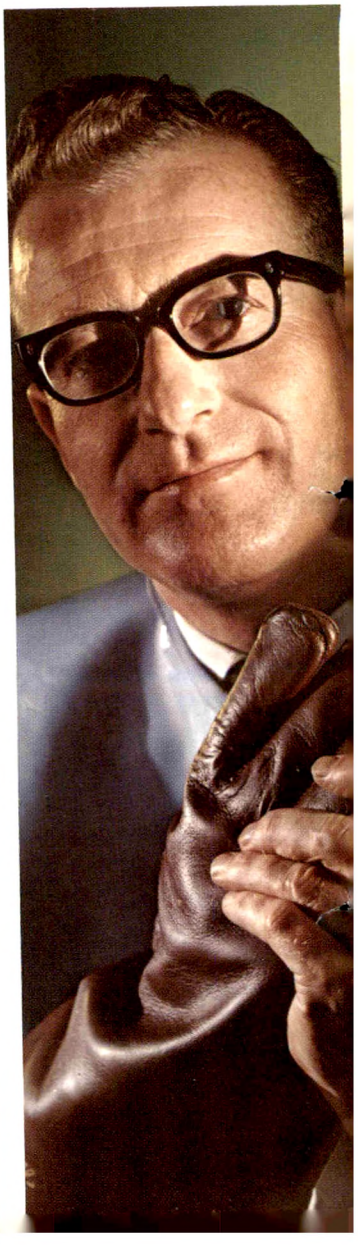
# FLUORICON

COMPACT-VIDEO IMAGE INTENSIFIER












# Critically Precise Fluoroscopy

The Fluoricon Compact-Video image intensifier combir a conversion factor of 80 and maximum detail with TV monitoring. The Relicon TV system is designed expre image intensification and clearly reproduces the total information available on the intensifier tube. The compact size and light weight of this unit make it eas position without power assistance. Optional additions to the Fluoricon Compact-Video include: 70-mm spot-film camera with automatic exposure control and video tape.

Continuous reliability for every viewing and recording technic is a major advantage of the entire General Electric line of image intensifiers — full-function Fluoricon II, Compact-Video or Compact-Optical. For accuracy, reliability and convenience, they are the fines diagnostic performers on the market. When there's no room for error, there's room for Fluoricon.



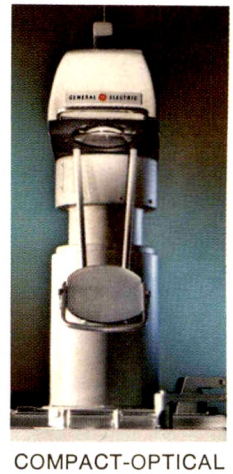
*Progress Is Our Most Important Produ*

GENERAL ELEC

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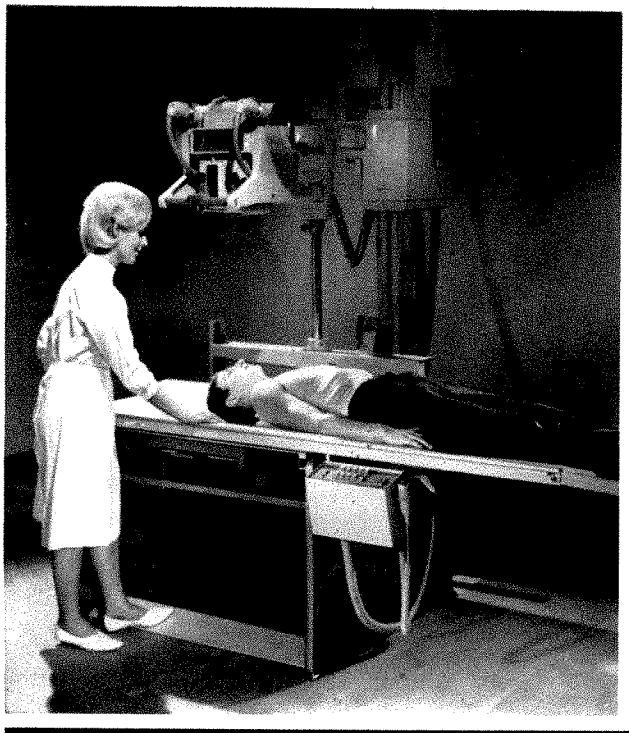
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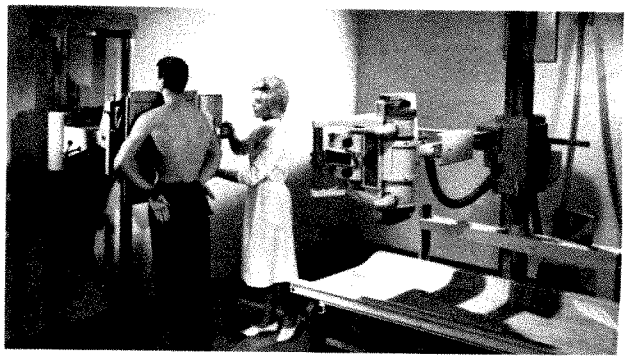


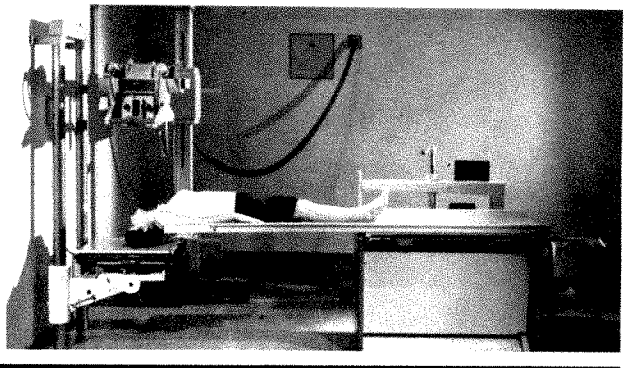
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**A multi-purpose unit**

for planigraphy with linear, elliptical, and circular obscuring movements

for radiography with small exposure angle

for Bucky radiography using a vertical or oblique beam projection

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Convenient and time-saving settings and reliable diagnostic results are ensured by the use of the SIRECON image-intensifier television assembly.

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# For a clear picture "prep" the colon with pleasant-tasting X-PREP Liquid

(standardized extract of senna fruit)

Specifically designed for preradiographic bowel evacuation. X-PREP Liquid permits excellent visualization in G.I. and urologic roentgenography.

No significant gas shadows. No residual oil droplets.

X-PREP Liquid reduces or eliminates the need for enemas prior to radiography.

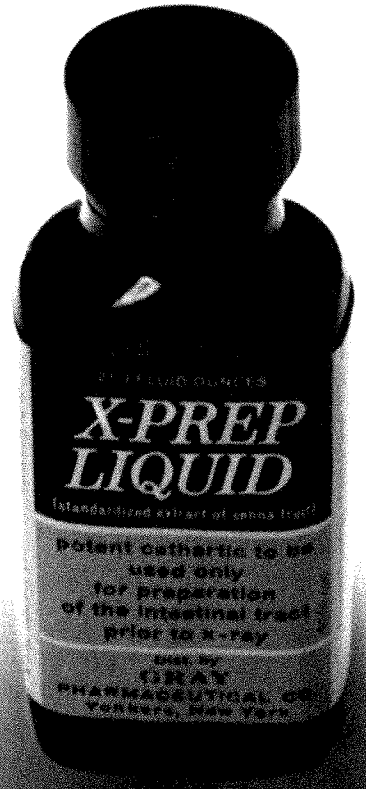
And delicious X-PREP Liquid gives a welcome reprieve for your patients from the obnoxious taste of castor oil.

Easy-to-take, X-PREP Liquid is also easy to administer. Premixed and premeasured, this effective bowel evacuant comes in small, single-dose bottles—to be taken between 2 and 4 p.m. on the day prior to radiography. Patients should be advised to expect strong purgative action.

**Contraindication:** Acute surgical abdomen.
**Supplied:** Ready-to-drink in 2½ oz. bottles (complete adult dose).
**Also available:** X-PREP Powder (standardized senna concentrate). Mixed with water, ¾ oz. canister provides complete adult dose.

GRAY PHARMACEUTICAL CO./Yonkers, N.Y.

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*X-ray visualization following barium enema. Patient prepared with single dose of X-PREP Liquid—2½ oz. Note absence of fecal retention. Courtesy of Statman, A. J.: An Effective Single-Dose Evacuant, J. M. Soc. New Jersey 63:95 (March) 1966.*

# "Now I have the choice I need."

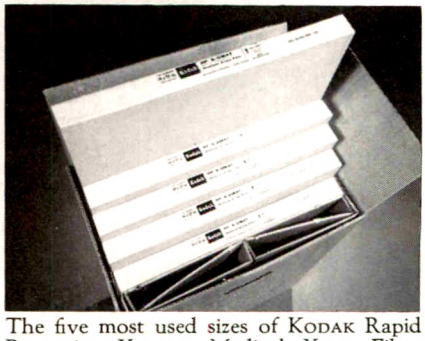
**ew from Kodak: a ird 90-second film.**

With the introduction of new DAK RP/L X-OMAT Medical X-ray m, you now have a choice of Kodak second films for all radiological ıations. The new film, with outnding brilliance in the lower denes and exceptional latitude in the per densities, gives you maximum gnostic range.



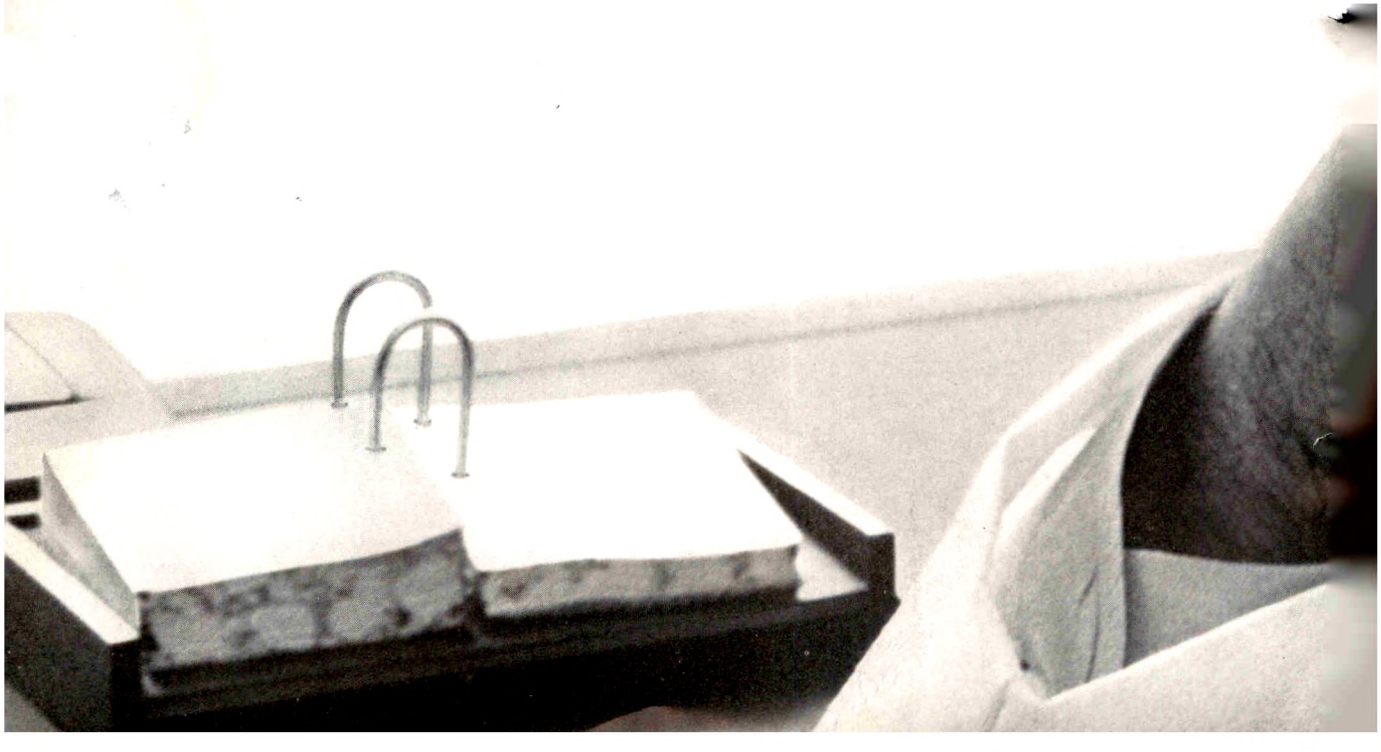
Joining KODAK RP X-OMAT Medical X-ray Film used for most situations and KODAK RP/S X-OMAT Medical X-ray Film for shorter exposures, it completes the picture; gives you the total capability you need in the fully integrated KODAK RP X-OMAT® System. No matter which film is right for a particular situation, you can develop a technic and rely on it. That's because Kodak quality and uniformity are built into each of the interdependent components of this system.

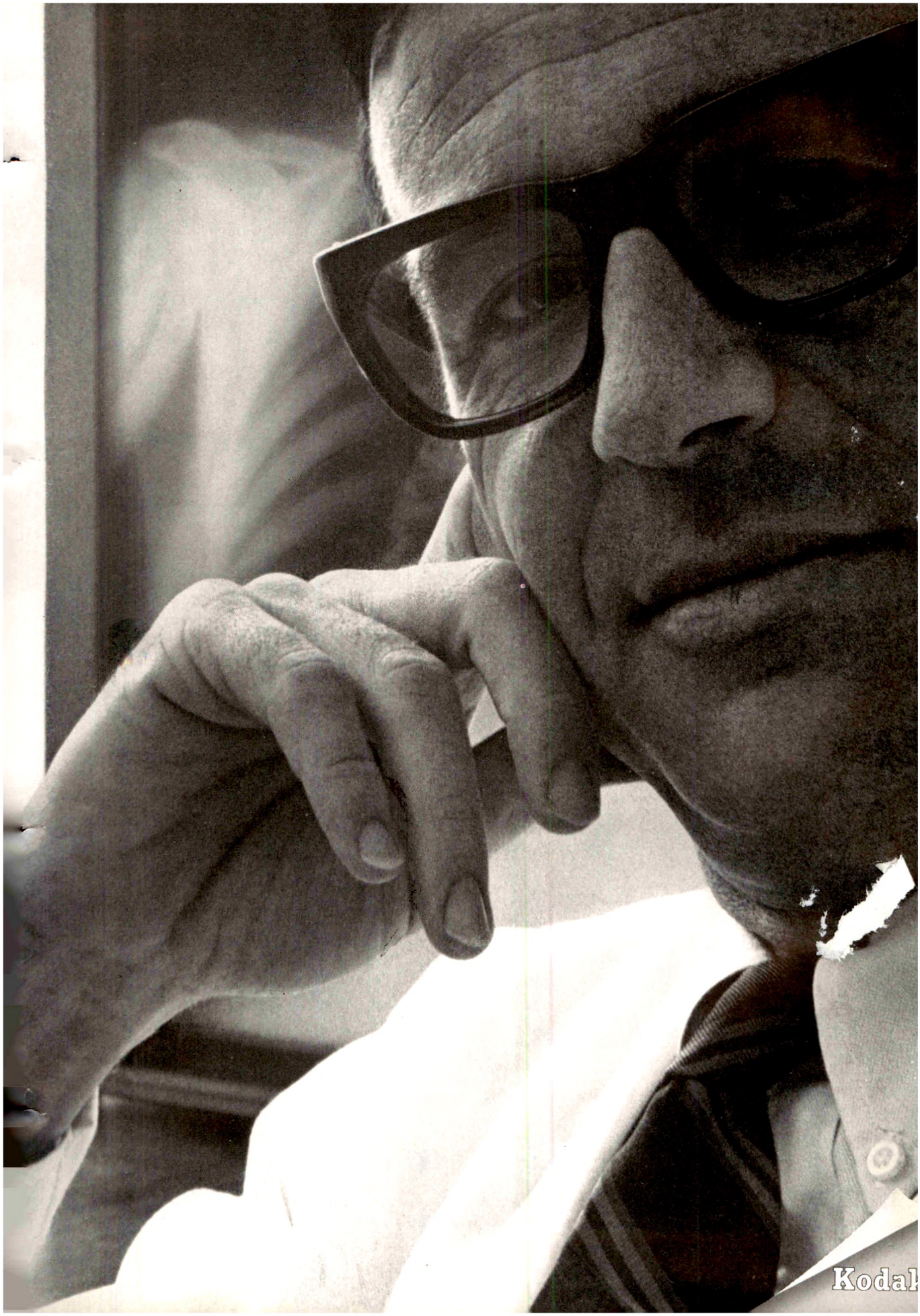
Your Kodak Technical Sales Representative will be happy to demonstrate the distinctive characteristics of each film. He'll also be able to tell you about the benefits of integrated 90-second Kodak processing . . . a complete choice of films, chemicals, and the new KODAK RP X-OMAT Processor, Model M6.

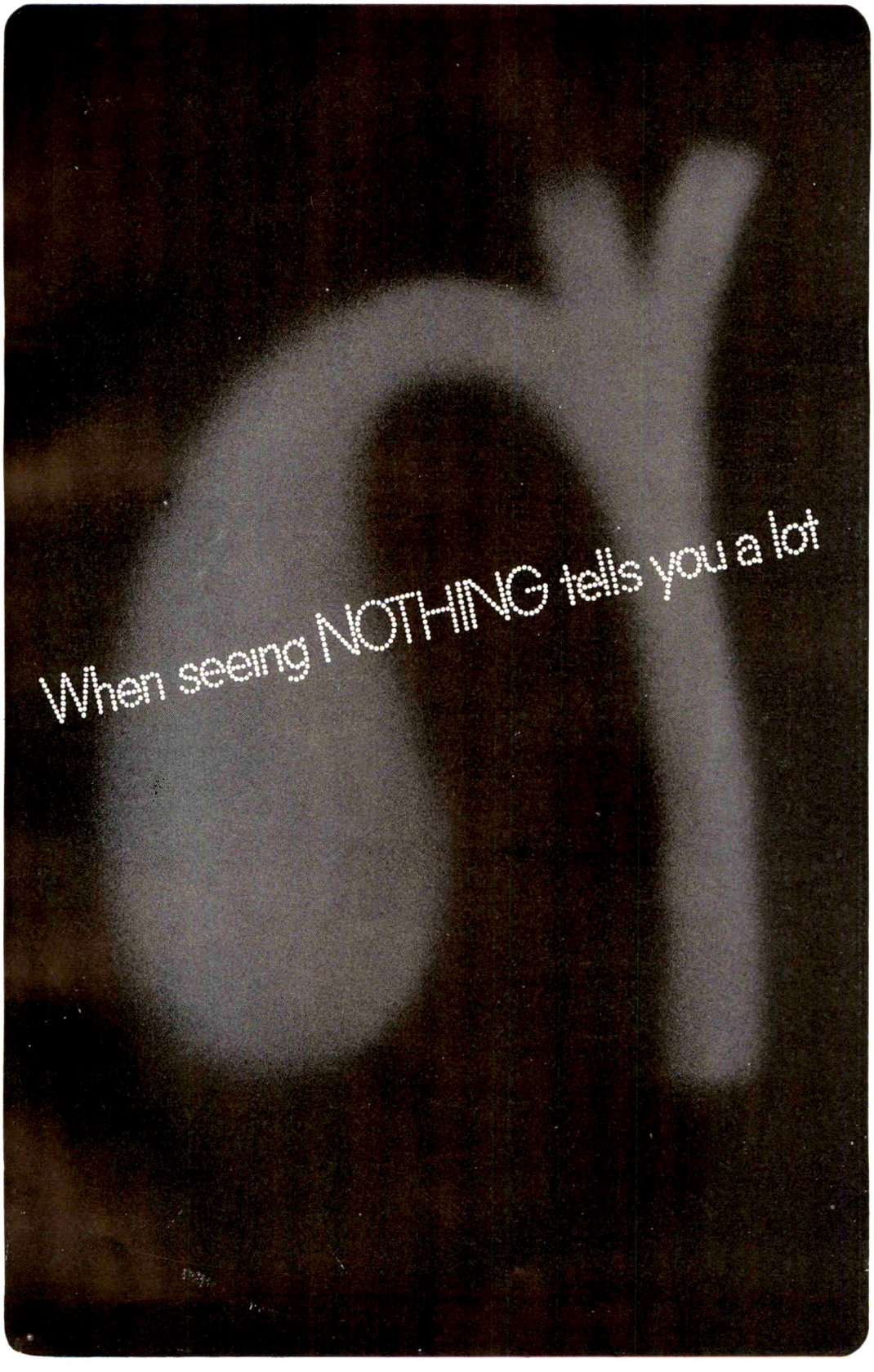


The five most used sizes of KODAK Rapid Processing X-OMAT Medical X-ray Films are, or soon will be, available in 500-sheet cases for added economy and convenience.

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# "Silent" gallbladders speak more clearly with Telepaque

With Telepaque *no* picture of the gallbladder tells you plenty—usually, *the presence of gallbladder disease**. When you get *full* visualization, you can be sure there is *no* gallbladder disease. And with Telepaque you can depend on your diagnosis!

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**Precautions:** Severe, advanced liver disease may interfere with metabolism of Telepaque, thus increasing the excretory load on the kidneys. Although renal difficulty has rarely been attributed to Telepaque, renal function should be assessed before cholecystography in severe, advanced liver disease, and renal output and hepatic function should be observed for a few days after the procedure. Patients with preexisting renal disease should not receive high doses of cholecystographic media. Possible renal irritation in susceptible individuals could result in reflex vascular spasm with partial or complete renal shutdown. Caution is advised in patients with coronary disorders, especially those with recent symptoms of coronary artery disease. Blood pressure should be observed after administration of cholecystographic media to these patients. Elevation of protein-bound iodine for several months and false positive urine albumin tests (for three days) may occur after ingestion of iodine-containing cholecystographic media.

**Adverse Reactions:** Most reactions are mild and transitory; serious side effects are very rare. Gastrointestinal effects (diarrhea, cramps, nausea, vomiting) are the most common. Usually the diarrhea consists merely of a few loose stools, although in isolated cases it may be severe. A mild stinging sensation during urination may occur, and rarely, skin rash, urticaria, pruritus, and flushing. One case of thrombocytopenia has been reported in a patient with a history of conjunctival hemorrhages. Subjective complaints have been: dryness of throat, burning on swallowing, heartburn, sore throat, dizziness, and headache.

**Usual Dosage:** 3 Gm. (6 tablets) at night after a light supper.

**Supplied:** Tablets of 500 mg., envelopes of 6 tablets, boxes of 5 and 25 envelopes; also bottles of 500.

*However other unusual causes of nonvisualization are occasionally encountered.

**Telepaque®** for precise oral cholecystography and cholangiography

brand of **iopanoic acid**

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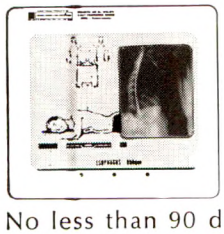
# gaf OVERHEAD TRANSPARENCY TEACHING AIDS can give your lectures added interest

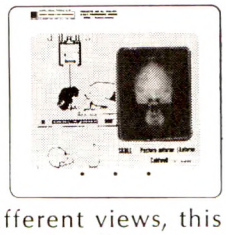
Now you can *visually* illustrate almost any point you wish with one of GAF's 250 overhead transparencies on basic x-ray technology.

And because they are designed for use with a conventional overhead projector, you can use them with the room lights on. You can read from your notes . . . your students can take them . . . and you can emphasize key points directly on the transparency.

There are a wide variety of basic courses—each with its own keyed study guide you can follow as a lecture guide or use to select those you want to supplement your own course outline.

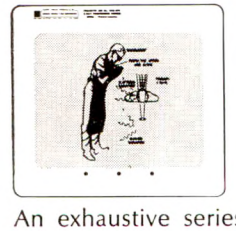
## Positioning Course for Radiologic Technology

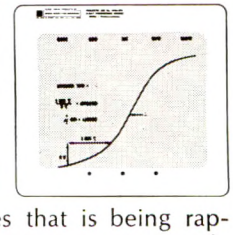




No less than 90 different views, this authorative series contains diagrams as well as reproduced radiographs. Extremely versatile, these transparencies can be used before, during or after practical demonstration.

## Radiologic Physics

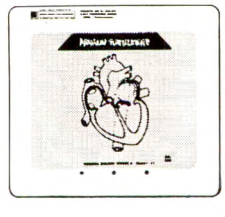




An exhaustive series that is being rapidly expanded. Four parts are currently available. From the "x-ray tube" through the "Patterson-Parker Tube", this series gives easily understandable illustrations of basic principles.

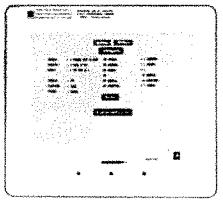
## Human Biology

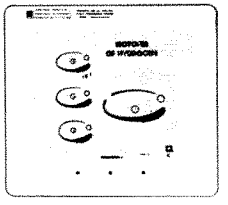




This series reproduces each system of the body. The skeletal muscles, digestive system, respiratory system, etc. And these sketches are not limited to major structures. For instance, the excretory system details the kidney tubule, as well as the human skin. Six separate and distinct transparencies are devoted to the nervous system.

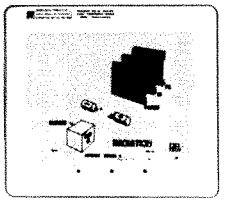
## Chemistry Series

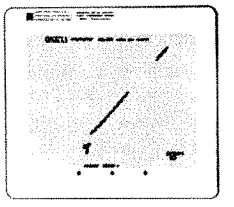




Take your students from the elements to Einstein in simple steps. All the basics are here—the laws, concepts and theories needed to clarify material in more advenced courses.

## Atomics and Electricity

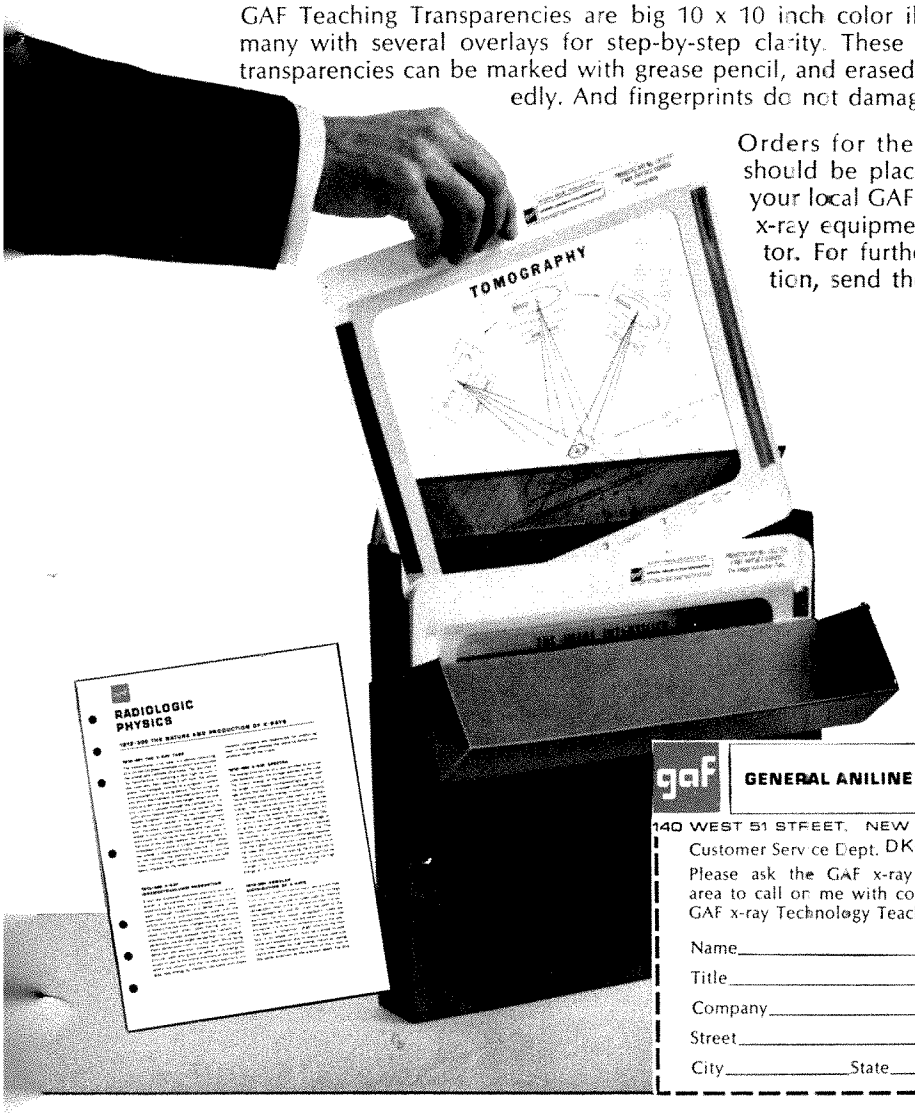




Here are two sets of transparencies in one. Atomic radiation, fission, fusion, etc. —plus the principals of magnetism, electrical transmission, circuits and symbols. Each is diagrammed simply.

GAF Teaching Transparencies are big 10 x 10 inch color illustrations, many with several overlays for step-by-step clarity. These big format transparencies can be marked with grease pencil, and erased . . . repeatedly. And fingerprints do not damage the film.

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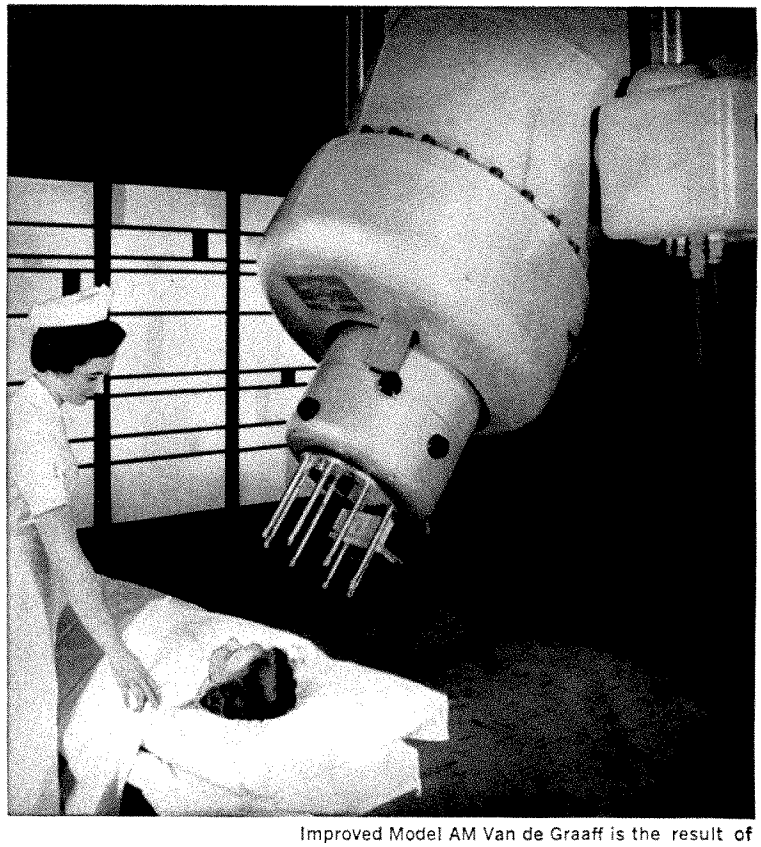
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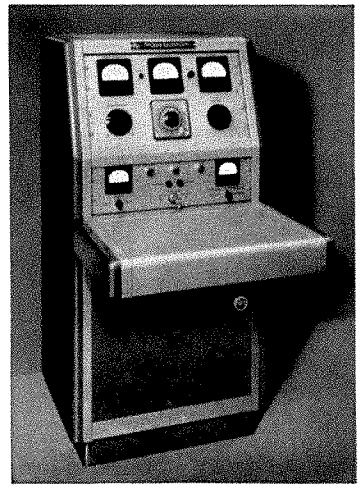


Improved Model AM Van de Graaff is the result of experience gained in 40 previous installations.

# IMPROVED *Van de Graaff*® ACCELERATOR MEETS THE MODERN NEED FOR LOW-COST PRECISION THERAPY

Today's Model AM 2 MeV Van de Graaff Therapy Unit continues to keep pace with the trend to low-cost precision therapy in leading hospitals and cancer clinics throughout the Free World. And for five good reasons:

- Its less than 3 mm "point source" of x-rays allows optimum technics for both large and small treatment fields.
- Its treatment beam is accompanied by negligible penumbra.
- Its Roentgen output exceeds that of a 5000 curie cobalt source.
- Its x-ray output will not fall off. You can hold treatment techniques and parameters constant.
- It can be operated easily by a technician without a staff physicist in attendance.

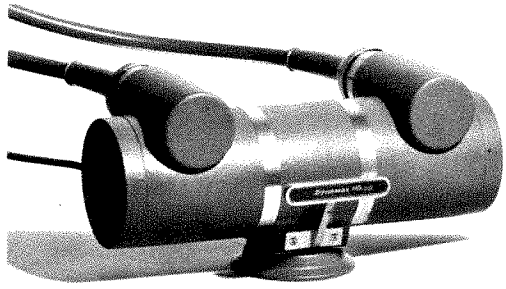


The Model AM Van de Graaff is a reliable, proven source of high-energy radiation. It offers more roentgens-per-dollar and has demonstrated less overall cost-per-treatment than any other comparable supervoltage source available today — including radioisotopes.

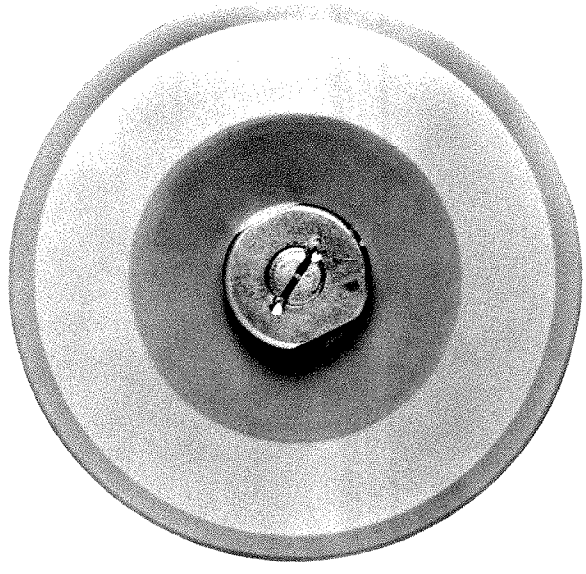
Full time use of the reliable Model AM 2 MeV Van de Graaff Therapy Unit will amplify your technic range and expand your patient handling capacity. It is backed by the international reputation of the leading manufacturer of particle accelerators — High Voltage Engineering Corporation. Write Medical Sales.

Today's Model AM is routinely operated by an easily-trained technician.

**HIGH VOLTAGE ENGINEERING CORPORATION**
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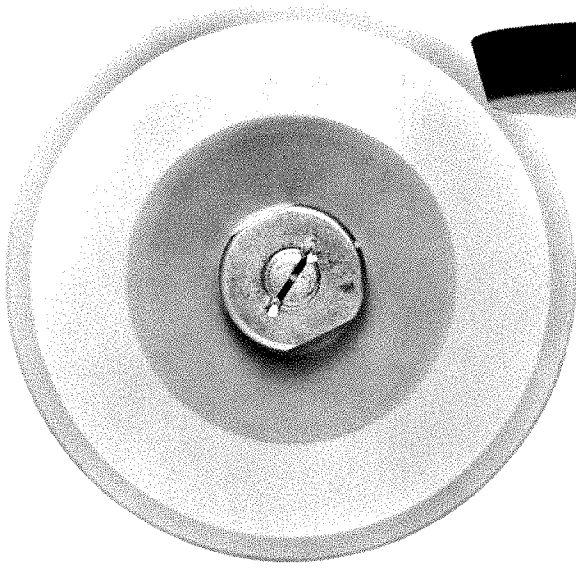
# To extend the range of performance: The Dynamax "HD50"

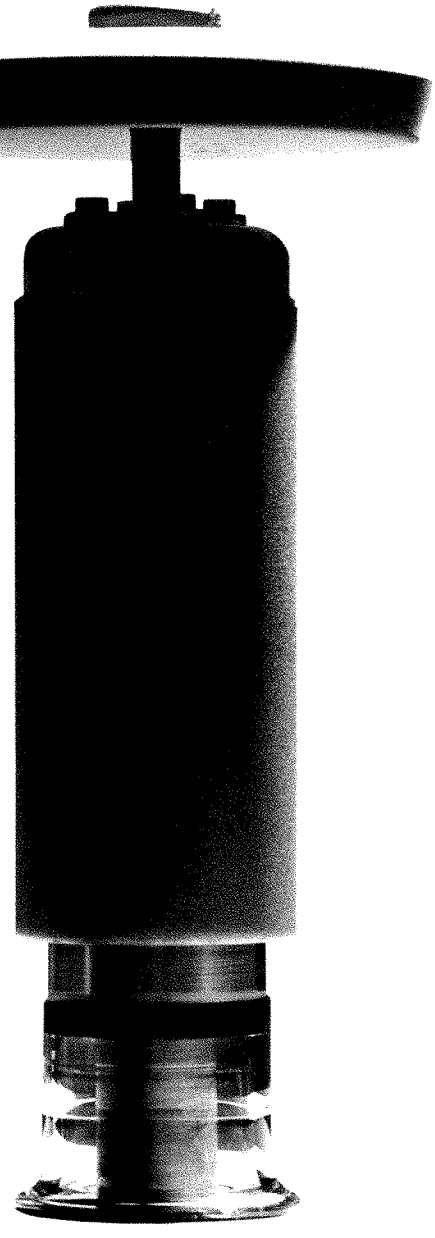


With this new compound 175,000 HU anode in the Dynamax "HD50", cine-radiographic and angiographic film run times can be extended, and routine work loads significantly increased.

MACHLETT

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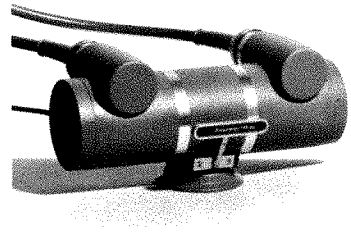


**The increased range of performance of the Dynamax "HD50"** is made possible by use of a tungsten-faced molybdenum anode, coated by a Machlett process, to increase its emissivity. This compound anode permits storage of larger thermal loads and rapid dissipation of anode heat, yet without increase in tube size. Despite the increased thickness of the Dynamax "HD50" target, it is lighter than the standard solid tungsten disc and has, therefore, improved mechanical as well as thermal performance.

**The "HD50" x-ray tube is available in all** high-speed or grid-controlled (or various combinations thereof) or standard.

**Note:** The Dynamax "HD50" insert may be used in standard Dynamax housings, including the Dynamax "HD25", "HD26"; Dynamax "40", "46", "48" and the Dynamax "50". The "HD50" insert may be obtained either as a replacement or when ordering a new tube.

For details on the Dynamax "HD50" rotating anode x-ray tube, write: The Machlett Laboratories, Inc., Springdale, Connecticut 06879.



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C. ALLEN GOOD, M.D.

AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 101 DECEMBER, 1967 No. 4

# EVOLUTION IN THE TRAINING OF RADIOLOGISTS*

## THE PRESIDENT'S ADDRESS

*By* C. ALLEN GOOD, M.D.
ROCHESTER, MINNESOTA

MEDICAL education is in ferment. Not since 1910 when Abraham Flexner made his famous report has there been so much turmoil or so many different opinions about what is wrong and what to do about it. Faculties of medical schools are changing curriculums, the Citizens Commission on Graduate Medical Education (Millis Commission) advocates abolition of the internship, the Federal Government promotes the concept of regional medical programs, and certain specialty boards establish procedures for recertification of diplomates after a number of years of practice.

Our own specialty is affected, too. The report of the National Advisory Committee on Radiation (NACOR) tells of the present and future scarcity of well-trained radiologists; full-fledged subspecialties arise within the framework of diagnostic radiology; and proponents are heard to advocate the complete separation of therapeutic radiology, and perhaps even nuclear medicine, from diagnostic radiology.

In such an atmosphere of confusion it seems wise to take stock, to review what has taken place in the past, and to try to set proper goals for the future. With this in mind I have chosen as my title for this inaugural address, "Evolution in the Training of Radiologists." I am fully aware that my understanding of the past is colored by personal experience, that my crystal gazing into the future is just that, and that my estimate of the present is not shared by all. Nevertheless, the license afforded me by my obligation to address you—and by a recent 60th birthday—gives me courage to philosophize and to submit one man's thoughts and opinions about our great specialty of radiology.

The science of radiology began in 1895 with Roentgen's discovery. Almost immediately uses for x rays were found by physicians and the literature of the time is replete with descriptions of medical applications. One had only to possess equipment to use it in his practice, and a few enterprising doctors of medicine, engineers, and scientists began to devote most of their

* Presented at the Sixty-eighth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., September 26–29, 1967.

From the Mayo Clinic and Mayo Foundation: Section of Roentgenology.

time to the study and treatment of disease using roentgen rays. The American Roentgen Ray Society was founded by some of these pioneers in 1900. Thus the specialty began.

Formal training in the science and art of radiology evolved slowly. Certain great teachers developed and aspiring young physicians went to them for instruction. A second generation of great teachers blossomed and their offspring began to populate the larger hospitals and teaching institutions of the country. By 1934, fully 18 years after the formation of the first specialty board (American Board of Ophthalmology, 1916), radiology came of age with the formation of the American Board of Radiology. Its avowed purposes were "to improve the quality of graduate education in Radiology, to encourage the study of and to elevate the standards of training in Radiology, to determine the competence of specialists in Radiology and to arrange, control and conduct investigations and examinations to test the qualifications of *voluntary** candidates for certificates to be issued by the Board, to grant and issue certificates in the field of Radiology or of a branch thereof to *voluntary** applicants who have been found qualified by the Board and to maintain a registry of holders of such certificates, and to serve the public, physicians, hospitals and medical schools by preparing and furnishing lists of practitioners who shall have been certified by the Board."

Two items stand out in this list of purposes: candidates for examination and certification came to the Board *voluntarily*, and certification of competence was given those physicians who met *minimal* requirements. Such is still the case today.

B. R. Kirklin, the first and longtime secretary of the American Board of Radiology, devoted his Caldwell Lecture before the American Roentgen Ray Society in 1948 to "Graduate Education in Radiology." He traced the development of radiologic training up ... told of the influence that the Boar... on training programs in general. In his preamble he made a statement so true regarding graduate education that it should be quoted here and thoughtfully considered by all present teachers of radiology. He said, "I shall deal only with those [problems] related to the present training program for prospective radiologists. I have chosen this subject because *our program is neither perfect nor permanent** but subject to change as increasing knowledge and experience warrant, and *any such change can be brought about only with the advice and consent of the organized radiologists of America.*"*

In 1948, at the time of Kirklin's lecture, the Board required 3 years of special training in radiology in a program approved by the Board and by the Council on Medical Education and Hospitals of the American Medical Association. This joint approval was, and still is, the function of the Residency Review Committee for Radiology. This committee consists of the four officers of the Board and four radiologists appointed by the Council on Medical Education. Every teaching program in the United States is reviewed every 3 years to determine if there are sufficient and properly motivated teachers, if there are sufficient patients with a broad enough spectrum of disease, if space and equipment permit unhurried thoughtful work, and if the service load does not overshadow the opportunity for instruction and learning.

In 1948, also, the examination carried out by the Board consisted of oral interviews of 30 minutes each with six examiners. Three asked questions related to diagnostic roentgenology, two examined in the therapeutic applications of roentgen rays and radium, and one tested the candidate's knowledge of radiation physics.

In the almost 20 years since 1948 many changes have taken place, as Kirklin predicted. Lately candidates have been required to have experience with and knowledge of radioactive isotopes. This require-

* Italics supplied by President Good (Editor).

ment has necessitated changes in all training programs and a seventh examiner for all candidates. At first the applicant for certification could choose to omit this portion of the examination if he had received insufficient training. Since June 1963, however, this experience and this portion of the examination have been mandatory.

Although the requirement of 3 years of special training in radiology has not changed, since July 1, 1960, a fourth year of either training or practice has been required before a trainee can be admitted to the examination.

For the examination in December 1959 an eighth examiner was added. His purpose was to test the candidate's knowledge of the special procedures used in neuroradiology and cardiovascular radiology. For the first several examinations thereafter the eighth examiner graded the student's performance, but these grades did not enter into the final score. When it became apparent that program directors had supplied more opportunity to learn about and have experience in these special procedures, the performance with this particular examiner entered into the decision of whether or not to award a certificate.

Two years ago a ninth examiner was added. Fields of examination in diagnostic radiology were redefined and the special procedures applicable to each field became the responsibility of the examiner in that field. This was done to assist the candidate; he would no longer have all his special procedure "eggs in one basket," so to speak.

Now the Board has announced a written examination to be given first in June 1968. All applicants for certification in radiology who are first eligible to take the oral examination in December 1968 or thereafter will be required to receive a passing grade on the written examination before being admitted to the oral examination. Questions will be of the multiple-choice type and will cover anatomy, physiology, and pathology as applied to radiology. Other questions will be on radiation physics, radiobiology, and nuclear medicine. It is hoped that the written examination will be more objective for the subjects covered and that it may replace specific oral examinations in physics, nuclear medicine, and radiobiology. Certainly the candidate's fate in any part of the examination will not depend on his ability to answer two or three specific questions only but will depend on his general knowledge of the subject and his ability to answer 60 or more of 100 multiple-choice questions.

A variety of changes have taken place in training programs. Although the amount of training required by the Board is still 3 years, many program directors have increased the training period to $3\frac{1}{2}$ or 4 years, and in at least one instance, to 5 years. For a certificate in radiology the candidate must spend a minimum of 2 years studying diagnostic radiology and 1 year in therapeutic radiology. Today some programs are designed so that the candidate is trained only in diagnosis or only in therapy. Kirklin predicted this step in his Caldwell Lecture in 1948 when he said, " . . . the division of radiology into two independent specialties, diagnostic roentgenology and therapeutic radiology, is being advocated. This may occur eventually, for it is a normal evolutionary and progressive step." In 1943 he believed such a division to be premature.

Today there are some who advocate the division of radiology into three rather than two separate specialties. They would prefer separate training programs and separate examinations in diagnostic radiology, in therapeutic radiology, and in nuclear radiology.

Other problems under discussion wherever radiologists gather are the lack of governmental funds available for research in diagnostic radiology, the lack of exposure of medical students to our discipline early in their training, the resulting difficulty in recruiting superior graduates to our specialty, and the burgeoning service load that sometimes almost forces good

teachers to abandon teaching.

Such are the changes and the challenges. The changes have been evolutionary not revolutionary. We must not permit the challenges to transform evolution into revolution.

After many years of service as an examiner for the American Board of Radiology, I can say, without reservation, that today young radiologists who are ready for certification are much better trained and much more knowledgeable in all phases of our specialty than they were 20, yes, even 10 years ago. This redounds to the credit of the many thoughtful program directors who plan and carry out training in radiology in schools and hospitals throughout our country. It is especially true when one considers that the amount of available knowledge and information doubles every 10 years.

So, what lies ahead? What further evolution is needed in the training of radiologists? Here are my own opinions. They are necessarily colored by my own background and experience. Many of you will disagree with some or even all of my ideas. No doubt some of these thoughts will not stand the test of time, and no doubt time will force me to change some of my beliefs. But permit me to gaze into the crystal!

In the first place, I am firm in my conviction that the radiologist is here to stay. Specialists in other disciplines can acquire knowledge and skill in radiologic procedures useful in their practice, but the more time they spend performing these tasks, the more they become radiologists in a very limited field. The practice of medicine today depends on the broad outlook and the skill of the well-trained radiologist whose daily activities place him in contact with patients of the internist, the surgeon, the orthopedist, the neurologist, the gynecologist, and the pediatrician.

But the growth of scientific and medical knowledge makes it impossible for any one person to learn all about everything. Therefore, some specialization within our field is necessary. The first division has already begun and it should be encouraged. Ultimately, and we should do all in our power to assist, there should be training for radiologists who will devote full time to the problems of diagnosis and different training to produce superior therapists. Separate certificates should be issued by the Board after the candidate satisfies separate training requirements and passes a separate examination. However, there should be nothing to prevent the zealous student from acquiring both certificates. I am not even opposed to the idea of separate training and separate examination in the field of nuclear radiology.

Somewhat farther into the future I see a need for certification in subspecialties of diagnostic radiology. This will require definition of fields and minimal training requirements, a subspecialty examination, and a subspecialty certificate. Such subspecialty training and certification should be available only to a person who holds a certificate in diagnostic radiology.

With all of these ideas in mind, How should we organize our training programs? How can we best supply the needs of all candidates with their wide variety of goals and ambitions? I should like to propose the framework for such a plan. Many of you will be able to see loopholes in it, but perhaps they will be loopholes that can be plugged. In any event, I hope many of you will give it careful consideration.

First, I would do away with the requirement of a year of internship of the types available today and would substitute a year of training in radiology. I would call this an "internship in radiology" and would plan it so that the medical graduate would obtain experience in diagnosis, therapy, and the use of radioactive materials. The purpose of this year of internship would be to acquaint the intern with all phases of radiology, to allow him time to decide whether or not he wants to continue in radiology and, if he does, to choose his field for future training.

Such an internship should be open to all students, even those who are not com-

mitted to radiology. I can even envisage the student dedicated to some subspecialty in internal medicine choosing such an internship to broaden his knowledge and understanding of his future endeavors. Some of these candidates might be recruited to radiology by the very fascination of the work during the year. More medical graduates might be willing to spend a year investigating radiology without committing themselves to a full 3 years of residency training. An attractive radiologic internship might be one of our better recruiting devices.

Residency training would follow the year of internship. It would be offered in diagnostic radiology, therapeutic radiology, and perhaps even in nuclear radiology. Residents would be selected from the graduating intern class. Undesirables would be weeded out, something that few program directors are willing to do now at the completion of a year of residency training.

With the background of experience obtained during the internship year, residency training could be complete after 2 or $2\frac{1}{2}$ more years. Examination and certification should take place at that time.

The person interested in a subspecialty could pursue his training in anticipation of another examination before obtaining the subspecialty certificate. Furthermore, the individual interested in obtaining certification in both diagnosis and therapy could begin training in the other field. This might be the preferred course of study for students interested in practicing radiology in a community hospital. Certainly these physicians would be better prepared than are those radiologists today who are certified in radiology. Also, the time spent after graduation from medical school to certification would be 5 years—the same as is required today.

It should be emphasized that the training requirements I have outlined are *minimal.* Nothing should be allowed to discourage program directors from offering and encouraging further training and experience.

A Board certificate in radiology or one of its branches is not the end of the trail. A conscientious radiologist continues to learn throughout his medical life. He does so by reading, by attending meetings of this society and other similar societies, and by enrolling in courses of postgraduate education. Such continuing education should be encouraged. Several methods are available to stimulate radiologists to study further. The American Academy of General Practice demands that its members attend meetings or take courses of study for a minimal number of hours each year. Certain specialty boards are experimenting with a recertification examination to be taken after the physician has been in specialty practice for a specified number of years. The Quality Evaluation Program of the American College of Radiology, although directed primarily at a department and not an individual radiologist, may act as a stimulus to additional education.

Whatever method or methods are used, opportunities should be readily available for all practicing radiologists to keep up to date. Most large teaching institutions offer "continuation courses." Others should be added, and all should be coordinated in order to eliminate too much duplication and to ensure proper geographic distribution. Courses offered by the Section of Instruction of this Society and by the Radiological Society of North America should be imitated by other radiologic organizations or sponsored in association with meetings of state and national medical societies. Such courses should be taught by a stimulating faculty offering wide opportunity for the students to ask questions and receive answers either in group or individual discussions.

Approval of courses for continuing education, of programs of scientific societies, and of recertification procedures should be in the hands of radiologists. Various organizations exist today that could share these responsibilities. I refer to the American Board of Radiology, the Residency Review Committee for Radiology, the

Commission on Education of the American College of Radiology, the Committee on Education and Training of the American Club of Therapeutic Radiologists, and others. It must be recognized that default of these responsibilities by radiologists will cause governmental agencies to assume the obligation. This we must avoid, if we can.

Evolution is defined by Webster as "an unfolding; a process of opening out what is contained or implied in something; a development leading to a definite end." Revolution is defined as "a total or radical change." I hope you will agree with me that the thoughts expressed are evolutionary. The various pieces of the future structure of our specialty are currently available. They require little reorientation to evolve into a better method of education for more competent radiologists. Certainly that is our goal, just as it was the goal of the founders of the American Board of Radiology 33 years ago. Let us all strive continually toward its attainment.

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# THE FORAMINA OF THE MIDDLE FOSSA: A PHYLOGENETIC, ANATOMIC AND PATHOLOGIC STUDY*

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DURING the early development of the human fetal skull, prior to the twelfth week of gestation, three principal fenestrations are present at the base through which pass cranial nerves and blood vessels. These openings, which have their counterpart in the primitive mammalian skull,[6] have been termed:

(1) *Foramen lacerum anterius.* This hiatus between the orbitosphenoid (lesser wing) and alisphenoid (greater wing) persists as the sphenoidal fissure for exit of cranial nerves III, IV, VI and the ophthalmic division of V.

(2) *Foramen lacerum medius.* The opening between the basisphenoid and periotic capsule is variably subdivided in different mammals. It persists in man as the foramen lacerum, with compartmentation into a medial carotid canal and a lateral petrosphenoidal fissure for the greater superficial petrosal nerve.

(3) *Foramen lacerum posterius.* The hiatus between the basiocciput and the auditory bullae persists as the jugular foramen for transmission of cranial nerves IX, X, and XI.

These large fenestrations become subdivided by the ingrowth of bony spicules, leading to the formation of multiple discrete foramina in the floor of the human skull. Failure of these bony spurs to develop fully is responsible for various foraminal abnormalities. The authors have studied an ascending group of mammals including subhuman primates in an effort to clarify the morphogenesis of foraminal anomalies at the base of the skull which seem to have a phylogenetic relationship.

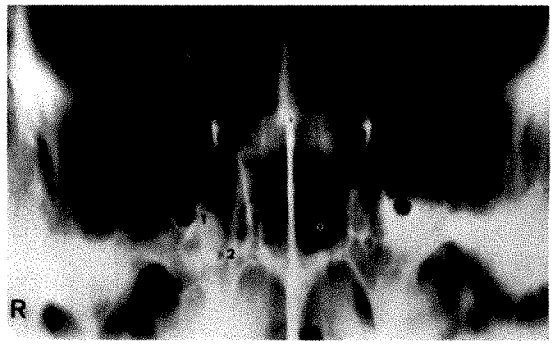



FIG. 1. Tomogram in the anteroposterior projection demonstrating the position of the foramen rotundum (1) in relation to the floor of the orbit. Note also the pterygoid canal (2) and the optic foramen (3).

## ANATOMIC CONSIDERATIONS AND CONGENITAL VARIANTS

The *foramen rotundum* is a short horizontal canal in the anteromedial portion of the greater sphenoidal wing which transmits the maxillary division of the trigeminal nerve and small emissary veins to the pterygopalatine fossa. The foramen lies directly below the medial end of the superior orbital fissure and is intimately related to the lateral wall of the sphenoid sinus. Its size, according to Lindblom,[11] may vary roentgenographically from 3×3 mm. to 4×5 mm. Since the foramen forms an angle of some 70 degrees with the plane of the skull base, it is not demonstrated in the routine submentovertical projection. However, it may be visualized just below the medial aspect of the orbital floor in the Caldwell or Waters projections, and in anteroposterior tomograms (Fig. 1). That significant congenital asymmetry of the foramen rotundum does occur, albeit uncommonly, should be borne in mind when

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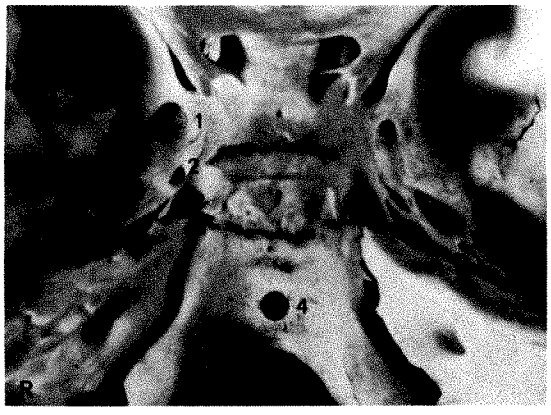



FIG. 2. Photograph of interior of skull of a 6 year old child showing striking asymmetry of the foramen rotundum. The right foramen rotundum (1) is much larger than its left counterpart. Note also the asymmetry of the sphenoidal emissary foramen (2) and the foramen ovale (3). The round hole in the clivus (4) is artifactual.

an enlarged foramen is encountered (Fig. 2). Taveras and Wood[19] cite an instance of unilateral enlargement of the foramen rotundum in a patient, with symptoms compatible with those of a lesion of the maxillary nerve, in whom surgical exploration revealed no tumor or other cause for increased foraminal size. Anomalous enlargement of the foramen may be distinguished from pathologic erosion by the presence of well-defined cortical margins. Rarely, the foramen may communicate with the superior orbital fissure as illustrated by LeDouble[12] (Fig. 3). This developmental variant results from incomplete ossification of the chondral root of the greater sphenoidal wing and failure of its anterior portion to fuse with the postsphenoid. In normal human embryogenesis, the foramen rotundum can be identified as a discrete orifice separate from the superior orbital fissure by the twelfth to the sixteenth week of fetal life[8] (Fig. 4). Before this, the foramen rotundum and the superior orbital fissure exist as a confluent orifice (foramen lacerum anterius). This conjoint relationship is present in certain mammals; *i.e.*, carnivores (seal and walrus) (Fig. 5), rodents (beaver and porcupine) and some ruminants.[12] In these species, the foramen ovale is often larger in comparison to the optic foramen than is the case in man. This is presumably related to the greater development of the muscles of mastication and their neural innervation in these animals.

The *sphenoidal emissary foramen* (foramen Vesalii) is a small, inconstant aperture in the greater sphenoidal wing, slightly anterior and medial to the foramen ovale (Fig. 6). The canal runs inferiorly to open extracranially near the scaphoid fossa. The foramen transmits a small vein which connects the cavernous sinus with the pterygoid venous plexus. In Boyd's study[1] of human and anthropoid skulls, the foramen was present bilaterally in approximately 15 per cent, unilaterally in 21 per cent, and totally absent in 64 per cent. In the authors' material, when the sphenoidal emissary vein was present on one side only, the

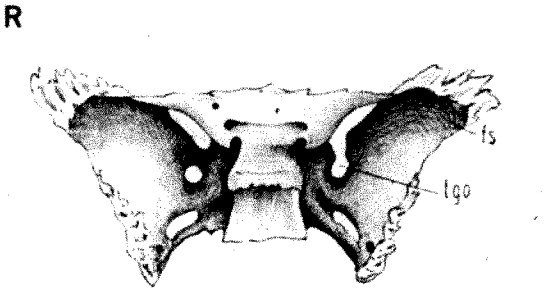



FIG. 3. Photograph of line drawing from LeDouble[12] illustrating communication of the left foramen rotundum with the sphenoidal fissure.




FIG. 4. Roentgenogram of human fetal sphenoid bone (18–20 weeks gestation) showing a discrete foramen rotundum (1) separated from the superior orbital fissure (2) by the optic strut (arrow). Note also the optic foramen (3), the lesser sphenoidal wing (4) and the greater sphenoidal wing (5).

ipsilateral foramen ovale was frequently smaller than its homologue on the opposite side.

The *canaliculus innominatus* (canal of Arnold) is an inconstant minute aperture posterior to the foramen ovale and medial to the foramen spinosum (Fig. 7). When present, it accommodates the lesser superficial petrosal nerve which transmits fibers from the facial and glossopharyngeal nerves to the otic ganglion. In the absence of a canaliculus innominatus, the lesser superficial petrosal nerve escapes from the skull through the foramen ovale.

The *foramen for the ophthalmomeningeal vein* (of Hyrtl) is infrequently encountered both in man and in subhuman primates, *e.g.* gorilla (Fig. 8). When present, the foramen is situated in the greater sphenoidal wing, usually in the lateral half of the orbit (Fig. 9). The foramen may occur on one or both sides and vary slightly in size. The ophthalmomeningeal vein which enters the foramen connects the orbital with the cerebral veins and commonly drains into the sphenoparietal sinus; the latter anastomoses with the anterior portion of the cavernous sinus. The foramen is not to be confused roentgenographically with an ectopic ethmoidal cell which may rarely be present in the greater sphenoidal wing. The misplaced ethmoidal cell is apt to be larger and is distinguished by the sharply defined mucoperiosteal border characteristic of pneumatized paranasal sinus cellules.

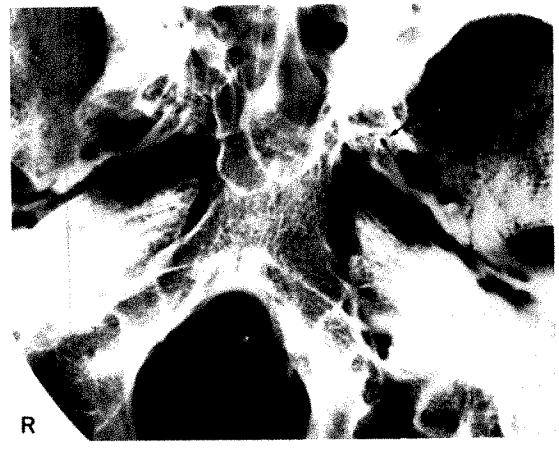



Fig. 6. Roentgenogram of skull in a slightly rotated submentovertical projection showing a sphenoidal emissary foramen (Vesalius) on the left (arrow). Note its typical location anterior to the foramen ovale.




Fig. 5. Tomogram of the left orbit of a walrus in a modified optic foramen projection demonstrating a prominent superior orbital fissure (1) extending back to the region of the pterygoid plate. Note the failure to visualize a separate foramen rotundum because the latter is confluent with the superior orbital fissure. The optic foramen (2) is visualized above and medial to the superior orbital fissure.

The *foramen ovale* is commonly an oval




Fig. 7. Canaliculus innominatus (arrow) on the left side in characteristic location behind the foramen ovale.

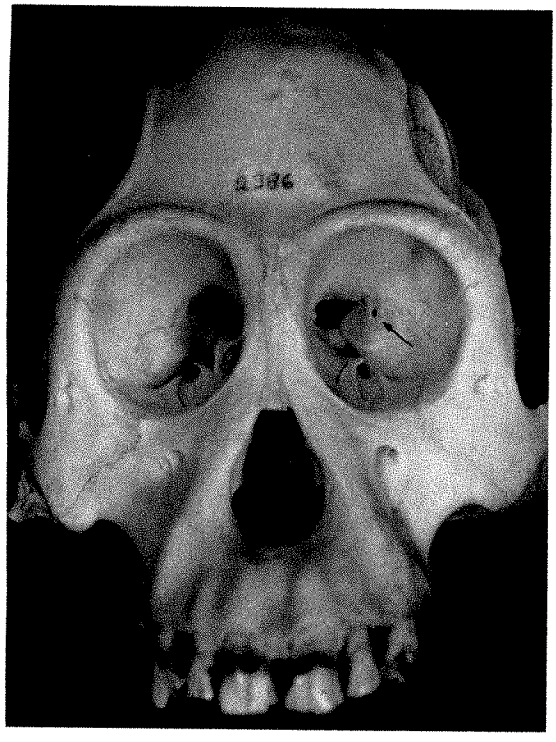


FIG. 8. Photograph of skull of a gorilla showing a foramen for the ophthalmomeningeal vein in the left orbit (arrow).

shaped hole in the greater sphenoidal wing slightly lateral and posterior to the foramen rotundum. Its external opening lies posterior to the root of the pterygoid process and anteromedial to the sphenoidal spine. The foramen transmits the mandibular branch of the trigeminal nerve, the accessory meningeal artery, and, in the absence of a canaliculus innominatus, the lesser superficial petrosal nerve. When the sphenoidal emissary foramen (Vesalii) is not present, the foramen ovale also transmits small veins connecting the cavernous sinus with the pterygoid plexus. The foramen is best visualized in the submentovertical projection. According to Lindblom,[14] its average roentgenographic dimensions in this view are 5×8 mm., ranging from 3×5 mm. to 7×11 mm.

In lower mammals, the foramen ovale and the foramen spinosum coalesce in the foramen lacerum medius[21] and are not differentiated as discrete foramina; the mandibular division of the trigeminal nerve passes through the petrosphenoidal fissure lateral to the carotid artery. In higher mammalian forms, a shallow groove for the nerve forms in the posteromedial aspect of the greater sphenoidal wing. The groove deepens into a notch which ultimately becomes a foramen by the ingrowth of bone

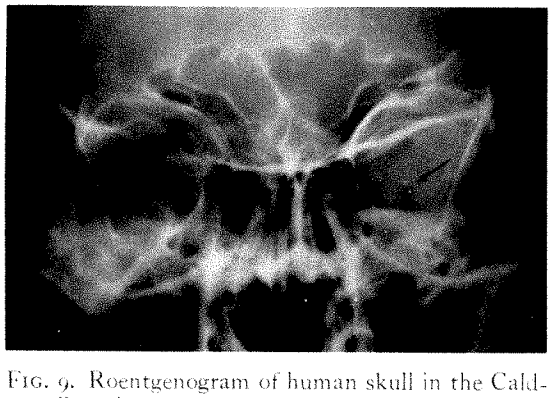


FIG. 9. Roentgenogram of human skull in the Caldwell projection demonstrating a foramen for the ophthalmomeningeal vein in the left greater sphenoidal wing (arrow).

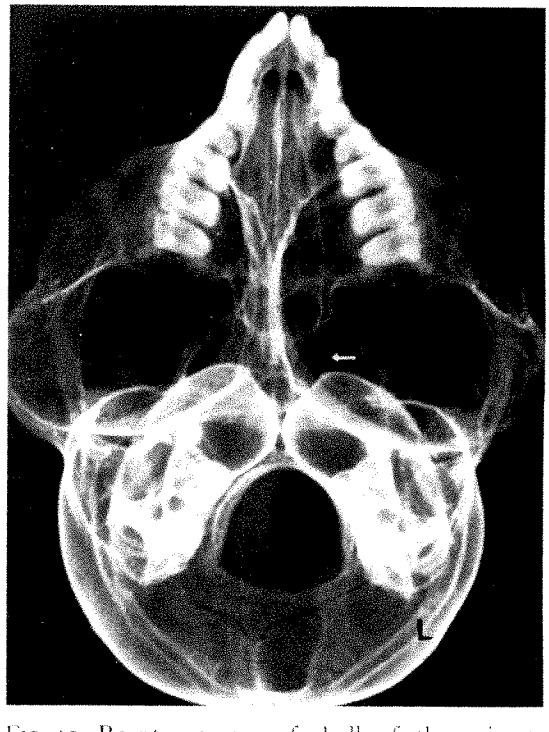


FIG. 10. Roentgenogram of skull of the primate tarsius in the submentovertical projection. Note the persistent foramen lacerum medius (arrow) and absence of a separate foramen ovale in this primate.

around the nerve. These transitional phases are well demonstrated in various primates.[6] Thus, a foramen ovale is not present in tarsius, a primitive primate, being represented within the lacuna of the foramen lacerum medius (Fig. 10). In the new world monkeys, the foramen ovale may be found as a deep groove, notch or incomplete foramen. In the orangutan and chimpanzee, the foramen is situated at the posterior margin of the greater sphenoidal wing, opening into the petrosphenoidal fissure (Fig. 11, *A* and *B*). In the gorilla, the foramen has a more lateral position close to the sphenosquamosal suture.

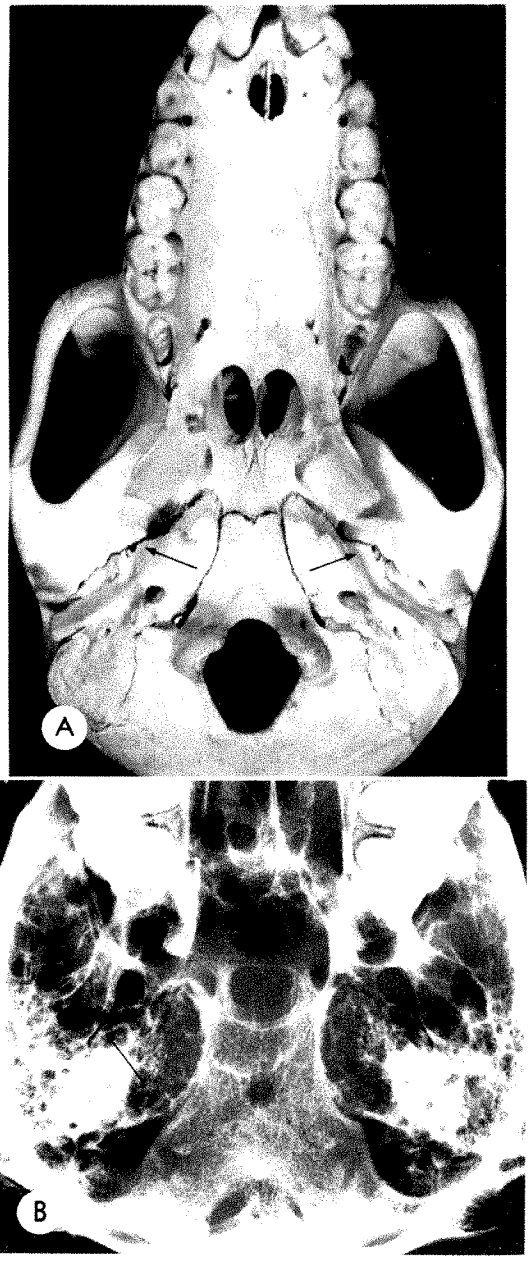



FIG. 11. Photograph (*A*) and roentgenogram (*B*) of chimpanzee skull in the submentovertical projection demonstrating the posterior position of the foramina ovale and spinosum opening into the petrosphenoidal fissure (arrows).




FIG. 12. Roentgenogram of adult male skull in the submentovertical projection showing bilateral persistence of the primitive foramen lacerum medius.

It is evident that this phylogenetic background may explain the wide range of variation found in the human foramen ovale.

(1) The foramen may not be present as a separate orifice. Under these circumstances, the primitive large foramen lacerum medius seen in tarsius is retained (Fig. 12).

(2) The foramen may be incompletely formed, its posterior margin represented by a thin spicule of bone partially separating the foramen ovale from the foramen lacerum medius (Fig. 13). In some instances, the foramen ovale, although completely separate from the foramen lacerum, may communicate with the latter through a suture line. The foramen ovale may communicate, at times, with the foramen spinosum by a narrow aperture or actually be confluent with the foramen spinosum (Fig. 14). On occasion, the foramen ovale may coalesce anteromedially with the sphenoidal emissary foramen.

(3) The location of the foramen ovale




FIG. 13. Roentgenogram of newborn skull illustrating an incomplete foramen lacerum medius on the left and more complete partition on the right.

may vary. When its posterior bony margin is unusually broad, the foramen is situated more anteriorly in the greater sphenoidal wing. On the other hand, it may have a somewhat more posterior location. Rarely, the foramen may be present along the lateral margin of the greater sphenodial wing near the sphenosquamosal suture, as in the gorilla (Fig. 15).

(4) The foramen may vary considerably in size and shape on one or both sides (Fig. 16). Thus, the foramen may be oval, round or pyriform. These variations may be influenced by differences in osteogenesis and by the venous channels passing through the foramen. According to Wood-Jones,[21] the foramen lacerum medius in the primitive mammal transmits venous channels which connect the cavernous sinus with the extracranial pterygoid plexus. With ascending development, these anastomotic veins tend to be included in the foramen ovale. Rarely, the foramen may be partially or completely subdivided by bony spicules. In the latter instance, the anterior portion of the foramen ovale may be considered to be the sphenoidal emissary foramen (Vesalii). In this circumstance, the sphenoidal emissary foramen is a fairly prominent channel anteromedial to a small, more rounded foramen ovale.

The *foramen spinosum* is a small orifice in the greater sphenoidal wing close to the sphenoidal spine, directly posterior and lateral to the foramen ovale. It transmits the middle meningeal vessels and a recurrent branch of the mandibular nerve. As in the case of the foramen ovale, the foramen spinosum in man is subject to a number of variations. These anatomic variants are related either to incomplete osteogenesis or to aberrant development of the middle meningeal artery.[10]

(1) The foramen may be incompletely

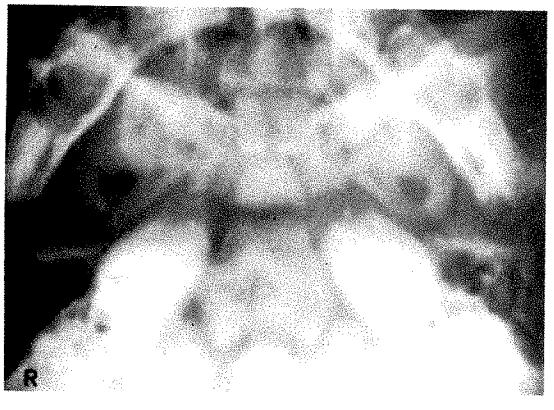



FIG. 14. Roentgenogram of skull in a newborn female showing bilateral communication of the foramina ovale and spinosum by a narrow channel.




FIG. 15. Lateral position of the foramen ovale close to the sphenosquamosal suture (arrow) in a stillborn phocomelic monster with anomalous development of the sphenoid bone. The lateral locus of the foramen ovale represents the normal position in the gorilla.




Fig. 16. Roentgenogram of dry skull in the submentovertical projection showing asymmetry of the foramen ovale. The left foramen is larger and rounder than the right.

separated from, or remain confluent with, the foramen ovale (Fig. 14). This anomaly is an expression of incomplete osteogenesis with deficient ingrowth of bony spurs rather than atypical vascular development.[10,17]

(2) When a complete foramen lacerum medius persists in man, the foramen spinosum is not defined as a discrete orifice and may be represented by a slight notch marking the site of entrance of the middle meningeal artery into the cranium. In the case of a partial foramen lacerum medius, the latter may communicate either with the foramen spinosum or with the foramen ovale.

(3) The foramen spinosum may communicate with the canaliculus innominatus.[21]

(4) The foramen spinosum may occupy a more posterior position and abut against the anterior margin of the petrous bone or open into the petrosphenoidal fissure (Fig. 17; and Fig. 11).

(5) The foramen spinosum may be asymmetric (Fig. 18).

(6) Rarely, the foramen may be absent in man in the presence of a normal foramen ovale, an occurrence apt to reflect anomalous formation of the middle meningeal artery[5,9,10,18] (Fig.




Fig. 18. Asymmetry of the foramen spinosum (left larger than right) in a normal adult male. The foramen ovale is also asymmetric.




Fig. 17. Photograph of the exocranial surface of the base of a human skull showing the foramen spinosum leading into the petrosphenoidal fissure by a groove (bold arrow). Note also the notch for the accessory meningeal artery in the foramen ovale (thin arrow); foramen ovale 1, foramen spinosum 2, carotid canal 3, jugular foramen 4.




Fig. 19. Absence of left foramen spinosum (arrow) in a patient with marked pneumatization of the paranasal sinuses and petrous bone. Multiple submentovertical projections in varying degrees of extension failed to visualize a foramen spinosum on the left side.




FIG. 20. Roentgenogram of dry human skull demonstrating spurious absence of the right foramen spinosum with the head and neck hyperextended (*A*). The foramen is clearly demonstrated in the usual projection (*B*).

19). At times, it may be necessary to utilize multiple submentovertical projections with varying degrees of tube angulation to exclude spurious absence of a small, obliquely coursing foramen spinosum (Fig. 20, *A* and *B*).

In the 15 mm. human embryo, the intermediate segment of the internal carotid artery is part of the dorsal aorta between the first and third aortic arches. Cephalad to the first aortic arch, the internal carotid artery gives off the ophthalmic artery. The dorsal portion of the second aortic arch becomes the stapedial ar-

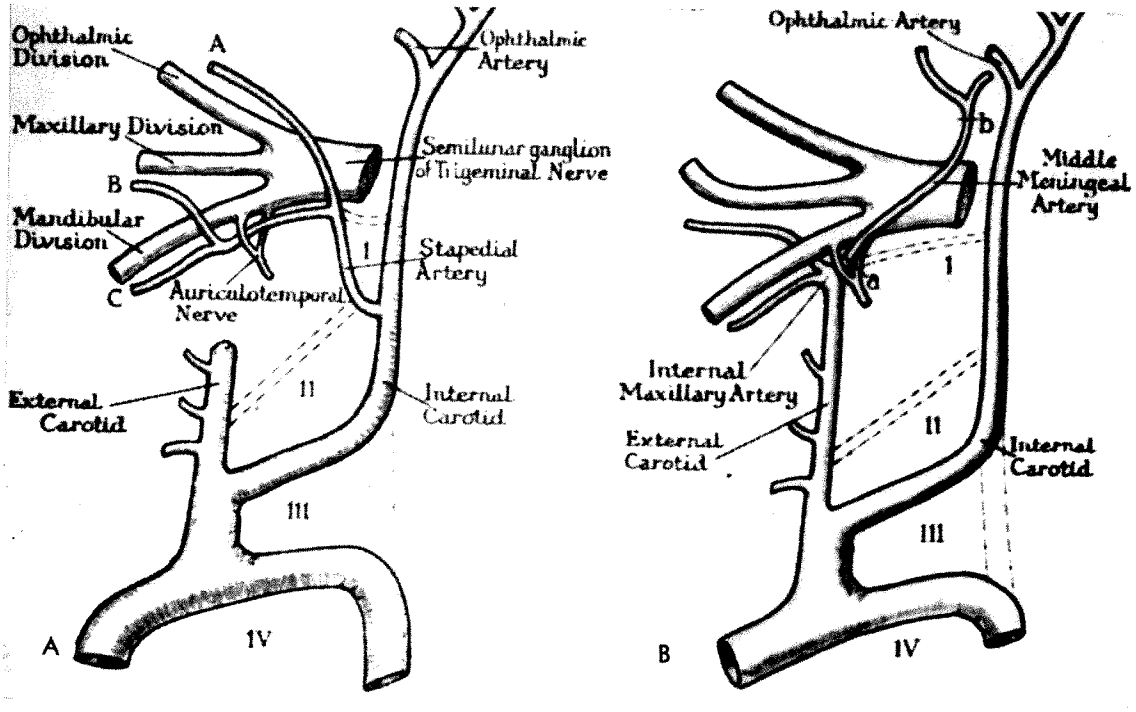



FIG. 21. (*A* and *B*) Diagrams showing the development of the stapedial artery and the formation of the middle meningeal artery from the descending branch of the stapedial artery (from Greig,[19] after Tandler-Fisher). I.II.III.IV = Aortic arches. A = Supraorbital division of the stapedial artery. B = Infraorbital division of the stapedial artery. C = Mandibular division from the descending trunk of the stapedial artery. When the foramen spinosum is truly absent, the segment of artery between *a* and *b* fails to develop.

tery. The latter, in turn, divides into supraorbital and descending branches. With further development, these branches lose their connections to the internal carotid artery and unite with the external carotid artery to form the internal maxillary artery. Thus, the middle meningeal artery is derived from the descending trunk of the stapedial artery. With excessive obliteration of the proximal part of the stapedial artery, the future middle meningeal artery fails to unite with the future internal maxillary artery and retains connections with the ophthalmic branch of the internal carotid artery (Fig. 21, *A* and *B*). From a comparative anatomic standpoint, it is of interest to note that the middle meningeal artery enters the cranial cavity by way of the foramen ovale in the sheep and ox[15] (Fig. 22; and 23), whereas in the horse, the middle meningeal artery enters through the foramen lacerum (Fig. 24).

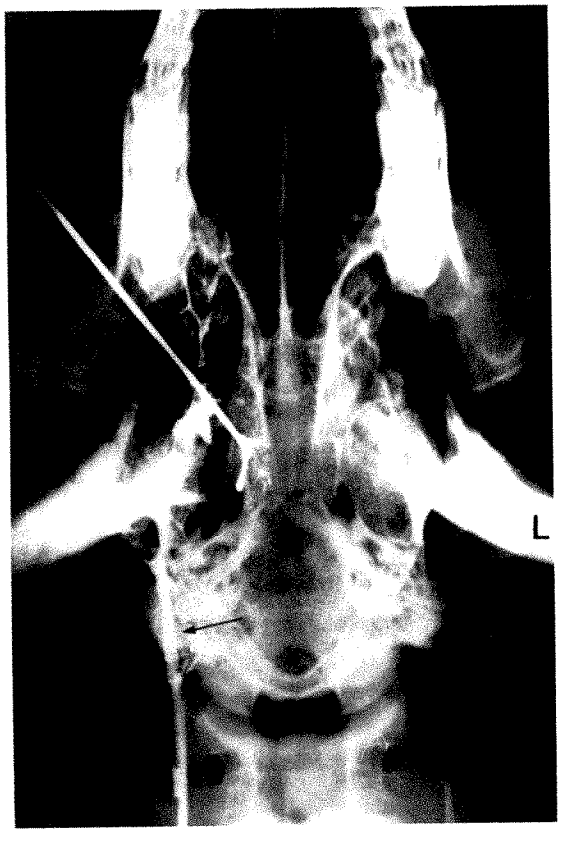



Fig. 22. Roentgenogram of skull of ewe in the submentovertical projection showing the tip of a metallic probe in the right foramen ovale. Note also the catheter (arrow) in the right internal maxillary artery.




Fig. 23. Polaroid roentgenogram of the skull of the same ewe following injection of 50 per cent hypaque into a catheter in the right internal maxillary artery. The arrow points to the middle meningeal artery arising from the internal maxillary artery, passing medially to enter the cranial cavity through the foramen ovale.

Not uncommonly, the fascia separating the internal and external pterygoid muscles may calcify or ossify, giving rise to pterygospinous and pterygoalar bars. The location of the calcification in the connective tissue partition determines whether a spinous or alar or both bars are formed. The *pterygospinous* bar attaches anteriorly to the spine of Civinini (a small spur near the root of the posterior margin of the lateral pterygoid plate) and posteriorly to the sphenoidal spine, *medial to the foramen spinosum* (Fig. 25; 26; and 27). The space en-

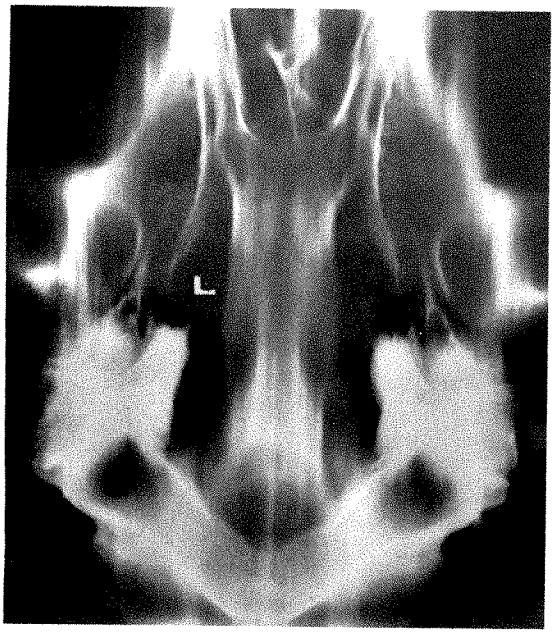



FIG. 24. Roentgenogram of equine skull in the submentovertical projection demonstrating the large foramen lacerum (L) through which the middle meningeal artery enters the cranial cavity.

closed by the pterygospinous bar, *i.e.* the pterygospinous foramen, is traversed by the pterygoid vessels and branches of the mandibular nerve supplying the internal pterygoid muscle. The *pterygoalar bar*[2,3,4] attaches anteriorly to the root of the lateral pterygoid plate close to the attachment of the pterygospinous bar, and posteriorly to the undersurface of the greater




FIG. 25. Photograph of the posteroinferior aspect of an isolated human sphenoid bone with metallic pins placed in the position of the pterygospinous (thin arrow) and pterygoalar (heavy arrow) bars. Spine of Civinini (arrowhead), foramen ovale (1), foramen spinosum (2). Note the more medial position of the pterygospinous bar.

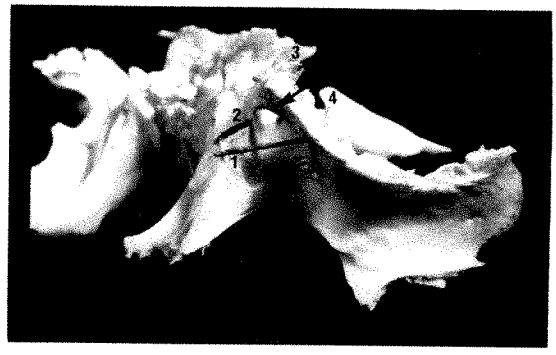



FIG. 26. Oblique-lateral photograph of an isolated human sphenoid bone seen from above with a metallic pin in the position of the pterygoalar bar (1) lateral to the foramen ovale (arrow). Note the more medial location of the pin in the position of the pterygospinous bar (2). The dorsum sellae is identified as 3, and the anterior clinoid process as 4.

sphenoidal wing *lateral to the foramen spinosum* (Fig. 28). The space enclosed by the pterygoalar bar, *i.e.* the pterygoalar foramen, was originally termed the porus crotaphiticobuccinatorius by Hyrtl[11] in 1862. This space provides passage for the major motor branches of the mandibular nerve supplying the muscles of mastication. A prominent lateral pterygoid plate may simulate a pterygoalar bar but this difficulty can readily be resolved by tomography (Fig. 29).

Since the pterygospinous bar has a more medial course, it does not compromise the foramen ovale, and therefore, has no clinical significance. On the other hand, the

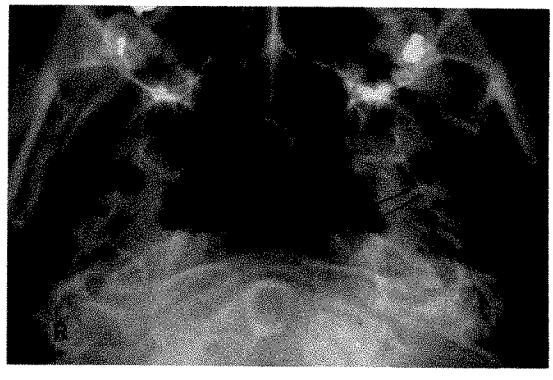


FIG. 27. Unilateral pterygospinous bar (left). Note the characteristic insertion medial to the foramen spinosum (arrows).

pterygoalar bar may interfere with injection of the mandibular division of the trigeminal nerve by way of the lateral approach. In Priman and Etter's[16] material, complete ossification of the pterygospinous and pterygoalar ligaments occurred in approximately 3 per cent of skulls. On the other hand, partial ossification of the pterygospinous ligament was noted in 8 per cent and of the pterygoalar ligament in 14.4 per cent of the same material. In the authors' experience, calcification or ossification of either ligament may be unilateral or bilateral.




Fig. 29. Simulation of pterygoalar bar by a prominent right lateral pterygoid lamina (arrow), accentuated by hyperextension of the skull.

## PATHOLOGIC CHANGES

A number of pathologic processes involving the base of the skull may alter the size and shape of the foramina of the middle fossa. In general, the lesions may be divided into two broad categories: I. diseases which destroy or erode bone, thereby enlarging the foramina; and II. diseases characterized by increased bone formation which may result in foraminal constriction.

### I. DISEASES WHICH MAY PRODUCE FORAMINAL ENLARGEMENT

A. *Tumors*

1. *Primary intracranial.*

a. Meningioma. Since these tumors receive the major portion of their arterial supply from the external carotid system, the middle meningeal artery frequently undergoes considerable hypertrophy. In these circumstances, the foramen spinosum may occasionally be enlarged. Since the foramen spinosum may exhibit normal asymmetry, unilateral enlargement of this foramen assumes greater significance in the presence of an enlarged middle meningeal vascular groove on the same side[20] (Fig. 3C).

Although meningiomas involving the floor of the middle fossa more commonly produce hyperostosis, they may, at times, destroy bone either by infiltration or erosion, thereby enlarging the neighboring foramina.

b. Neurogenic tumors. Neurolemmomas (Fig. 31) and malignant neural tu-

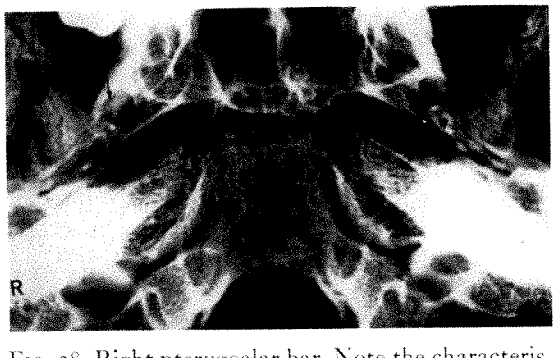



Fig. 28. Right pterygoalar bar. Note the characteristic attachment lateral to the foramen spinosum (arrow).

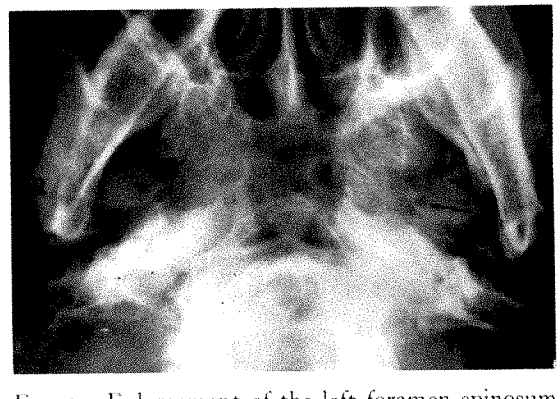


Fig. 30. Enlargement of the left foramen spinosum (arrow) together with a dilated, tortuous meningeal vascular groove (arrowhead) in a patient with a left pterional meningioma.

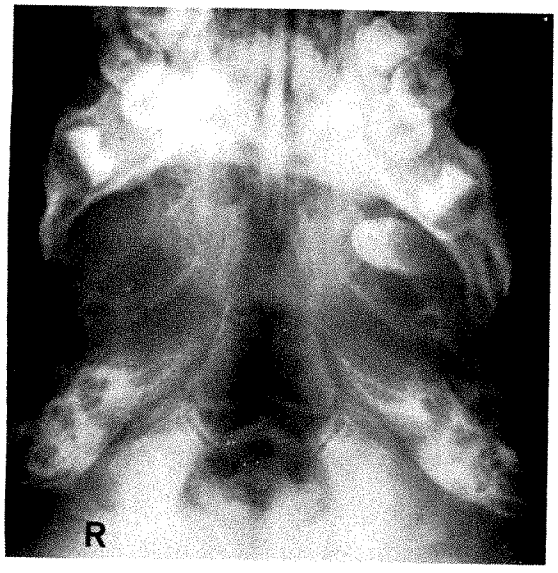



Fig. 31. Neurofibromatosis in a 3 year old boy with multiple neurolemmomas and optic glioma. Note the striking enlargement of the foramina ovale and spinosum on the right.

mors involving the trigeminal nerve in Meckel's cave (or a branch of the trigeminal nerve) (Fig. 32) may produce enlargement of the corresponding middle fossa foramina. While congenital absence of the greater sphenoidal wing may occur as an isolated calvarial defect in neurofibromatosis without cerebral involvement, the authors have not observed a grossly enlarged middle fossa foramen without an associated neural tumor in this disorder.

c. Epidermoid (cholesteatoma). Intraosseous or intracranial epidermoid tumors involving the floor of the middle fossa may erode bone and produce foraminal enlargement. This type of erosion is usually sharply defined and frequently exhibits a scalloped, sclerotic margin.

d. Temporal lobe glioma. Rarely, slowly growing astrocytomas of the temporal lobe may cause thinning of the floor of the middle fossa on the same side as the tumor, with loss of definition of the margins of one or more foramina. Usually, these tumors also produce secondary changes of the sella turcica, erosion and thinning of the ipsilateral sphenoidal wing, and widening of the sphenoidal fissure.

Rarely, long-standing increased intracranial pressure may produce unilateral or bilateral thinning of the floor of the middle fossa.[7] The changed bone has poorly defined margins which make it difficult to outline the walls of the foramina. Under these circumstances, characteristic secondary changes of the sella and/or sutural diastasis are usually present.

2. *Metastatic.* Destruction of the floor of the middle fossa with foraminal enlargement may result from blood-borne metastases from a remote focus (carcinoma, sarcoma, neuroblastoma) or by direct extension from malignant lesions of the nasopharynx (Fig. 33; and 34). In the latter instance, recognition of the soft tissue mass in the nasopharynx is the key to the correct diagnosis. Chemodectomas (glomus jugulare tumor) may destroy the floor of the middle fossa if they extend forward from the region of the jugular foramen or petrous bone. Bone destruction below and behind the petrous bone should suggest the possibility of a glomus jugulare tumor.




Fig. 32. Malignant neurolemmoma involving the right middle and posterior fossae. The tumor arose in Meckel's cave and extended into the posterior fossa. Tomogram clearly demonstrates the destructive changes involving the foramina of the right middle fossa.

B. *Vascular lesions*

1. *Aneurysm.* Aneurysms of the intracavernous portion of the internal carotid artery may erode the floor of the middle fossa (Fig. 35). Usually, the margins of the bone defect are sharply defined. Carotid arteriography provides conclusive evidence of the aneurysm.
2. *Vascular malformation.* Carotid-cavernous sinus fistulas may produce localized erosion of bone in the floor of the middle fossa on the side of the lesion. The appearance is indistinguishable from the erosion caused by an aneurysm.

C. *Trauma*

A linear fracture of the floor of the middle fossa may frequently be undetected on the roentgenogram. If the arachnoid herniates through an associated dural rent, pulsation of cerebrospinal fluid in the subarachnoid space may cause localized erosion of bone over a period of time. The bone defect usually has a smooth, well-defined periphery that may be scalloped.

D. *Reticuloendothelioses*

Primary diseases of the reticuloendothelial system, particularly histiocytosis X and eosinophilic granuloma, may be responsible for a localized area of bone destruction in the floor of the middle fossa (Fig. 36, *A* and *B*). The margins of the bone defect are often lobulated and the lesion tends to exhibit some bone reaction as it undergoes healing.

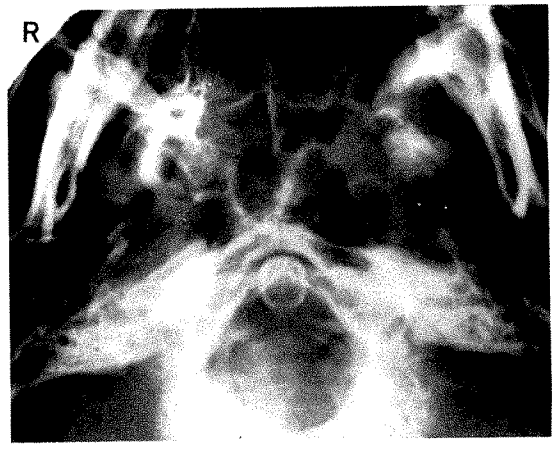



Fig. 34. Destruction of the floor of the left middle fossa secondary to carcinoma of the nasopharynx.

E. *Infection*

Specific or nonspecific osteomyelitis, secondary to fracture, hematogenous dissemination or paranasal sinus infec-

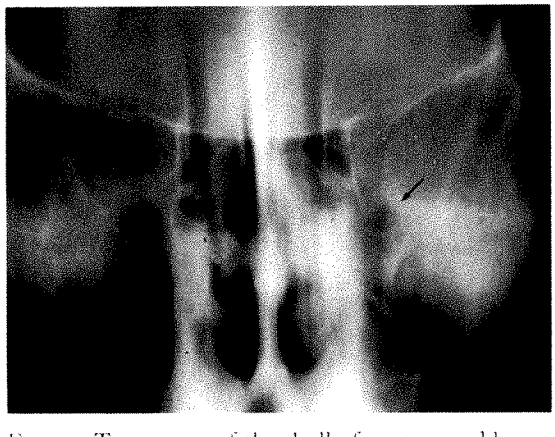


Fig. 33. Tomogram of the skull of a 75 year old man with epidermoid carcinoma of the left nares, antrum, base of skull and meninges. Note the enlargement of the left foramen rotundum (arrow) and loss of its cortical margins due to direct tumor invasion.

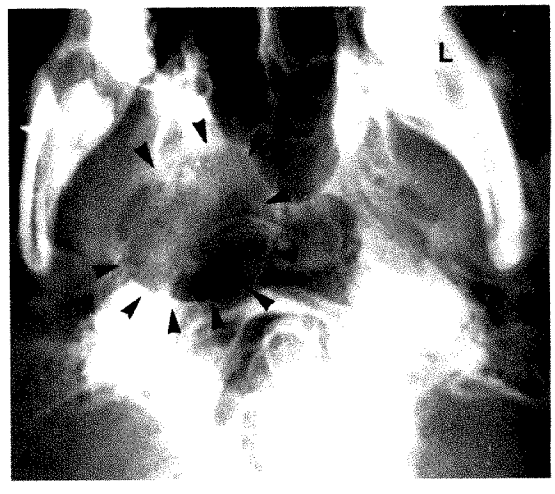



Fig. 35. Extensive, sharply defined erosion of the floor of the right middle fossa due to a large aneurysm of the internal carotid artery (arrows); surgically confirmed. (Courtesy R. Birkenfeld, M.D.)

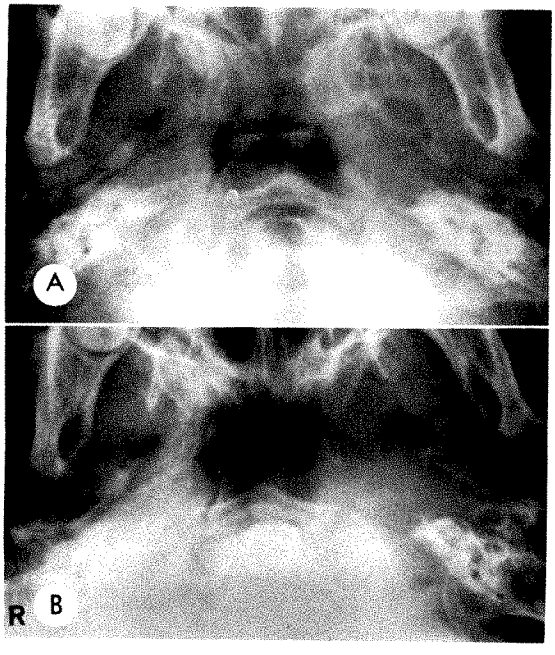



FIG. 36. Histiocytosis X with destruction of the floor of the left middle fossa. The foramina ovale and spinosum are still intact (*A*). Re-examination 1 year later shows progressive bone destruction with loss of the margins of the middle fossa foramina on the left side (*B*).

tion rarely involves the base of the skull. However, it can result in irregular moth-eaten destruction of bone which may be difficult to differentiate from osteolytic metastases on roentgen evidence alone.

### II. DISEASES WHICH MAY CAUSE FORAMINAL NARROWING

#### A. *Fibrous dysplasia*

The sclerotic variety of fibrous dysplasia may involve the vault, the base or the facial bones, singly or in combination[13] (Fig. 37). The disease may or may not be present in other portions of the skeleton as well. When the skull base is diffusely affected, the face and the vault are almost invariably involved. However, this may not occur in localized fibrous dysplasia involving a part of the skull base. In the absence of facial bone involvement, the localized sclerotic lesion of fibrous dysplasia may resemble meningioma en plaque. The abnormally thickened bone has a bizarre trabecular pattern and a somewhat ground-glass appearance which is disproportionately radiolucent compared with the over-all bone thickness.

#### B. *Paget's disease*

When the floor of the skull is affected by the sclerotic form of Paget's disease, the vault also commonly exhibits increased density. The thickened bone with patchy, ill-defined areas of sclerosis, the abnormal trabecular mosaic, and the increased width of the diploe are characteristic of this disease (Fig. 38).

#### C. *Osteoblastic metastasis*

Localized sclerotic metastases, particularly from the prostate and breast, may occasionally involve the floor of the middle fossa. Rarely, however, are the osteoblastic lesions limited to the middle fossa. The presence of similar

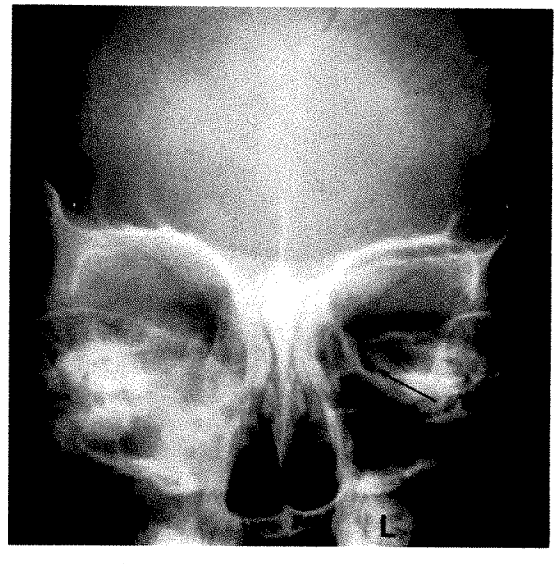



FIG. 37. Fibrous dysplasia in a young boy involving the anterior and middle fossae of the skull. Note the normal left foramen rotundum (arrow) and obliteration of the outline of the foramen on the right side by the sclerotic bone. (Courtesy M. H. Wittenborg, M.D., Children's Hospital Medical Center, Boston, Mass.)

lesions in the facial bones, especially in older people, should suggest the diagnosis of metastasis.[17]

D. *Osteopetrosis*

This disease is thought to be due to a congenital defect in bone resorption. Usually, the entire skeleton, and particularly the axial skeleton, are affected. The vault and base are increased in density but are not significantly thickened. Various foramina, including those of the middle fossa, may be narrowed causing neural compression (Fig. 39).

SUMMARY

The authors have presented the anatomic relationships of the major foramina of the middle fossa of the human skull and various factors to be considered in their roentgenographic demonstration. From comparative studies of representative mammalian skulls, including those of subhuman primates, phylogenetic relationships can be recognized in the morphogenesis of the foraminal variations encountered in man. An effort is made to point out those variations which may be clinically significant. In addition, disease processes which affect the bone structures at the base of the skull and alter the foramina of the middle fossa by erosive or constrictive change, are discussed in terms of roentgenographic recognition and interpretation.

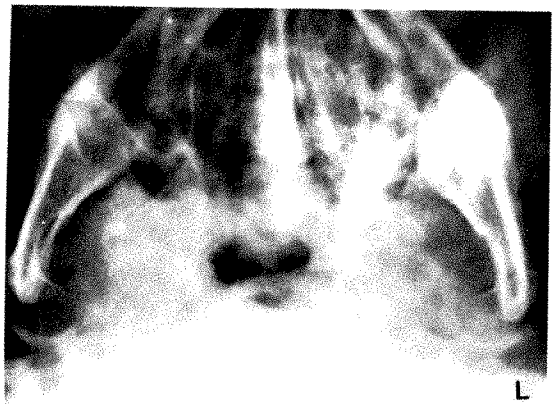



Fig. 38. Roentgenogram of skull in a 65 year old man with advanced Paget's disease. Note the loss of definition and constriction of the foramina of the middle fossa due to the ill-defined, dense sclerotic bone.




Fig. 39. Posteroanterior roentgenogram of postmortem specimen of skull in a 32 month old female with osteopetrosis. Note the abnormally dense bone around the base of the skull, orbits and face. The arrows point to the markedly constricted foramina rotunda. (Courtesy M. H. Wittenborg, M.D., Children's Hospital Medical Center, Boston, Mass.)

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# TRAUMATIC SPHENOID SINUS EFFUSION*

## AN ANALYSIS OF 50 CASES

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AN AIR-FLUID level in the sphenoid sinus following acute head trauma can represent an early roentgenographic sign of significant basilar skull injury.

Although post-traumatic effusion of the sphenoid sinus has previously been described,[1,2,3,7] the etiology and significance of this finding has not been established. Dong and his associates[1] reported 33 such patients with appropriate skull roentgenograms obtained following head trauma. A large majority (90 per cent) had clinical signs of basilar skull injury. They concluded that cerebrospinal fluid or sinusal hemorrhage at the floor of the anterior fossa could drain by gravity into the sphenoid sinus. Reynolds[7] found frontal injury a prominent characteristic in his 14 patients but felt that nasal hemorrhage would not readily drain into an intact sinus. Duboulay[2] has described the air-fluid level to be due to cerebrospinal fluid leakage.

It is evident from the above that the significance of a sphenoid effusion found on skull roentgenograms following trauma is difficult to assess in the individual case. The question arises as to whether this finding represents (1) drainage of blood or cerebrospinal fluid from facial trauma through the sphenoid ostium into an intact sinus, or (2) extension of an undetected basilar or anterior fossa fracture into the sphenoid body with mucosal laceration or cerebrospinal fluid leakage.

The purpose of this paper is to attempt to determine more accurately the significance, appearance, and composition of this roentgenographic finding. A clinical review of 50 patients with traumatic sphenoid effusion was made.

### MATERIAL AND METHODS

A horizontal beam, brow-up lateral projection of the skull has been instituted in the *routine* post-traumatic skull roentgenographic series at Duke University Medical Center. Adequate roentgenographic visualization of the sphenoid sinus in this position is also obtained in cerebral angiography, pneumoencephalography, and many initial lateral cervical spine roentgenograms following trauma. From our roentgenographic material of the preceding 6 years, we have collected 50 cases of sphenoid sinus effusion following acute head trauma. Two nontraumatic cases were found during special neuroradiologic procedures. One patient had cerebrospinal fluid rhinorrhea following several bouts of meningitis (Fig. 1), the other, a young woman, had subarachnoid hemorrhage from a ruptured aneurysm of the basilar

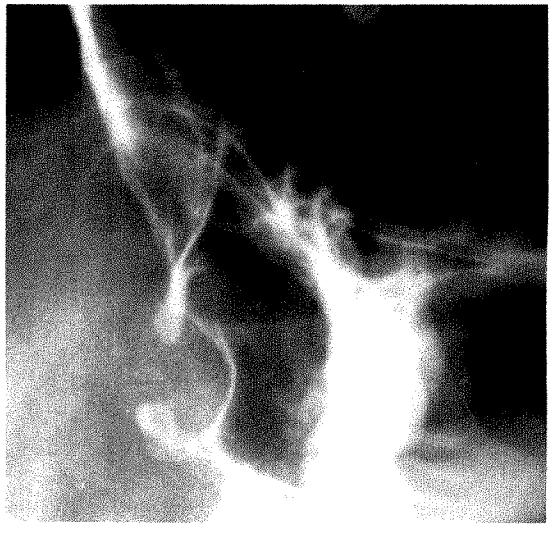


FIG. 1. Nontraumatic sphenoid effusion following meningitis.

* From the Department of Radiology, Duke University Medical Center, Durham, North Carolina.

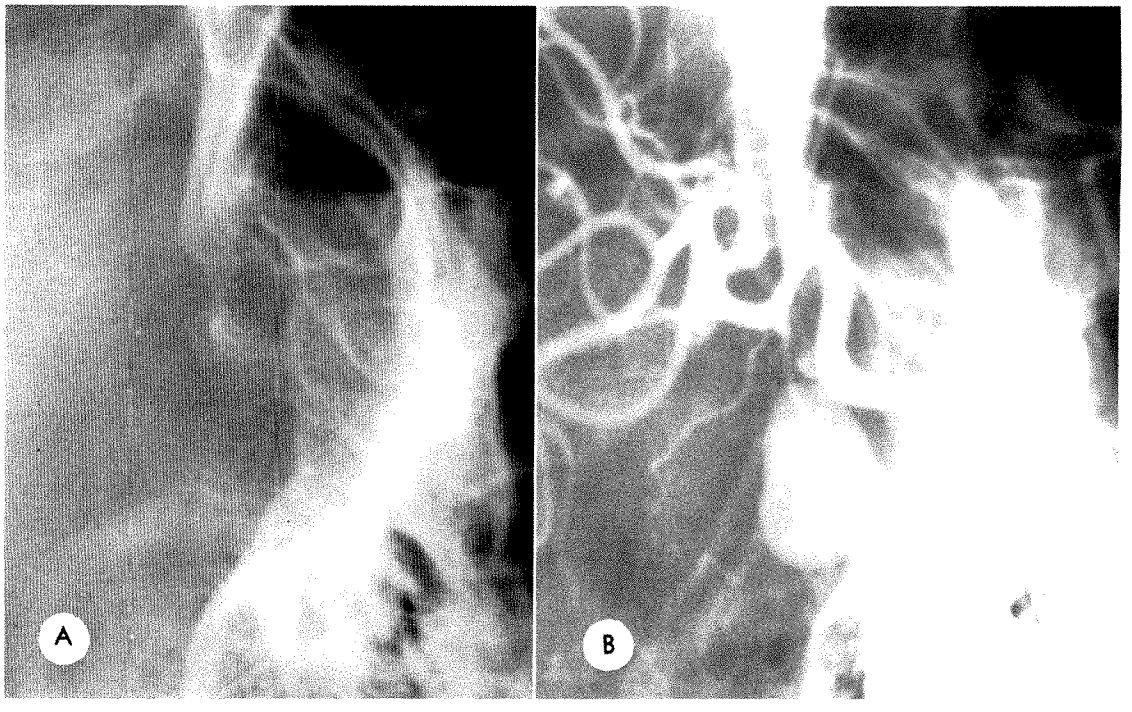



FIG. 2. (*A* and *B*) Nontraumatic sphenoid effusion as an incidental finding in patient with basilar artery aneurysm.

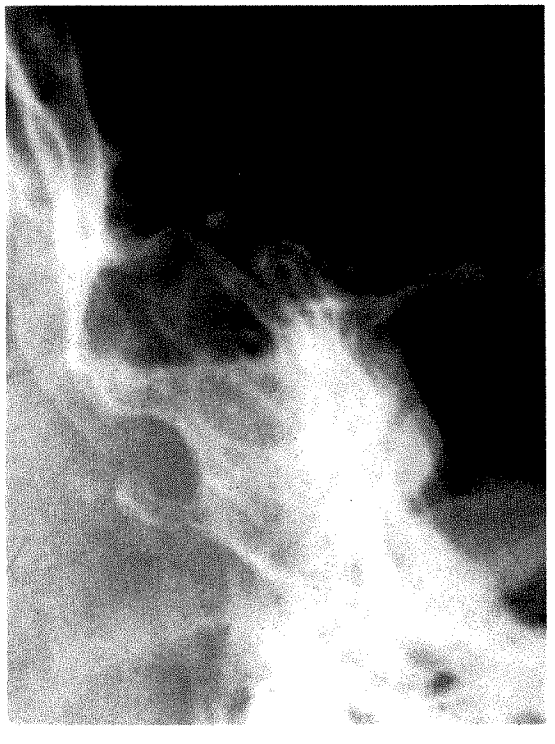


FIG. 3. Traumatic sphenoid effusion with air-fluid level well demonstrated.

artery without a history of sinus infection (Fig. 2, *A* and *B*).

The presence of a sphenoid sinus effusion was determined by either the demonstration of a definite air-fluid level (Fig. 3) or complete clouding within the cortical bone margins of the sinus[7] (Fig. 4). The type of trauma, appearance of effusion, extent of injuries, and final outcome are recorded in Table I.

## RESULTS

Moving vehicle accidents with frontal head trauma were the predominant initial injuries in most cases (68 per cent). Falls, direct blows to the head, and gunshot wounds comprised the remainder with about equal frequency (Table I). Although distinct air-fluid levels were usually seen, there were 4 patients (8 per cent) with a totally opacified sphenoid sinus. The detection of an opaque sinus requires the roentgenographic demonstration of a distinct cortical margin of the sinus wall.

Nasofacial hemorrhage was present in 66 per cent of the cases (Table II) but was found without evidence of cranial injury in only 1 patient. This was a 50 year old man who had been severely beaten about the face without loss of consciousness or demonstrable fracture. Bilateral nasal packs were in place at the time of the roentgenographic examination. Shortly thereafter, this patient developed cardiac arrest with subsequent permanent brain damage.

Cranial injury was considered under four criteria: 1. Loss of consciousness; 2. blood or spinal fluid draining from an ear or visualized behind an eardrum; 3. roentgen evidence of cranial fracture; and 4. surgical or autopsy evidence of fracture or intracranial hemorrhage.

At least one of the above criteria was fulfilled in all but one case (Table III). Roentgenographic evidence of cranial vault fracture on routine examination was found in 78 per cent of the cases (Table II), with the force of injury demonstrated in the frontal and parietal-temporal areas about equally (Table IV).

The absence of both cranial vault fracture and nasofacial hemorrhage in the presence of a traumatic effusion of the sphenoid sinus was found in 4 patients (Table II). A 52 year old male (Patient 25) suffered multiple hammer blows to the head with no demonstrable fracture but a history of loss of consciousness and clinical evidence of cerebrospinal fluid otorrhea. A second case (Patient 39) was a 69 year old female who was found unconscious following a fall. Although no fracture was demonstrated, a subdural hematoma was found by carotid angiography. The patient expired following surgery. The third case (Patient 48) was that of a 19 year old female automobile accident victim with superficial evidence of an occipital injury as well as thoracic injuries. The patient expired in respiratory distress several days later and never regained consciousness. An autopsy revealed basilar contusions of the brain. No definite fracture was found, although a specific study of the sphenoid was not performed. The fourth case (Patient 3) was a 19 year old male injured in an automobile accident 24 hours prior to roentgenographic examination. Although no fracture was demonstrated, blood was visualized behind the left eardrum in association with a superficial contusion over the left occiput. The patient recovered except for permanently decreased hearing on the affected side.

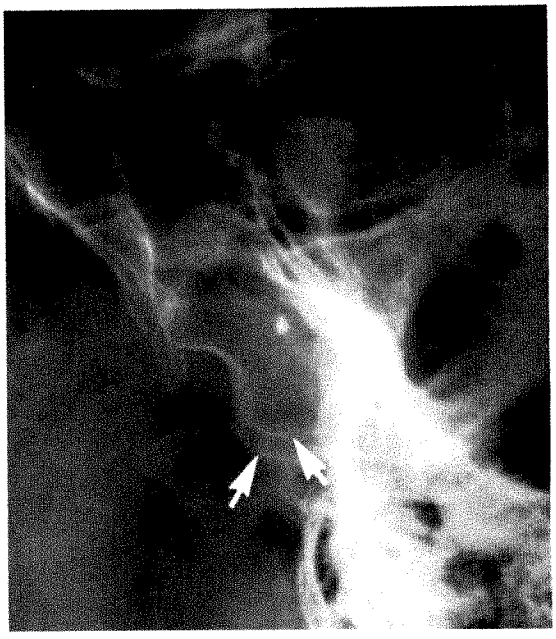


Fig. 4. Traumatic effusion with nearly complete opacification of the sphenoid sinus. The posterior cortical margins of the sinus are indicated by arrows.

In one instance (Patient 37, Fig. 3), we had the opportunity to carefully evaluate the sphenoid bone at postmortem examination. Well organized blood clots without fracture were found indicating traumatic mucosal hemorrhage.

Follow-up evaluation revealed a death rate of 23 per cent. If permanent neurologic complications were included, 48 per cent of the patients were seriously affected (Table V). One patient developed a carotid-cavernous fistula with severe epistaxis several months following the initial head

TABLE I

SUMMARY OF CASES

| Patient | Initial Episode | Effusion | Nasofacial Hemorrhage | Clinical Cranial Injury | | | | Outcome |
|---|---|---|---|---|---|---|---|---|
| | | | | Unconscious | Otorrhagia | Roentgen Fracture | Surgery Autopsy | |
| 1. JW | auto | a/f | − | + | + | T | — | ↓ hearing |
| 2. TB | fall | a/f | + | + | + | O | — | recovered |
| 3. JW | auto | a/f | − | − | + | — | — | ↓ hearing |
| 4. TS | blows | a/f | − | + | + | P-T-O | s.h. | brain damage |
| 5. WW | auto | a/f | + | − | − | F | — | recovered |
| 6. EA | auto | a/f | − | − | − | T | sp. fx.+s.h. | recovered |
| 7. SW | fall | a/f | + | + | + | P-T | cont. | *died* |
| 8. RP | auto | a/f | + | + | + | T-T-P | sp. fx.+s.h. | *died* |
| 9. RS | auto | a/f | + | + | + | T-P | — | recovered |
| 10. JL | auto | a/f | − | + | + | O | — | recovered |
| 11. JS | auto | a/f | + | + | + | O | — | recovered |
| 12. AL | auto | a/f | + | + | − | — | — | recovered |
| 13. FS | auto | a/f | + | + | + | P-F | sp. fx. | recovered |
| 14. AH | auto | a/f | + | − | − | F | — | recovered |
| 15. JM | auto | a/f | + | + | + | — | — | car.-cav. fist. |
| 16. JS | auto | a/f | + | + | + | F | — | recovered |
| 17. JM | auto | T.O. | + | + | + | F-P | s.h. | *died* |
| 18. TM | fall | a/f | + | + | − | T-P | sp. fx.+s.h. | *died* |
| 19. NB | auto | a/f | + | + | + | T-P | sp. fx.+s.h. | *died* |
| 20. LH | auto | T.O. | + | + | − | F | s.h. | *died* |
| 21. RS | auto | a/f | + | − | − | F-P | eth. fx. | recovered |
| 22. OA | blows | a/f | − | − | + | F | — | recovered |
| 23. JJ | fall | a/f | − | + | − | T-P | — | recovered |
| 24. WW | auto | a/f | + | + | + | F-T | sp. fx. | recovered |
| 25. AR | blows | a/f | − | − | − | — | — | recovered |
| 26. WM | auto | T.O. | + | + | + | F | sp. fx. | *died* |
| 27. JP | auto | a/f | + | + | − | F | — | *died* |
| 28. AC | auto | a/f | + | + | NE | — | — | recovered |
| 29. TH | machine | T.O. | + | − | + | F-T | sp. fx. | recovered |
| 30. CC | auto | a/f | + | + | − | T-P | s.h. | recovered |
| 31. GG | auto | a/f | + | + | − | — | — | recovered |
| 32. JB | auto | a/f | − | + | NE | P | cont. | coma |
| 33. ES | blows | a/f | + | − | − | — | — | neurodeficit |
| 34. LW | gunshot | a/f | + | − | − | F | — | recovered |
| 35. KB | blows | a/f | − | − | − | P | — | paresis |
| 36. WS | fall | a/f | − | + | − | P-O-P | s.h. | coma |
| 37. WD | auto | a/f | + | + | + | P-O | — | *died* |
| 38. LA | auto | a/f | + | + | − | — | — | paresis |
| 39. EB | fall | a/f | − | + | − | — | s.h. | *died* |
| 40. CW | gunshot | a/f | + | − | NE | F | s.h. | *died* |
| 41. RE | scooter | a/f | + | + | − | T | — | brain damage |
| 42. JP | auto | a/f | − | + | − | P | s.h. | recovered |
| 43. DD | auto | a/f | + | + | + | — | — | recovered |
| 44. JH | auto | a/f | + | + | − | T | e.h. | *died* |
| 45. JM | auto | a/f | − | + | + | T | s.h. | *died* |
| 46. RM | train | a/f | − | + | − | O | — | seizures |
| 47. EA | auto | a/f | − | − | − | P-T | — | recovered |
| 48. GW | auto | a/f | − | + | NE | — | cont. | *died* |
| 49. SH | gunshot | a/f | + | + | − | F | — | recovered |
| 50. FC | gunshot | a/f | + | − | − | F | — | recovered |
| *51. MS | s.a.h. | a/f | − | + | − | — | — | *died* |
| *52. WH | men. | a/f | − | − | − | — | — | recovered |

CODE:

a/f = air-fluid level
T.O. = total opacification
− = absent
+ = present
NE = not evaluated
T = temporal bone
O = occipital bone
P = parietal bone
F = frontal bone
s.h. = subdural hematoma
e.h. = epidural hematoma
sp. fx. = sphenoid fracture
eth. fx. = ethmoid fracture
cont. = contusion of brain
s.a.h. = subarachnoid hemorrhage
men. = post meningitis
↓ hearing = decreased hearing
* nontraumatic cases

Table II

NASOFACIAL HEMORRHAGE

| | | |
|---|---|---|
| Present | | 33 |
| with vault fracture | 26 | |
| without vault fracture | 7 | |
| Absent | | 17 |
| with vault fracture | 13 | |
| without vault fracture | 4 | |

injury. Dong and his associates[1] reported a similar complication in their series.

## DISCUSSION

We have directly demonstrated a fluid level proven to be blood in one case (Patient 37), and indirectly shown that cerebrospinal fluid was present in another case (Patient 52) without trauma. We have also followed one patient (Patient 36) with serial lateral roentgenograms demonstrating a haziness to the air-fluid interface several weeks following injury (Fig. 5, *A–C*). The appearance of the opacified sinus also failed to change with variable positioning of the patient's head. These findings indirectly suggest the formation of clotted mucosal hemorrhage. Therefore, it seems that blood and/or spinal fluid can produce the air-fluid level.

Paranasal sinusitis was not clinically evident in any case. Although sphenoid effusion from inflammation alone has been described,[5] we did not evaluate any patient for sinusitis.

Although no definite sphenoid body fracture was found by roentgenographic examination, 8 of 22 cases with either surgical or postmortem exploration revealed definite sphenoid fractures. The necessity of an associated sphenoid fracture has not been determined. In this regard, it should be noted that Patient 37 revealed a fluid level (Fig. 3) without a sphenoid fracture found at autopsy. Reynolds[7] studied 30 cases of nasal hemorrhage alone and failed to demonstrate significant opacification of the sphenoid sinus. Proetz[6] has described a method of displacement of oily contrast material into an intact sinus for mucosal evaluation. It is possible, therefore, that nasopharyngeal blood, or blood and/or spinal fluid from an anterior fossa fracture, might drain into an intact sinus in a patient who has been in the supine position prior to the skull roentgenographic examination. It is difficult to conceive that sufficient fluid can ordinarily drain against normal mucosal activity to completely, or near completely, opacify a large sphenoid sinus. Our one case of sphenoid effusion following trauma but without evidence of cranial injury possessed the added factor of positive pressure from bilateral nasal packs to displace blood into a possibly intact sinus.

Table III

CLINICAL CRANIAL INJURY

| Criteria | No. | Per Cent |
|---|---|---|
| Unconsciousness | 37 | 74 |
| Otorrhagia (or otorrhea) | 22/46 | 48 |
| Roentgen fracture | 39 | 78 |
| Surgical or autopsy fracture or intracranial hemorrhage without demonstrable fracture | 2 | 4 |
| Any of the above | 49 | 98 |

Table IV

PREDOMINANT FRACTURE

| | | |
|---|---|---|
| Frontal-facial | 21 | 42% |
| Parietal-temporal | 19 | 38% |
| Occipital | 6 | 12% |

Lewin and Cairns[4] described common methods of fracture of the sphenoid sinus, one being a blow to the side of the head with extension along the coronal plane. Other mechanisms involved facial or frontal head-on blows, as often seen in

Table V

FINAL OUTCOME

| | | |
|---|---|---|
| Death | 14 | |
| Permanent neurologic deficit | 10 | |
| Carotid-cavernous fistula | 1 | |
| | 25 | 50% |

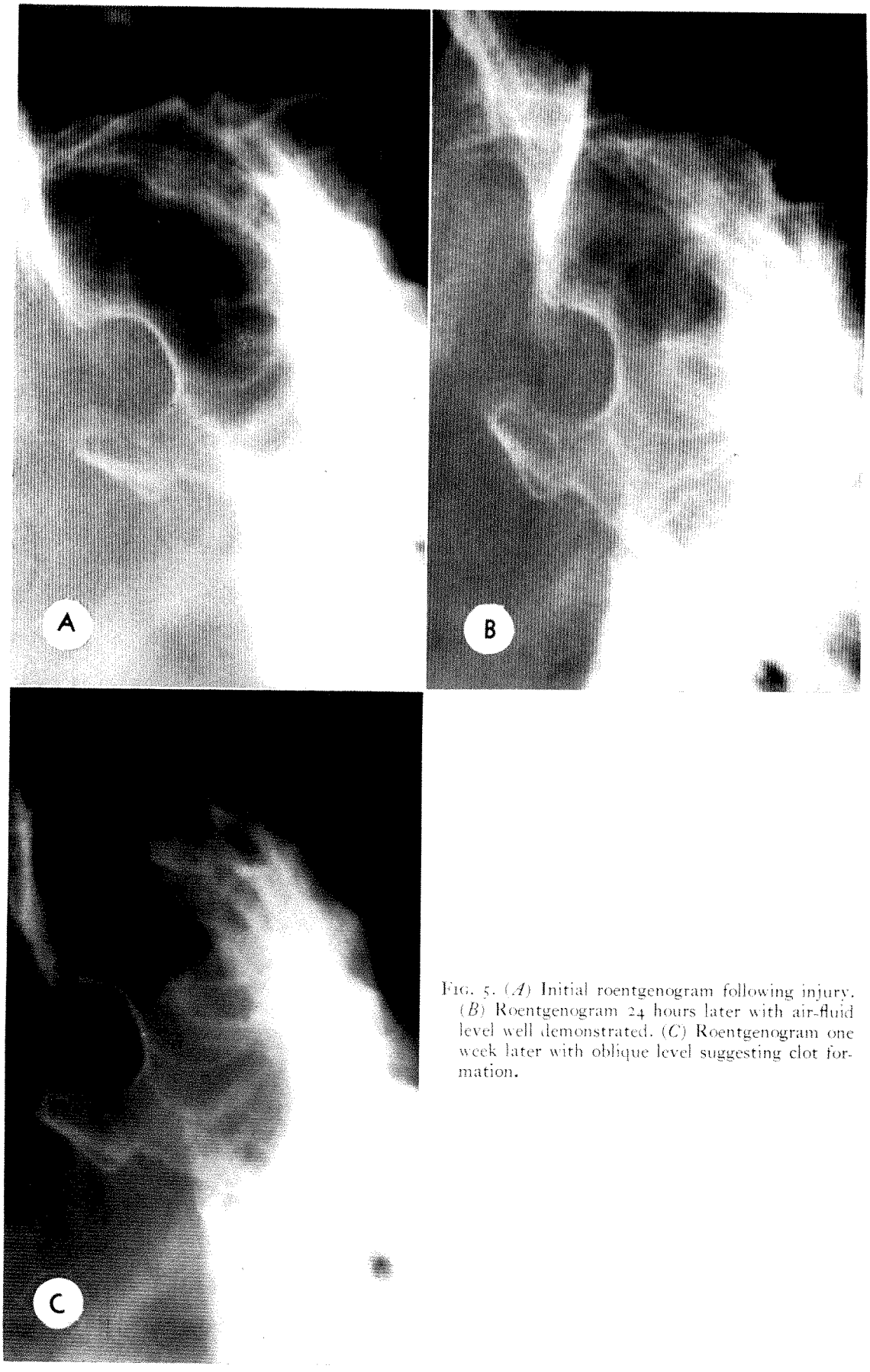



FIG. 5. (*A*) Initial roentgenogram following injury. (*B*) Roentgenogram 24 hours later with air-fluid level well demonstrated. (*C*) Roentgenogram one week later with oblique level suggesting clot formation.

automobile accidents, with direct extension to the base of the skull.

It should be added that a brow-up lateral, horizontal beam projection of the skull has the additional value of offering a true lateral view of the atlanto-occipital articulation.[7] This is another area of concern in the injured patient with neurologic deficit.

## CONCLUSION

In a review of 50 patients with the roentgenographic demonstration of a sphenoid sinus effusion following trauma, the final outcome was death or permanent neurologic deficit in about one half of the cases (48 per cent).

A brow-up, horizontal beam lateral projection included in the *routine* post-traumatic skull roentgenographic series allows detection of a sphenoid sinus effusion of blood and/or spinal fluid and can increase the index of suspicion of a more severe cranial injury. A sphenoid effusion, in the absence of demonstrable cranial fracture, cannot be attributed to nasofacial trauma alone without further considering the possibility of a basilar skull fracture.

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# MUCORMYCOSIS INFECTION OF THE CRANIOFACIAL STRUCTURES*

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INFECTION in humans by fungi of the order *Mucorales* (mucormycosis) has only recently been widely recognized. Craniofacial infection by these fungi often leads to a rapidly fatal course. Recent advances in therapy have led to occasional cures. The roentgen manifestations of this disease are such that the radiologist may play a significant role in its early recognition. Although the clinical manifestations of craniofacial mucormycosis have been extensively described in recent literature, little attention has been given to its roentgen changes.

## REPORT OF A CASE

H. L. (#396464), a 45 year old Negro male, was admitted to the Philadelphia General Hospital on October 13, 1966 because of diabetic acidosis. He was lethargic, with Kussmaul respirations and uremic breath odor. Deep feet ulcerations were present; 6 toes had been previously amputated. Oral temperature was 100°F. Admission blood sugar was 630 mg. per 100 cc. Urine acetone was 3+. Blood urea nitrogen was 132 mg. per 100 cc.

Control of the blood sugar was promptly attained. Cephalothin (keflin) was administered parenterally from October 14 to October 28, 1966 because of the feet ulcerations. His temperature never exceeded 100°F. Blood urea nitrogen remained elevated at approximately 110 mg. per 100 cc. throughout his hospital course.

On the eighth hospital day and thereafter, frequent episodes of confusion and belligerence occurred. On the evening of the fifteenth hospital day, right periorbital swelling and ptosis were noted. The right pupil was dilated and fixed. Superior gaze, corneal reflex and facial sensation were lost on the right side. The right superior and inferior nasal turbinates appeared black and necrotic. This latter finding led the intern* to suspect mucormycosis. Biopsy and fungal cultures of the necrotic turbinates were obtained that same evening. Cultures on Sabouraud's agar revealed no evidence of *Mucorales fungi*. The biopsy sections, however, confirmed the clinical suspicion of mucormycosis by showing broad, nonseptate, fungal hyphae within the necrotic debris (Fig. 1). Cerebrospinal fluid examination revealed 31 leukocytes and 64 red blood cells per cu. mm., with 150 mg. protein and 294 mg.



FIG. 1. Biopsy specimen from right nasal turbinate demonstrates necrotic tissue containing large, nonseptate fungi characteristic of mucormycosis. (Hematoxylin and eosin, ×300.)

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sugar per 100 cc. (Blood sugar was 420 mg. per 100 cc. at this time.) Smear and culture of the cerebrospinal fluid revealed no organisms.

*Roentgenographic Findings.* Roentgenograms of the skull and paranasal sinuses, including tomograms in the Waters projection, (Fig. 2; 3, *A* and *B*; and 4) revealed nodular thickening of the soft tissue lining of both maxillary antra and opacification and indistinctness of the anterior ethmoid air cells. These findings were more pronounced on the right side. Multiple small areas of bone destruction were present in both orbital floors and the medial wall of the right orbit.

Specific therapy for mucormycosis with amphotericin B was not started because of the markedly elevated blood urea nitrogen. The patient expired on the eighteenth hospital day.

*Autopsy Findings.* At autopsy, necrosis of the palate was observed. The right periorbital fat was hyperemic. The ethmoid sinuses were engorged and swollen.

Examination of the brain revealed thickening of the arachnoid at the base extending from the frontal pole to the midbrain with purulent exudate in the adjacent subarachnoid space. This was most severe over the cribriform plate. There was no evidence of cavernous sinus thrombosis.

Microscopically, hyphae and spores of mucormycosis were seen in the purulent subarachnoid space exudate. The subarachnoid arteries and veins in these areas showed invasion of their walls and occlusion of their lumina with fungi. Small vessels penetrating into brain tissue also showed fungal invasion. The right third nerve was extensively invaded by fungi.



FIG. 2. Roentgenogram, Waters' projection, shows marked nodular thickening of the soft tissue lining of both maxillary antra and indistinctness of the bony margin of the right orbital floor.

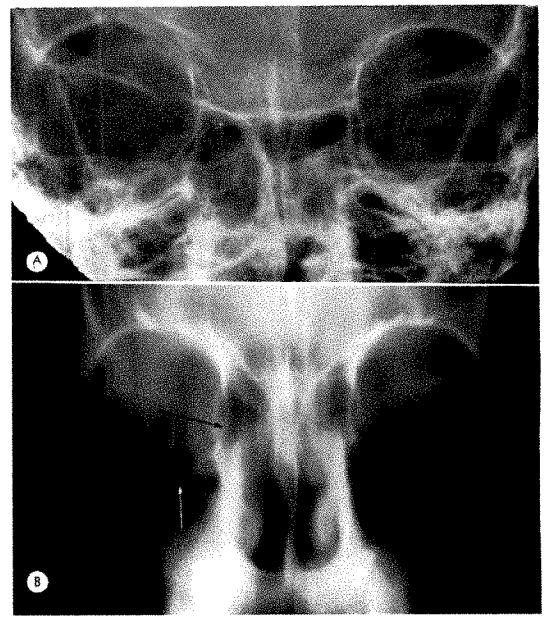



FIG. 3. (*A*) Caldwell's projection shows nonuniform opacification of ethmoid air cells, particularly on the right side. There is indistinctness of the right orbital plate of the ethmoid bone suggesting bone destruction. (*B*) Anteroposterior tomogram at the level of anterior ethmoid cells confirms small area of bone destruction of right ethmoid orbital plate (black arrow). A separate area of bone destruction is seen in the right orbital floor (white arrow).

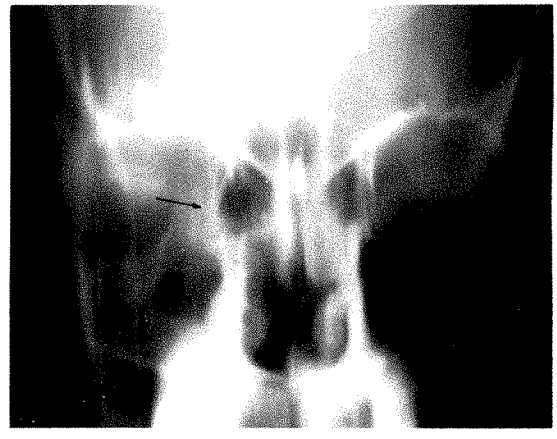


FIG. 4. Anteroposterior tomogram at the level just posterior to Figure 3*B* demonstrates additional destruction of right orbital plate of ethmoid bone (arrow). There is suggestion of another small zone of destruction in the left ethmoid orbital plate.

No fungi were found elsewhere in the body outside the craniofacial structures. Microscopic examination of the kidneys showed evidence of severe Kimmelstiel-Wilson disease.

## HISTORICAL REVIEW

Human mucormycosis was first reported by Paltauf[21] in 1885. In 1943, Gregory *et al.*[11] revived interest in this entity with a report of 3 fatal cases of craniofacial mucormycosis. There was a striking similarity between all 3 of his cases: a diabetic patient in acidosis developed mucormycosis in the paranasal sinuses or orbit which rapidly spread to the adjacent brain causing prompt death. Other reports, including an early description from this hospital,[27] confirmed this pattern.[4,20,24,30] These reports led to antemortem recognition of this entity and eventually to some cures of mucormycosis with potent antifungal agents,[3,6,18] extensive surgery[9,19] or desensitization.[13]

## DISCUSSION

Besides mucormycosis of the craniofacial structures, various areas of the body may become infected, including lungs,[1] kidneys,[22] and the gastrointestinal tract.[2,28] Infection of these other organ systems will not be discussed in this report.

Infection in the head by mucormycosis appears to start by fungal invasion of the nasal mucosa.[4] These ubiquitous saprophytes have ample contact with nasal mucosa but generally become invasive only when body physiology is markedly disturbed, usually by diabetic acidosis. Antibiotic or cortisone therapy makes this invasion more likely. Nondiabetic debilitated patients, such as leukemics on suppressive therapy, are also susceptible to mucormycosis but then the portal of entry is usually the lung with a far different resulting syndrome.[1,14,25] Craniofacial mucormycosis with fungi entering by invasion of the nasal mucosa has been reported in nondiabetic patients, including uremic children on cortisone or antibiotic therapy[7,18] and children developing acidosis while on antibiotic and fluid therapy for gastroenteritis.[12,15] The majority of cases of craniofacial mucormycosis, however, occur in poorly controlled diabetics.

After invasion of the nasal mucosa, the organism spreads by direct extension to the maxillary sinuses and orbits.[4] Black necrotic turbinates are visible on nasal examination in most cases. Biopsy of such turbinates is the most practical method of establishing the diagnosis. These fungi have an unusual propensity for invasion of arterial walls. This disease has been viewed as a fungal arteritis and this concept seems quite useful.[29] The necrosis of the turbinates is certainly related to fungal invasion and occlusion of regional arteries. Surgical[9,10,19] and autopsy[8,29] material has demonstrated extensive necrosis involving the hard palate and the walls of the maxillary antra, ethmoids and orbits due to this phenomenon. Extension beyond these facial structures may be of two types, both rapidly fatal: fungal invasion of multiple small arteries at the adjacent undersurface of the brain,[4,17,27] or fungal invasion and occlusion of the carotid arteries.[5,18,20,23,24]

Many reports of craniofacial mucormycosis briefly mention roentgenographic findings.[3–8,13,16,24,26] Unfortunately none of these include either detailed roentgen description or reproductions of the roentgenograms. Most reports describe "thickening of the lining membranes" of the maxillary and, occasionally, the ethmoid and sphenoid sinuses "without fluid levels." Gass,[10] in a report of 2 cases, mentions some destruction of the bony walls of the ethmoid sinuses. These meager descriptions gleaned from the literature, combined with our experience with craniofacial mucormycosis in 2 cases (one of the authors has prior experience with another case), indicate that there is a characteristic combination of roentgenographic changes in craniofacial mucormycosis.

The combination of roentgenographic changes in the paranasal sinuses which we believe to be characteristic for craniofacial mucormycosis consists of the following: (1)

Nodular thickening of the soft tissue lining of many paranasal sinuses with sparing of the frontal sinuses; (2) absence of fluid levels in erect roentgenograms; and (3) spotty destruction of the bony walls of multiple paranasal sinuses.

Other conditions may produce some of the roentgen changes seen with craniofacial mucormycosis. Sinus opacification and bone destruction may be found with invasive tumor of the paranasal sinus region. Although multiple sinuses may be involved late in the course of the disease, bone destruction is adjacent to a single tumor mass in contrast to the multicentric bone destruction found in mucormycosis. The absence of fluid levels and the presence of bone destruction differentiate mucormycosis from acute inflammatory sinus disease. In chronic sinusitis, there occasionally may be bone destruction but usually combined with sclerosis.

Evidence of bone wall destruction should be visible on roentgen examination even early in the course of craniofacial mucormycosis. The fungal arteritis directly responsible for this early bone destruction has already been discussed.[8–10,19,29] Areas of bone destruction tend to be small and may be difficult to appreciate in routine paranasal sinus roentgenograms. Tomographic study was extremely valuable to us in evaluating the extent and nature of the bone destruction in the case reported here (Fig. 3*B*; and 4).

SUMMARY

Mucormycosis is an important, often rapidly fatal, human fungal infection. Infections have been reported in the lungs, kidneys, gastrointestinal tract and head. The typical clinical findings and unusual pathogenesis of the craniofacial form are discussed in this report. This form of mucormycosis, which is being recognized with increasing frequency in uncontrolled diabetics, has a combination of roentgen changes which are probably characteristic:

(1) Nodular thickening of the soft tissue lining of many paranasal sinuses, with sparing of the frontal sinuses.
(2) Absence of fluid levels in erect roentgenograms.
(3) Spotty destruction of the bony walls of multiple paranasal sinuses.

Roentgenographic recognition of craniofacial mucormycosis is important because cures have been reported following specific therapy.

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# THE ROENTGEN MANIFESTATIONS OF SCLEROMA*

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SCLEROMA is a chronic granulomatous affliction of the respiratory tract characterized by an indolent course, occasional remissions and frequent relapses with death ultimately resulting from sequelae such as sepsis, airway obstruction and hemorrhage. Spontaneous cures are rare. It was first described by von Hebra[28] and his colleague Kaposi as a form of cutaneous cancer or "granulation sarcoma" although according to prevailing opinion at the time it was considered a manifestation of syphilis. It is now generally regarded as an infection although it has much in common with neoplasia in terms of its natural history.[16,18]

The disease has shown certain endemic foci, but, in recent years, possibly owing to increased travel, it has become neither geographically nor ethnically limited. It was principally described in Central and South East Europe. It is relatively frequent in Asia, particularly in China and India, and in northern Africa (Egypt and Morocco). Its frequency is increasing in the Western Hemisphere with important foci in South and Central America. A review of the world literature up to 1963 revealed 4,000 reported cases.[16] Undoubtedly many more exist. Approximately 171 cases have been reported in the United States and Canada, of which 47 were native born. Some of the latter spent their lives in the vicinity of their birthplaces which were scattered throughout the United States and were not knowingly exposed to scleroma.[3,4,11,13,17,21,25,26]

Since lack of familiarity with the entity in nonendemic areas results in failure to recognize it, we are adding 2 cases to the literature, 1 of which occurred in a native born American. The greatest number of cases occur in the age group of from 16 to 35 years; however, it has also been observed in children.

The distribution among 2,361 cases[1] was as follows: nose 60 per cent, larynx 60 per cent, nasopharynx 28 per cent, pharynx 16 per cent, trachea and bronchi 9 per cent and other sites 5 per cent. Frequently more than one area is involved and primary laryngotracheal lesions are not uncommon.[5,19,22]

## CLINICAL ASPECTS

The disease usually progresses through 3 stages; the rhinitis or atrophic stage, the infiltrative or nodular stage and the stenotic or cicatricial stage. The first stage is characterized by watery nasal discharge, frontoethmoidal headaches and difficulty in breathing. Physical examination may reveal a hypertrophic nasal mucosa, mainly on the septum, or a thin, dry mucous membrane covered with large yellow crusts. Discrete, small bluish-red edematous nodules may be seen. This stage may last for months or years. It is gradually superseded by the second or florid stage, which is marked by progressive infiltration and the disappearance of coryza. Coalescence of the nodules occurs forming granular masses. These are seen in the nasal fossae and the septum which enlarges. When invasion is directed upwards, nasal respiration is hindered, when directed inferiorly, however, there is a tendency to complete obstruction. Anosmia occurs with forward extension and the masses sprout out of the nostrils to involve the nasal tip, alae, upper lip and gums. The masses bleed easily and become secondarily infected. Pressure necrosis occurs but ulceration is the exception and not the rule. The tissues become indurated and cartilaginous in consistency. Death may occur from asphyxia, infection,

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hemorrhage and debilitation. The masses gradually become avascular in the third or cicatricial stage with adhesions of the soft structures, contraction of the scleromatous tissue and further scarring and stenosis of the air passages.

Although the first complaint may not be referable to the nose, it may still be the site of origin, the unobtrusive rhinitis having been overlooked until more serious symptoms presented in the pharynx and larynx.

The disease most frequently begins in the nose and paranasal sinuses with gradual extension down the respiratory tract. Involvement has been reported of the septum, skin of upper lip, cheek, gums, lacrimal ducts, sinuses, eustachian tubes, nasopharynx, hard and soft palate, tonsils, pharynx, larynx, trachea and bronchi to the smallest ramifications.[22] The symptoms depend on the area, extent, and duration of involvement. The nares may be completely blocked and the nasal architecture so distorted that the features are unrecognizable. Interference with mastication, swallowing, defects in speech, respiratory tract infection, tinnitus, headache and deafness have been noted. Laryngeal and tracheal hemorrhage during the vascular granulomatous phase may be serious. Spontaneous regressions have occurred rarely, mainly in those with disease localized to the septum with residual nasal atrophy. Once established, the disease is slowly progressive and may extend over several decades. Usually, if a patient survives long enough, he will die of the disease unless its progress is arrested. In the 10 patients of Shaw and Martin,[25] the median duration of the disease was 14 years with a range of from 3 to 46 years. The same yardstick for "arrest" was used as for carcinoma, *i.e.*, 5 years free of active disease. As an example of chronicity they described an arrested case observed for 26 years. The nares were completely plugged with scarred fibrotic tissue, the soft palate was fused with the pharyngeal wall, the epiglottis was distorted, the sinuses and nasopharynx were sealed off from the external opening and the process had extended into the subcutaneous tissue with plugging of the tear ducts. Therefore, although the disease was apparently arrested, it was meaningless to say that he was cured.

The disease has been said to be limited to the skin and mucous membranes with cartilage, lymph nodes, bone and muscle being spared. There have, however, been isolated reports of muscle and bone invasion.[10]

Clinically, the induration of the lesions, stenosis and deformity with absence of ulceration, systemic involvement and pain throughout a chronic course should alert one to the possibility of scleroma.

## PATHOLOGY

Generally, the microscopic appearance is that of a granulomatous chronic inflammatory lesion with an infiltrate composed chiefly of plasma cells, mainly perivascular in distribution, and lymphocytes. Although the early and scarred end stages are nonspecific, the hallmarks of florid scleroma include Mikulicz's cells, plasma cells, Russell bodies, gram negative rods and hypertrophied epithelium. Mikulicz's cells are large and vacuolated (100–200 $\mu$ in diameter) with lacy foamy cytoplasm in which gram negative, bipolar von Frisch bacilli may be seen with routine stains. The Russell bodies are much smaller than Mikulicz's cells, are refractive to light, and are believed to be degenerated plasmocytes. Russell bodies and Mikulicz's cells are not peculiar to scleroma, however, and other causes of chronic granulomas and neoplasia must be eliminated. The pseudoepitheliomatous hyperplasia of the mucosal epithelium seen may be confused with carcinoma. The amount of cellular infiltration varies with the stage of the disease. After many years cellularity is replaced by fibrosis, which gives the disease its clinical wooden hardness and the name scleroma. In general, a granulomatous respiratory tract lesion showing plasma cells, Russell bodies, Mikulicz's cells in which gram negative bacilli can be demonstrated and in

which no acid fast organisms can be found, is almost certainly scleroma.

### BACTERIOLOGY AND SEROLOGY

Discovery of the von Frisch bacilli[27] within the Mikulicz cells and in cultures from nasal secretions, washings and biopsy material has favored a bacterial cause. However, the pathogenic action of *Klebsiella rhinoscleromatis* was not fully accepted until Levine[18] established the cultural characteristics for 19 of its strains and identified it as a distinct species and the probable cause of the disease. The organisms belong to the order of Eubacteriales (IV), tribe Escherichieae (I), family Enterobacteriaceae (IV), genus Klebsiella, species rhinoscleromatis.[2] Its recovery is considered essential for diagnosis by some,[11,18] important but not crucial by others[21] and unnecessary by others.[23] It is now considered a low grade infective disease needing prolonged exposure akin to leprosy in its transmission. Contagiousness is suspected on the basis of its occurrence in several members of the same family.[4,11,12] The greatest objection to the bacterial theory is that only two of Koch's three postulates have been fulfilled; *i.e.*, (1) the bacillus is found in the lesions, and (2) it can be cultured. However, the disease cannot be experimentally reproduced.

Although a viral etiology has been postulated, electron microscopy has failed to reveal such pathogens.[6] Others suggested the possibility of a virus and the bacilli acting symbiotically. The consensus of opinion incriminates *Klebsiella rhinoscleromatis* as the etiologic agent.[15,16,18]

A complement fixation test first described in 1909 by Goldzieher and Neuber[8] and later modified has been found to be of value by some authors.[9] The reaction has also been used as a guide in the management of treatment since with a positive reaction in a clinically cured case one must assume the existence of a residual focus and continue treatment.[16] An intradermal test similar to the Mantoux and agglutination tests has not been widely used.

### TREATMENT

A host of therapeutic agents have been advocated but many reported cures have been only briefly followed. Both external irradiation and radium have been used;[20,24,25] however, their value cannot be established on the basis of available evidence. Early surgery has been advised by some,[25] whereas others feel that procedures other than emergency tracheotomy and plastic surgery in the fibrotic end stage are contraindicated and may lead to dissemination or irreversible scar formation.[16,24]

Since the antibiotic era, therapy has undergone a marked change. Sulfonilamides were first employed unsuccessfully. The antibiotics now most frequently used, depending on sensitivity tests, are streptomycin, aureomycin, terramycin and chloromycetin. Disappearance of the Klebsiella bacilli from the respiratory passages accompanied by dramatic clinical improvement lends weight to the evidence that it is the etiologic agent. Despite claims for the efficacy of antibiotics, however, the relapse rate, chronicity and mortality are still high and prolonged follow-up is mandatory.

### REPORT OF CASES

Case 1. This 54 year old white, American born (Missouri) male entered the hospital because of nasal obstruction and watery discharge of 2 years' duration. Physical examination revealed almost complete obstruction of the nasal passages by a granular mass involving the nasal septum. Roentgenograms of the paranasal sinuses demonstrated evidence of pansinusitis (Fig. 1). The polypoid masses were excised and a partial ethmoidectomy was done. Microscopic examination revealed a moderately vascular tissue containing large clusters of lymphocytes, plasma cells, Russell bodies and collections of large cells with vesicular nuclei, small nucleoli, and clear cytoplasm. A diagnosis of rhinoscleroma was suggested.

After several symptom free months, he was readmitted because of recurrent nasal obstruction. Three firm, smooth masses were excised from the floor and lateral walls of the nose. Gram negative stains revealed bacilli within large clear cells, consistent with rhinoscleroma.

The nares were again occluded 7 years later

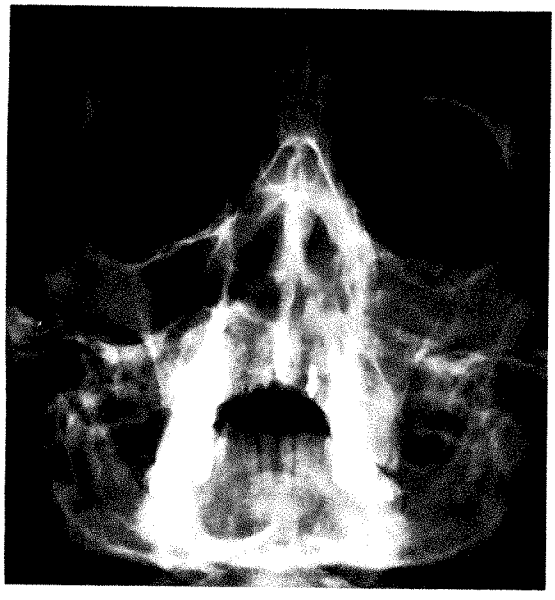


FIG. 1. *1954:* Marked clouding of the paranasal sinuses with minimal aeration of the left antrum and ethmoids is demonstrated. The nasal septum is irregularly thickened. Numerous masses based on the septum and lateral walls project into the nares.



FIG. 2. *1961:* The nares and all the paranasal sinuses are homogeneously opacified and occluded by large blocks of soft tissue.

(Fig. 2) when he was admitted because of increasing dyspnea. Round masses covered with reddish mucosa extended posteriorly. The hard

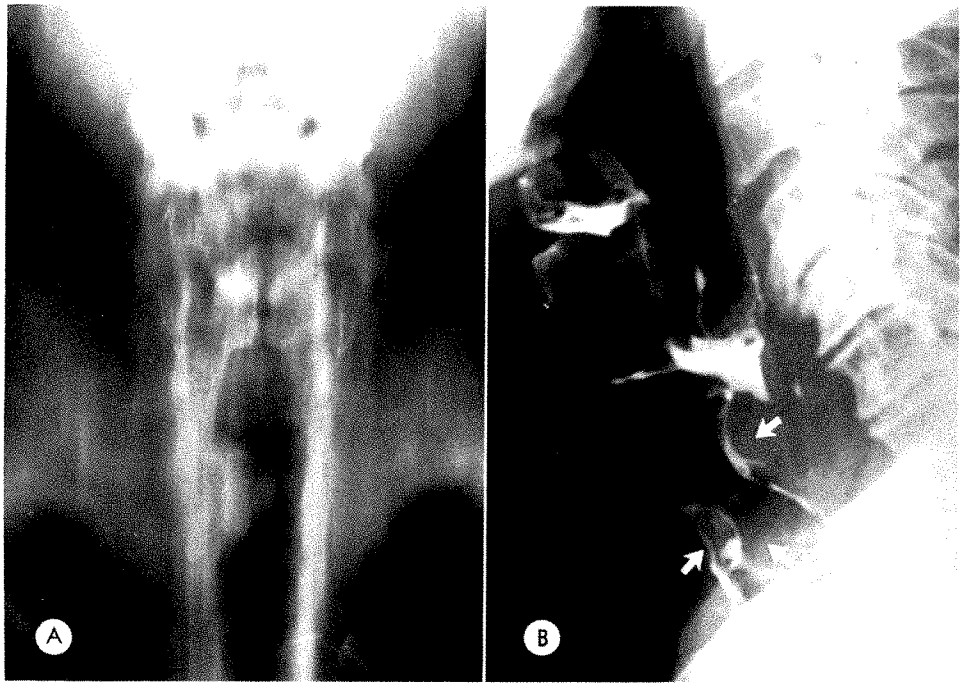



FIG. 3. *1961:* (*A*) Frontal laminagram demonstrates a large semicircular mass arising from the right lateral wall and projecting into the tracheal lumen. A smaller left sided mass is at a slightly different level. (*B*) Lateral contrast laryngogram outlines 3 masses arising from the posterior subglottic region and from the anterior and posterior lateral walls at a lower level (arrows).

palate was also covered with soft, smooth, spongy pink tissue. The oropharynx and vocal cords were normal. Throat culture revealed normal flora. Nose culture revealed *Klebsiella rhinoscleromatis*. Laminagraphy (Fig. 3*A*) and laryngography (Fig. 3*B*) revealed three soft tissue masses in the subglottic region and upper trachea.

Several additional hospital admissions occurred because of epilepsy. He was last seen in 1967 when roentgenography, utilizing pluridirectional laminagraphy, demonstrated evidence of bone destruction in the sphenoid, ethmoid and maxillary sinuses. A large soft tissue mass filling the entire sphenoid sinus and destroying its floor and anterior wall was defined (Fig. 4*A*). There was destruction of the alveolar ridge posteriorly and the anterior wall of the right antrum, with suggestive evidence of new bone formation at the latter site (Fig. 5*C*). Destruction of the medial and superior walls of both antra, particularly near the ethmoid recesses (Fig. 5*A*) as well as destruction of the floor of the middle fossa was also demonstrated.

Case II. This patient, a 49 year old white male who was born in the British East Indies and lived most of his life in Barbados, had complained of nasal obstruction for 25 years. This was gradual in onset and was later associated with ulceration of the palate and pharynx with dysphagia but no pain. Previous treatment had included radium therapy and bilateral antrotomies.

Physical examination disclosed irregular granulation of the hard and soft palate, gingiva and tonsils. The tonsillar pillars were fused to the posterior pharyngeal wall. The nasopharynx and hypopharynx could not be examined due to contraction of the oropharynx.

Biopsies of firm yellow tissue showed atrophic, acanthotic epithelium and submucosal exudate consisting of foamy mononuclear cells containing bacteria and Russell bodies on microscopic examination. This was deemed consistent with rhinoscleroma.

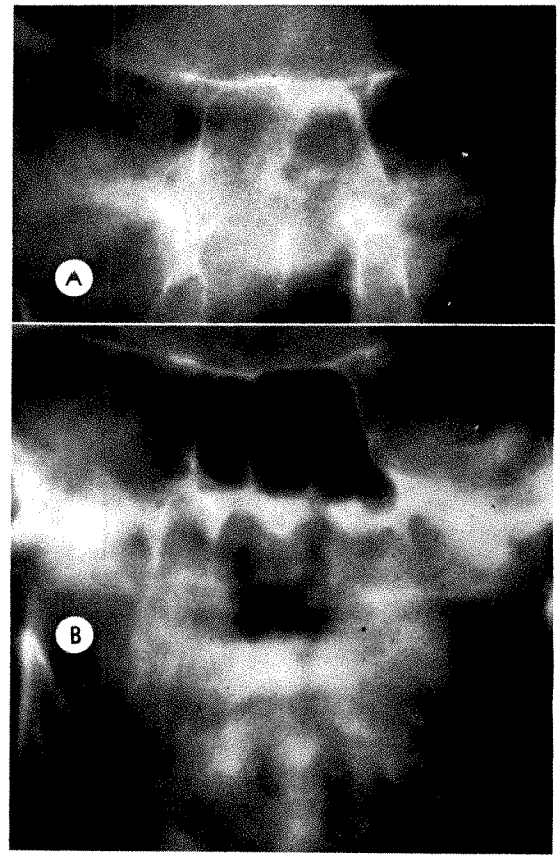



Fig. 4. *1967:* (*A*) Pluridirectional laminagram. Coronal section showing a large soft tissue mass completely filling the sphenoid sinus with destruction of its floor. (*B*) Appearance of the sphenoid sinus at this level in a normal control.

### ROENTGENOGRAPHIC MANIFESTATIONS

The nose is most often affected. The nasal septum may be irregularly thickened and deviated and the site of soft tissue masses which occlude the nares. The paranasal sinuses become opaque and often contain fluid levels. In advanced cases, bone destruction may occur and thus simulate a malignant process.[14,24] Shaw and Martin[25] described a case in which the disease had invaded the orbits and caused exophthalmos and blindness. In a patient reported by Hara and Lonser[11] an erroneous diagnosis of a meningioma was made because of erosion of the sphenoid wing. At autopsy, a 4×3×3 cm. mass in the middle fossa was found. Other masses extended in continuity into the orbit, floor of the nose and ethmoid sinus. Since pyogenic bacteria are frequently present, one cannot be certain whether the bone destruction is a result of the primary disease or of secondary infection.

When the granulomatous masses involve

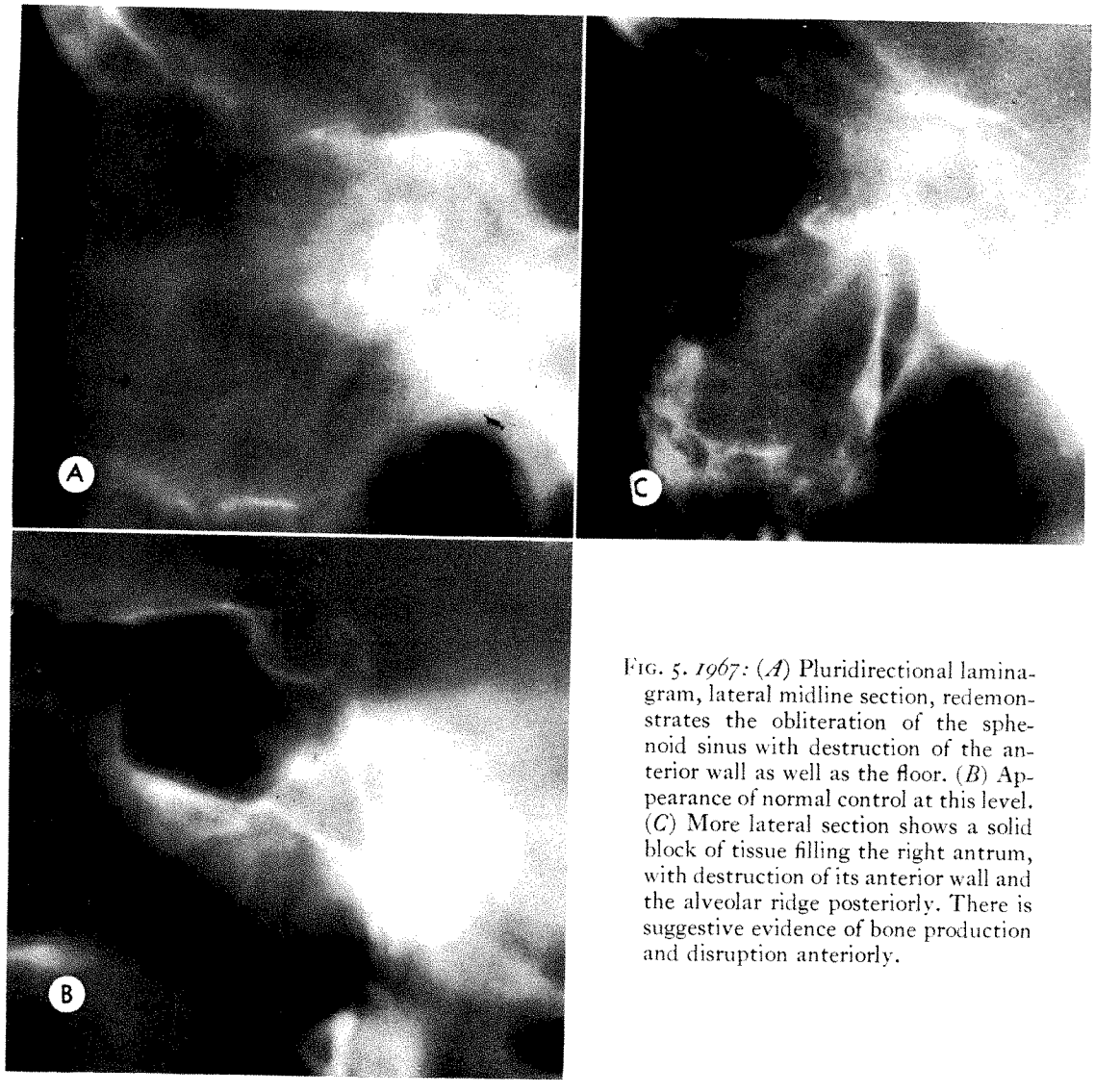



FIG. 5. *1967:* (*A*) Pluridirectional laminagram, lateral midline section, redemonstrates the obliteration of the sphenoid sinus with destruction of the anterior wall as well as the floor. (*B*) Appearance of normal control at this level. (*C*) More lateral section shows a solid block of tissue filling the right antrum, with destruction of its anterior wall and the alveolar ridge posteriorly. There is suggestive evidence of bone production and disruption anteriorly.

the palate and epiglottis or extend into the nasopharynx from the nose, this may be evident on lateral roentgenograms of the face and neck.

Laryngotracheal involvement, either primary or due to extensions from a focus in the nose, may be evident on chest roentgenography. The usual site of laryngeal involvement is the subglottic area, a region frequently difficult to visualize by laryngoscopy. Contrast laryngography may be rewarding in these cases.[7] Tracheal involvement is usually manifested by a uniform prominence of the mucosa which produces concentric narrowing of the tracheal air space. This may simulate thickening of the prevertebral tissue on lateral roentgenography. Discrete nodular lesions also occur with the appearance of one or more tumor like masses. With severe narrowing, endoscopy may be impossible making roentgenography particularly important. In advanced cases, the disease must be differentiated from intrinsic or extensive neoplastic lesions. Bronchial involvement with obstruction of large bronchi has been reported.[22]

## SUMMARY

Two cases of scleroma, one of which oc-

curred in a native born American, are presented with a discussion of the characteristic histologic, clinical and roentgen features.

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# ALTERATIONS OF THE SPHENOIDAL FISSURE PRODUCED BY LOCAL AND SYSTEMIC PROCESSES*

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EROSION of the margins of the sphenoidal (superior orbital) fissure is a valuable diagnostic sign of regional intracranial or intraorbital disease. While most reports stress the role of vascular lesions and hypophyseal tumors in the production of widening of the fissure, the differential diagnosis of these and other lesions has not received sufficient emphasis.[2,4,15] Furthermore, narrowing of the sphenoidal fissure as a manifestation of systemic as well as local disease has received little or no consideration. As a background for understanding developmental and pathologic conditions affecting the fissure, the authors have reviewed pertinent embryogenic and structural anatomic features.

## EMBRYOLOGY

The lesser sphenoidal wing and that portion of the greater wing bordering the sphenoidal fissure develop from the primitive mesenchyme which undergoes chondrification prior to actual ossification. At 9 to 12 weeks of fetal life, bone deposition has not occurred, as yet, around the well developed neural structures in the fissure (Fig. 1). However, by 20 weeks, there is well-defined ossification of the greater and lesser sphenoidal wings and of the optic strut, separating the rostral portion of the sphenoidal fissure from the optic foramen (Fig. 2). Ossification proceeds progressively throughout the lesser and greater sphenoidal wings so that in the mature fetus, these structures are predominantly ossified, although islands of residual unossified mesenchyme may still be seen (Fig. 3).

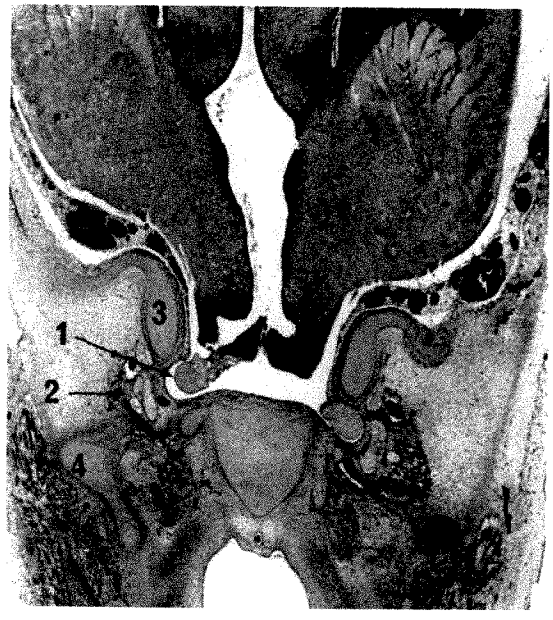



FIG. 1. Section through plane of forebrain and sphenoid in a 9 week old fetus showing embryonal cartilaginous matrix surrounding vascular and neural structures within the anlage of the sphenoidal fissure. At this age, bone formation has not yet occurred. (1) Optic nerve; (2) neurovascular structures within sphenoidal fissure; (3) embryonal cartilaginous matrix of lesser sphenoidal wing; (4) embryonal cartilaginous matrix of greater sphenoidal wing. (Paraffin embedded section, modified Masson stain.)

## ANATOMY

The sphenoidal fissure is a curvilinear aperture leading from the middle fossa to the orbit, bounded by the lesser sphenoidal wing superiorly, the greater sphenoidal wing inferiorly, the body of the sphenoid medially, and the orbital plate of the frontal bone laterally. Through this aper-

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ture pass the third, fourth, and sixth nerves, the ophthalmic division of the fifth cranial nerve, and some sympathetic fibers from the carotid artery. The ophthalmic veins, the orbital branches of the middle meningeal artery, and the recurrent branch of the lacrimal artery also traverse the fissure. The fissure lies inferolateral to the optic foramen from which it is separated by a strut of bone projecting from the lesser sphenoidal wing (Fig. 4, *A* and *B*; and 5, *A* and *B*). The foramen rotundum lies below the medial end of the fissure.[19]

The configuration of the normal sphenoidal fissure is subject to variability in size, shape and symmetry. The fissure is best demonstrated in the Caldwell and optic foramen projections. From a roentgenographic standpoint, the normal fissure is characterized by its discrete, well-defined cortical margins.

## NORMAL VARIANTS AND CONGENITAL ANOMALIES

In a study of 157 anatomic specimens, Kornblum and Kennedy[10] reported asymmetric sphenoidal fissures in approximately 9 per cent (Fig. 6); there were also significant symmetric variations in shape. The most common configuration noted was

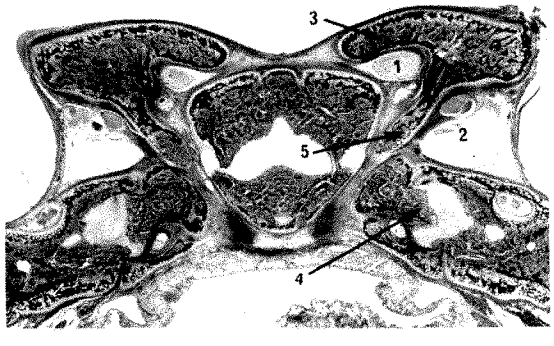



Fig. 2. Coronal section in a 20 week old fetus through plane of optic foramen showing the optic nerve (1) and neurovascular structures (2) within the sphenoidal fissure. Note the well defined ossification of the lesser (3) and greater (4) sphenoidal wings and the optic strut (5). The darker areas represent a more advanced stage of ossification. (Celloidin embedded section, modified Masson stain.)

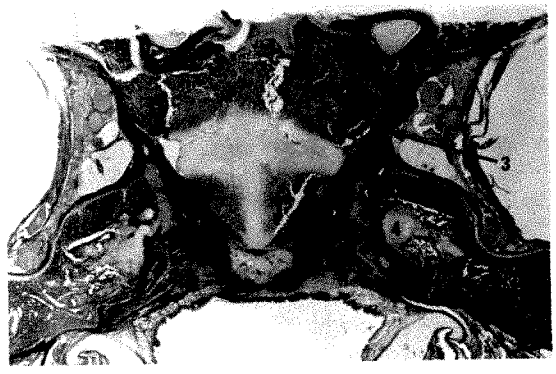



Fig. 3. Coronal section through plane of optic foramen and sphenoidal fissure in a 36 week old fetus. Note the relatively complete state of ossification of the lesser (1) and greater (2) sphenoidal wings and optic strut (3). There are a few remaining islands of unossified embryonal cartilage in the greater sphenoidal wings (4). (Celloidin embedded section, modified Masson stain.)




Fig. 4. Coronal section of adult skull through the planum sphenoidale, optic foramina and sphenoidal fissures. (*A*) A general view of both sides for orientation and (*B*) an enlargement of the left side for better detail. (1) Planum sphenoidale; (2) optic nerve; (3) sphenoidal fissure containing the third, fourth and sixth cranial nerves, the ophthalmic division of the fifth cranial nerve; the anterior portion of the cavernous sinus (4) lies medial to the aforementioned neural structures; (5) sphenoidal sinus; (6) optic strut. (Paraffin embedded section, hematoxylin-eosin stain.)

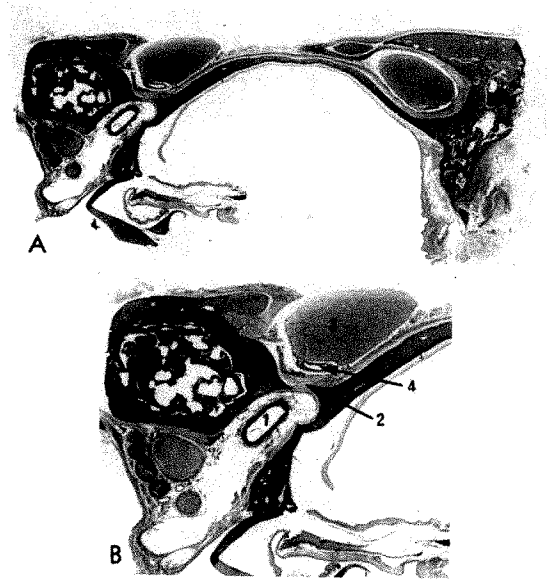



FIG. 5. Slightly more posterior coronal section of adult skull through the posterior rim of the optic foramen showing the relationship of the internal carotid artery (1) to the sphenoidal fissure. (*A*) A general view of both sides and (*B*) an enlargement of the left side for detail. Note that the most anterior extent of the internal carotid artery projects into the superior margin of the sphenoidal fissure. This relationship is important in the development of the characteristic bone erosion found with intracavernous aneurysms of the internal carotid artery and carotid-cavernous fistulas. In this plane, the bony strut from the lesser sphenoidal wing is incomplete (2) and periosteum separates the optic nerve (3) and ophthalmic artery (4) from the internal carotid artery. (Paraffin embedded section, hematoxylin-eosin stain.)

narrow at the upper anterior border and bulbous at the lower medial end (56 per cent). Other configurations included a




FIG. 6. Caldwell projection of adult dry skull demonstrating asymmetry of the sphenoidal fissures. Note the well defined cortical margins, the hallmark of the normal fissure.

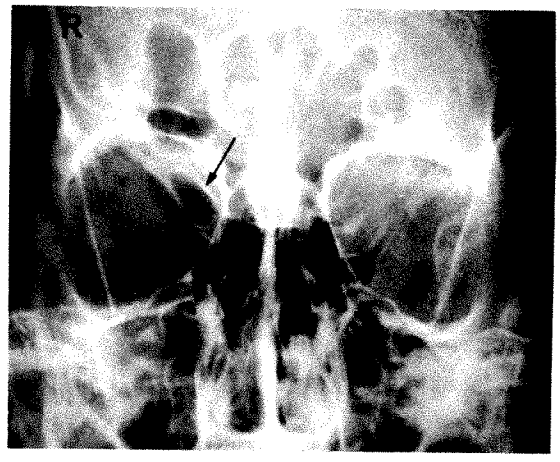



FIG. 7. Asymmetry of the sphenoidal fissures and the lesser sphenoidal wings due to ectopic ethmoidal cell formation (arrow) in the right lesser sphenoidal wing of a 26 year old healthy person.

long, narrow fissure (12 per cent), a dumbbell shape (12 per cent), and a cone appearance (11 per cent). The present authors have observed significant asymmetry of the sphenoidal fissure, produced by unilateral pneumatization of the lesser sphenoidal wing by aberrant ethmoidal cells (Fig. 7). Rarely, the fissure may communicate with the foramen rotundum because of incomplete ossification of the chondral root of the greater sphenoidal wing and failure of its anterior portion to fuse with the postsphenoid[11] (Fig. 8). Indeed, this anomaly represents the normal state prior to the 12th week of fetal life (foramen lacerum anterius). LeDouble[11] has described communication of the sphenoidal fissure and the optic foramen presumably

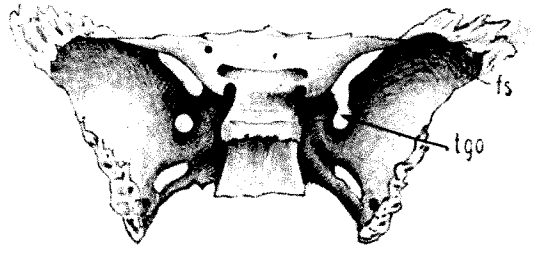



FIG. 8. Photograph of drawing from LeDouble[11] showing unilateral communication of the foramen rotundum with the sphenoidal fissure (arrow).

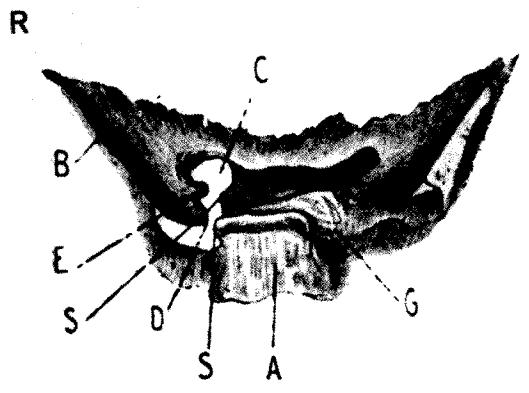



Fig. 9. Photograph of drawing from LeDouble[11] demonstrating communication of the right optic foramen with the sphenoidal fissure due to incomplete formation of the optic strut.

due to failure of development of the optic strut from the lesser sphenoidal wing (Fig. 9).

## PATHOLOGY

### 1. WIDENING OF THE SPHENOIDAL FISSURE

A. *Vascular lesions.* The first noteworthy contribution to the roentgenology of aneurysms of the internal carotid artery and the circle of Willis was made by Sosman and Vogt[22] in 1926. These authors summarized the roentgenologic findings in the Cushing series, pointing out the significance of calcification in the aneurysmal wall, sharply defined erosion of an anterior or a posterior clinoid process and of the lateral half of the body of the sphenoid bone. No specific reference was made to changes in the sphenoidal fissure. The roentgenologic criteria for the diagnosis of aneurysm of the intracranial portion of the internal carotid artery were detailed further by McKinney, Acree, and Stoltz[13] in 1936, based upon observations of 8 patients with "unruptured" aneurysm. Calcification, erosion of the carotid canal, enlargement or unilateral erosion of the sella turcica, optic foramen, and sphenoidal fissure were emphasized as significant features of this lesion. Erosion of the ventrolateral wall of the optic foramen resulting in coalescence of the optic canal and the sphenoidal fissure was also observed. Since several of their patients exhibited orbital bruit, exophthalmos, and papilledema, and postmortem studies were not available, it would seem likely that some had arteriovenous fistulas secondary to spontaneous rupture of the aneurysm into the cavernous sinus.

Jefferson[7] noted evidence of the vascular lesion on the plain skull roentgenograms in 10 of 16 patients with saccular aneurysm of the intracavernous portion of the internal carotid artery, and regarded calcification in the wall of the aneurysm as the most reliable diagnostic roentgenologic sign. Enlargement of the sphenoidal fissure and erosion of the inferior lateral wall of the optic canal seen in 1 patient were considered important criteria in differentiating aneurysm from a primary intrasellar tumor. According to Jefferson, enlarging aneurysms in the anterior portion of the cavernous sinus tend to impinge upon the sphenoidal fissure and erode the contiguous margins of the sphenoidal wings. In particular, the ipsilateral lesser wing and the anterior clinoid process are apt to be elevated, and the superior margin of the sphenoidal fissure and the optic strut destroyed. Interestingly enough, Jefferson also described a patient with a sizable intracavernous aneurysm and total ophthalmoplegia in whom no bone erosion was observed.

Rischbieth and Bull[15] also studied the effects of internal carotid artery aneurysm and carotid-cavernous fistula upon the sphenoidal fissure. In a series of 23 patients with intracavernous aneurysm proven by arteriography, erosion of the greater sphenoidal wing and widening of the sphenoidal fissure were the most common roentgenographic findings encountered, being present in 17 instances. Erosion of the inferior margin of the lesser sphenoidal wing, erosion of the inferolateral wall of the optic canal, and aneurysmal calcification were each present in approximately one-third of the patients. According to Rischbieth and Bull, bone erosion becomes evident only if the aneurysm fills the entire caver-

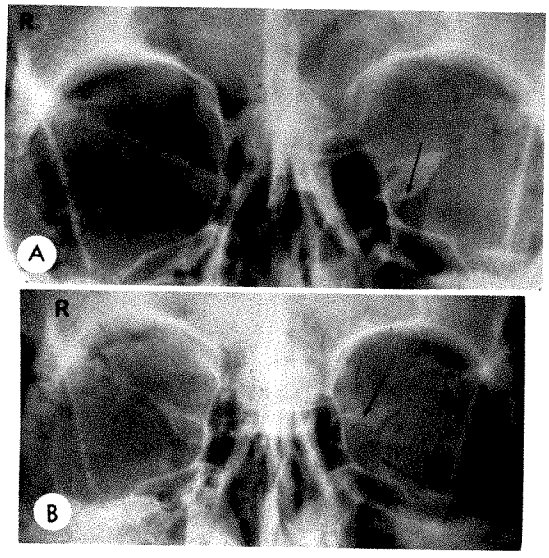



FIG. 10. (*A*) Early erosion of the medial superior and inferior margins of the left sphenoidal fissure (arrow) by an intracavernous aneurysm of the internal carotid artery. (*B*) Progression of the bone changes 6 years later showing the sharply defined defect characteristic of arterial pulsation on bone.

nous sinus. In a group of 3 patients with spontaneous carotid-cavernous fistula, they found 2 patients with symptoms of 5 and 8 weeks' duration, respectively, with no bone erosion and 1 patient with demonstrable widening of the sphenoidal fissure 8 months after the onset of symptoms. In this regard, it is remarkable that Ruggiero and Castellano[16] noted enlargement of the ipsilateral sphenoidal fissure in only 1 of 11 patients with carotid-cavernous fistula.

From their personal experience, the present authors believe that:

1. In the presence of a normal sella turcica, a sharply defined, concave

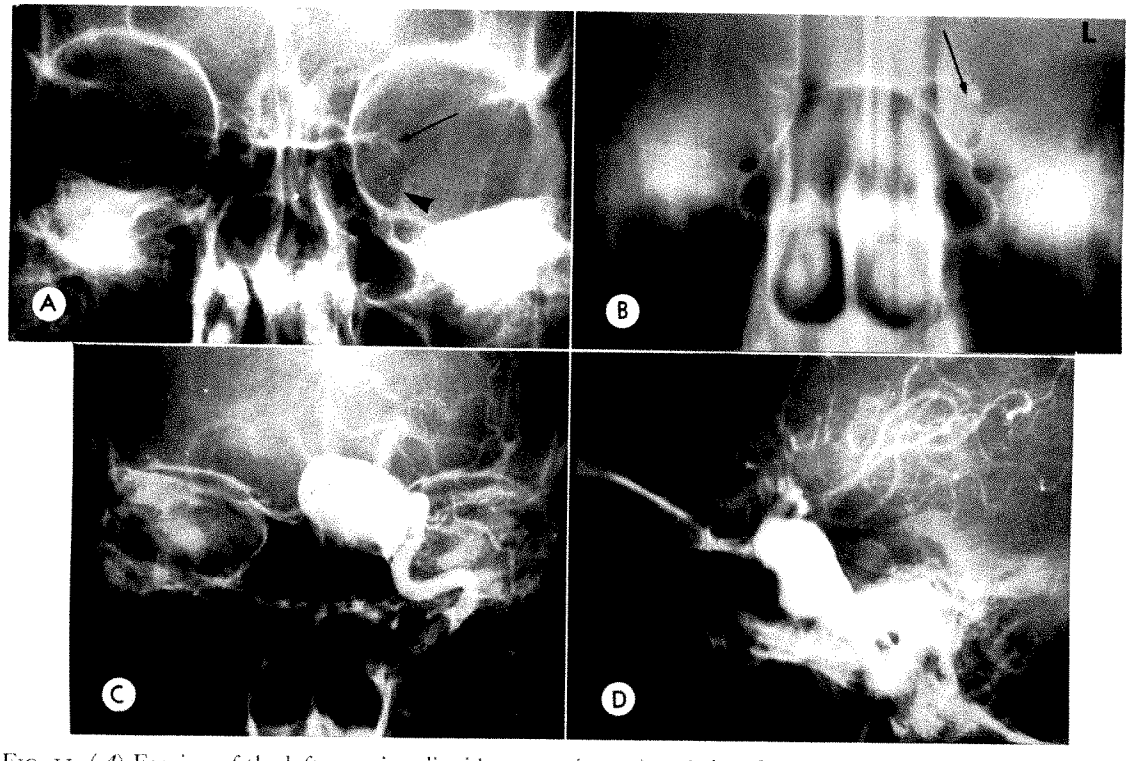



FIG. 11. (*A*) Erosion of the left anterior clinoid process (arrow) and sharply defined scalloping of the inferior medial aspect of the sphenoidal fissure (arrowhead) in a middle-aged female with a large intracavernous aneurysm of the internal carotid artery. (*B*) The laminagraphic section shows the erosion of the anterior clinoid process (arrow) to better advantage. (*C*) The anteroposterior and (*D*) lateral carotid arteriograms demonstrate the intracavernous position of the large aneurysm. Note the incompletely filled anterior inferior portion of the aneurysm probably due to blood clot.

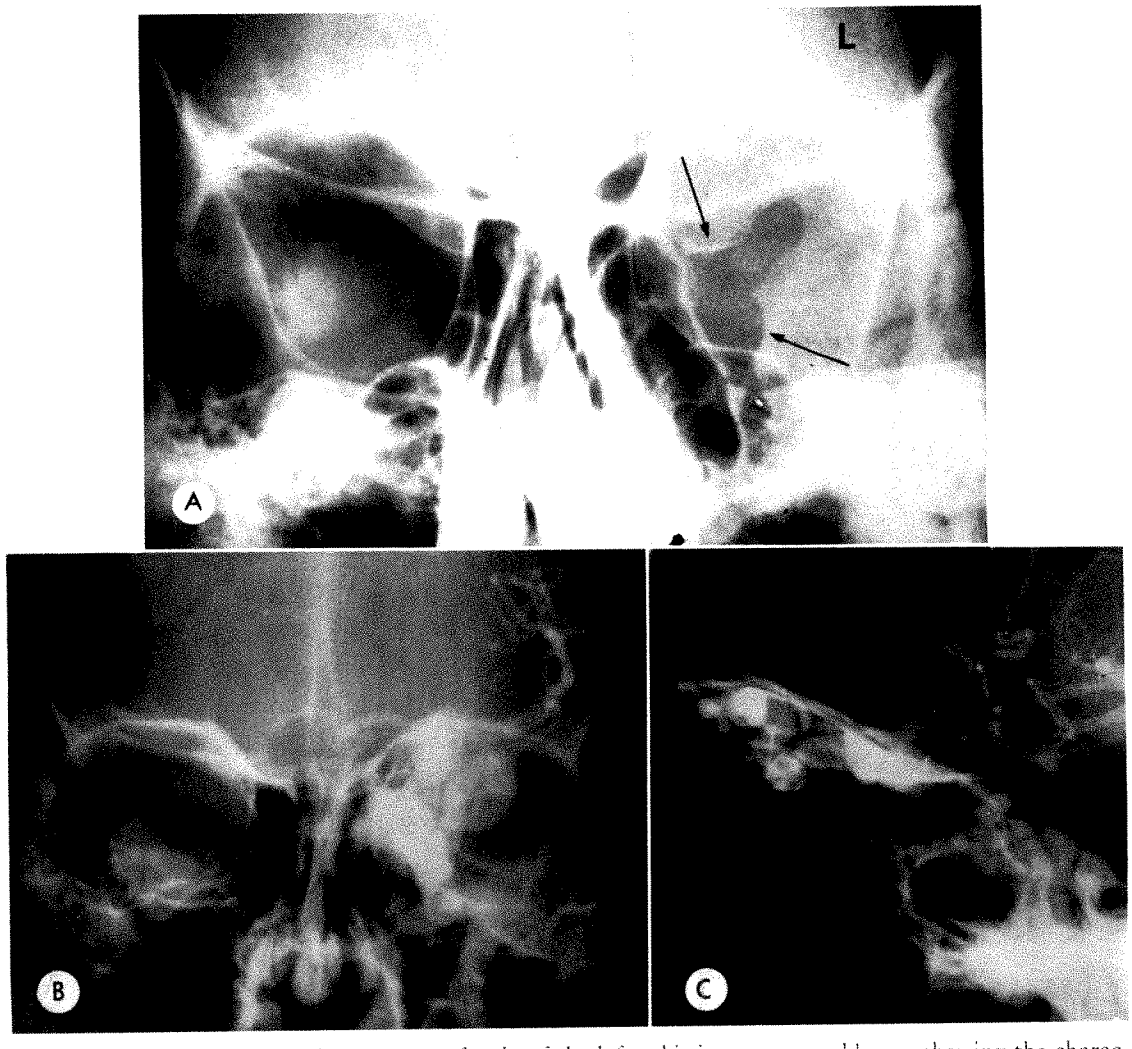



FIG. 12. (*A*) Traumatic carotid-cavernous fistula of the left orbit in a 30 year old man showing the characteristic discrete notching of the superior and inferior medial margins of the sphenoidal fissure (arrows). (*B* and *C*) Left carotid arteriograms demonstrate the arteriovenous fistula with filling of the ophthalmic veins.

erosion of the superior or inferior medial aspect of the sphenoidal fissure strongly suggests either an intracavernous aneurysm of the internal carotid artery (Fig. 10, *A* and *B*; and 11, *A–D*) or a carotid-cavernous fistula (Fig. 12, *A–C*). In the normal patient, the medial superior margin of the sphenoidal fissure is separated from the optic foramen by the well defined, osseous optic strut.

2. Aneurysms of the internal carotid artery confined to the posterior half of the cavernous sinus do not produce erosion of the sphenoidal fissure. On the other hand, aneurysms in the anterior half of the cavernous sinus (*i.e.* involving the anterior end of the carotid siphon where the artery normally grooves the sphenoid bone as it curves up to pierce the dura) tend to erode that portion of the greater sphenoidal wing which forms the lower margin of the sphenoidal fissure.
3. The characteristic bone erosion involving the medial margins of the sphenoidal fissure bears a relationship to the duration of the aneurysm and,

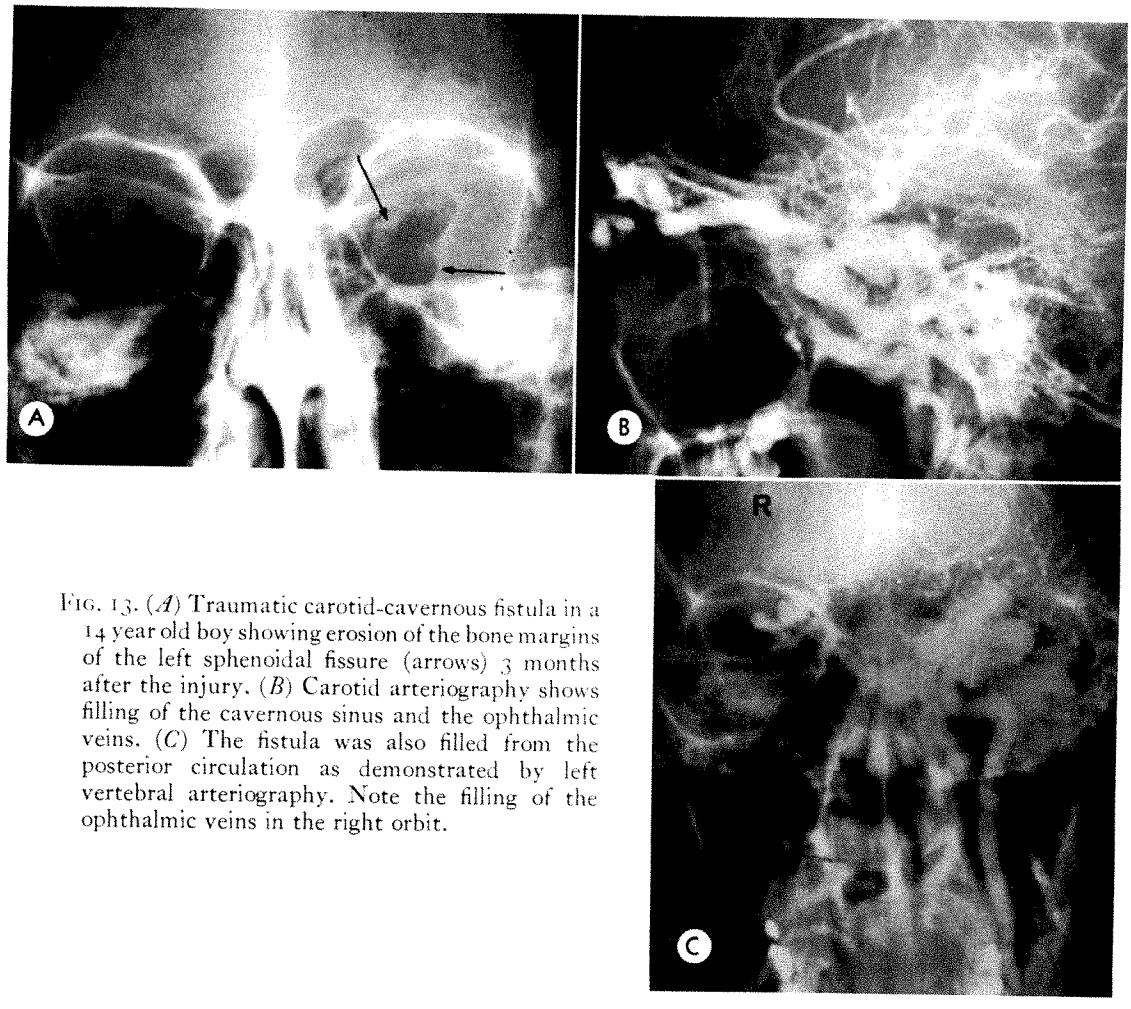



FIG. 13. (*A*) Traumatic carotid-cavernous fistula in a 14 year old boy showing erosion of the bone margins of the left sphenoidal fissure (arrows) 3 months after the injury. (*B*) Carotid arteriography shows filling of the cavernous sinus and the ophthalmic veins. (*C*) The fistula was also filled from the posterior circulation as demonstrated by left vertebral arteriography. Note the filling of the ophthalmic veins in the right orbit.

more particularly, the carotid-cavernous fistula. In this regard, it is pertinent that the authors have noted considerable bone erosion as soon as 3 months following injury in a 14 year old boy with a traumatic carotid-cavernous fistula (Fig. 13, *A–C*).

B. *Pituitary tumors*. Most pituitary tumors do not extend beyond the bony sella turcica and the immediate suprasellar region. Dott *et al.*[3] and Jefferson[8] encountered major extrasellar extensions in only approximately 14 per cent of their cases of pituitary adenoma. The common extrasellar extensions were in the direction of the third ventricle, frontal, temporal and hypothalamic regions, and the cavernous sinus. Assessment of the extent of growth in all but the last situation can be more accurately achieved by pneumoencephalography than from the clinical findings or the plain skull roentgenograms. The precise incidence of lateral extension of pituitary tumors into the cavernous sinus is difficult to determine since much depends upon the diagnostic criteria employed. While cavernous sinus extension occurred in approximately 5 per cent of pituitary tumors in Jefferson's experience, Weinberger *et al.*[23] reported an incidence of such occurrence in 12 per cent of the University of Pennsylvania series. With regard to the onset and progression of symptoms in patients with cavernous sinus extension, it should be appreciated that the anatomic relation-

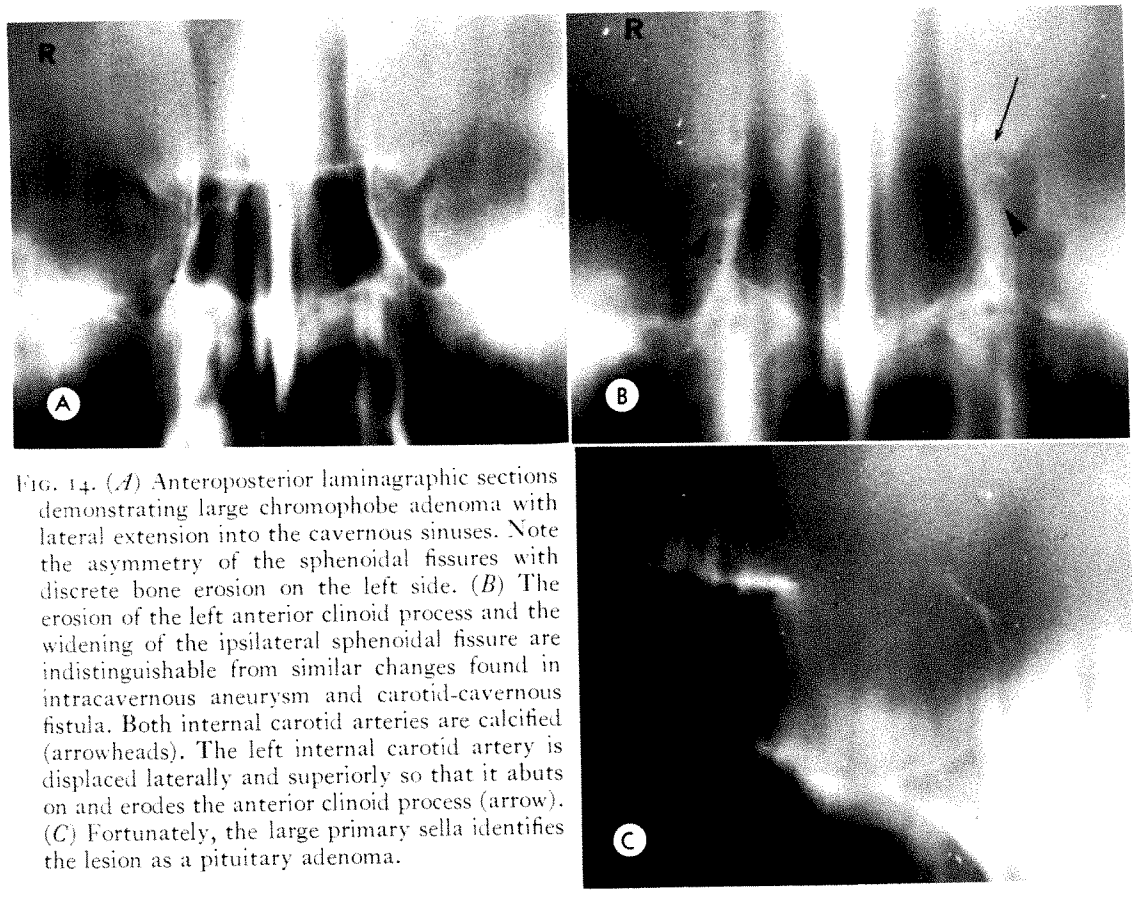



FIG. 14. (*A*) Anteroposterior laminagraphic sections demonstrating large chromophobe adenoma with lateral extension into the cavernous sinuses. Note the asymmetry of the sphenoidal fissures with discrete bone erosion on the left side. (*B*) The erosion of the left anterior clinoid process and the widening of the ipsilateral sphenoidal fissure are indistinguishable from similar changes found in intracavernous aneurysm and carotid-cavernous fistula. Both internal carotid arteries are calcified (arrowheads). The left internal carotid artery is displaced laterally and superiorly so that it abuts on and erodes the anterior clinoid process (arrow). (*C*) Fortunately, the large primary sella identifies the lesion as a pituitary adenoma.

ships of the internal carotid artery and the extraocular motor nerves (III, IV and VI) are subject to variation. According to Sluder,[20,21] the third cranial nerve, which usually lies high in the sinus, occasionally occupies a lower position, whereas the sixth cranial nerve, which ordinarily lies more inferiorly and laterally, may pursue a superior course alongside the fourth cranial nerve. The position and configuration of the loop of the internal carotid artery as it exits from the foramen lacerum may also vary.[17]

Gross extension of a pituitary tumor into the cavernous sinus does not, of itself, imply malignancy although this impression may be gleaned from the literature since tumors exhibiting such growth have been designated by some as "malignant adenomas."[8] Most commonly, the pituitary tumor, a benign chromophobe adenoma, extends laterally into the cavernous sinus, displacing and surrounding the neurovascular structures within the sinus without histologic evidence of invasion or destruction of these structures or metastases to distant organs.[24] In contradistinction to intracavernous growth of the benign adenoma, the less common hypophyseal carcinoma demonstrates actual infiltration of the blood vessel walls, connective tissue, neural structures, and, at times, the adjacent bone.[1,5,9]

Pituitary adenomas which grow into the cavernous sinus do not widen the sphenoidal fissure unless the extension is anterior as well as lateral. Hence, all patients with cavernous sinus invasion do not necessarily exhibit erosion of the bone margins of the sphenoidal fissure. When present, the erosion may be unilateral or bilateral. In the latter instance, differentia-

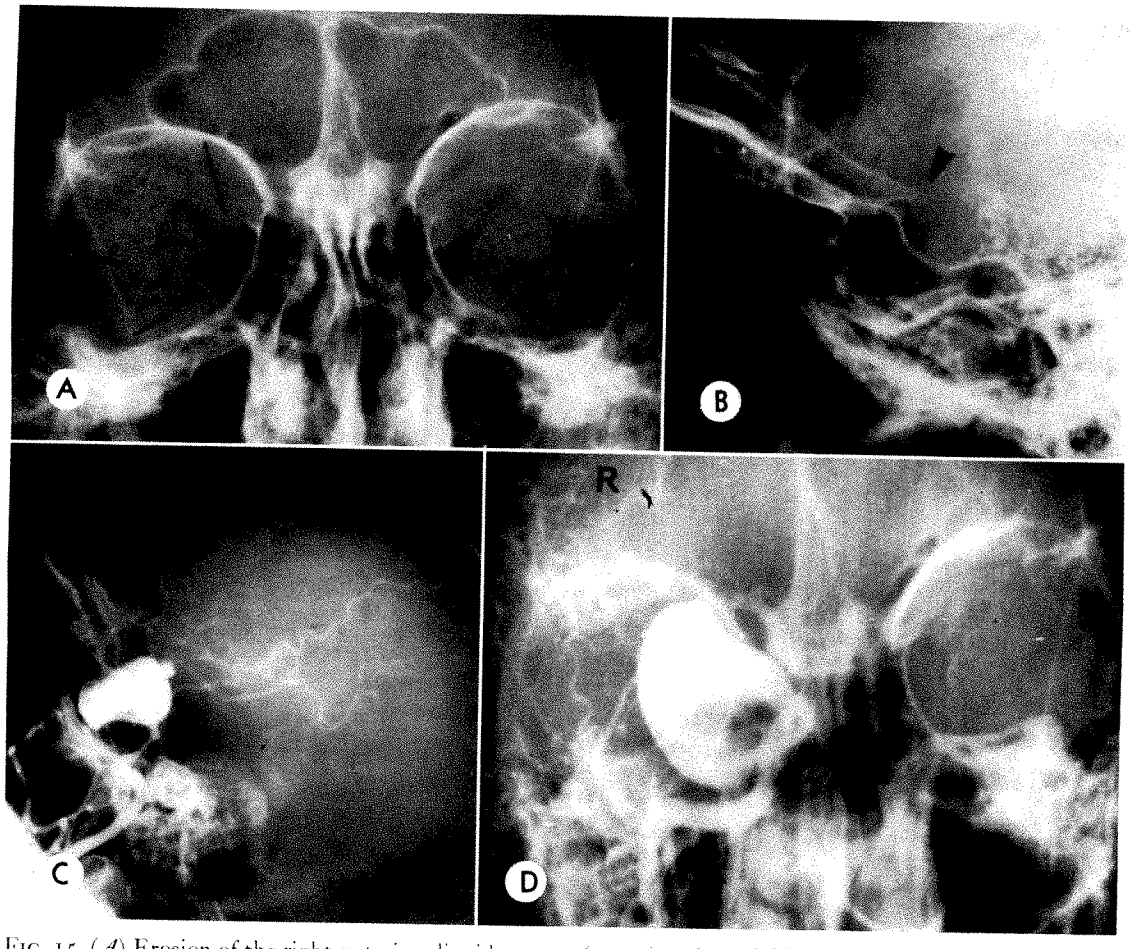



FIG. 15. (*A*) Erosion of the right anterior clinoid process (arrow) and medial inferior margin of the sphenoidal fissure (arrow) by a large intracavernous aneurysm. (*B*) Note the normal size of the sella turcica in the presence of sharpening of the right anterior clinoid process (arrowhead). This serves to differentiate an extrasellar vascular lesion from a primary intrasellar tumor. (*C*) Carotid arteriography demonstrates the relationship of the intracavernous aneurysm to the ipsilateral anterior clinoid process and (*D*) sphenoidal fissure. The incomplete filling of the floor of the medial aspect of the aneurysm is probably due to clot formation.

tion from aneurysm presents no problem. When unilateral erosion is present, it may be sharply defined and resemble that produced by intracavernous aneurysm or carotid-cavernous fistula (Fig. 14*A*). An explanation for this similarity was originally suggested by Twining[8] and is concurred in by the present authors. As it grows laterally and anteriorly beyond the confines of the sella turcica, the tumor displaces the internal carotid artery so that the vessel abuts against the ipsilateral anterior clinoid process and inferior edge of the sphenoidal fissure (Fig. 14*B*). Fortunately, in most instances, patients with a pituitary adenoma also exhibit the roentgenographic features characteristic of a "primary sella," thereby permitting differentiation from the usual aneurysm (Fig. 14*C*; and 15, *A–D*). In doubtful cases, carotid arteriography further serves to distinguish aneurysm from pituitary tumor.

C. *Meningioma.* The vast majority of middle fossa meningiomas produce sclerosis of the ipsilateral sphenoidal wing with either no change or narrowing of the sphenoidal fissure. However, localized erosion of the sphenoidal wings and widening of

the fissure occur infrequently with meningioma (Fig. 16). In Rischbieth and Bull's observations on the McKissock series of 192 middle fossa meningiomas, there were only 4 patients with widening of the fissure. However, in 3 of these, there were additional diagnostic features of meningioma; *i.e.*, sclerosis, compression of the optic canal, destruction of the floor of the middle fossa, and calcification within the tumor. Meningiomas do not usually produce the discrete, sharply defined bone erosion found with intracavernous aneurysm or carotid-cavernous fistula.

D. *Orbital tumors.* Neoplasms involving the orbit may be primary or metastatic and either group of tumors may produce bone destruction with resultant widening of the sphenoidal fissure. The osseous destruction usually involves more than the immediate margins of the fissure and, at times, is quite extensive.

Most primary intraorbital tumors are non-neurogenic in origin;[6,25] such diverse benign lesions as hemangioma, dermoid cyst, lipoma, and pseudotumor (a granuloma and not a true neoplasm) are found in this group (Fig. 17). The most common primary malignant orbital tumor is lymphosarcoma. Neurolemmomas of the extraocular motor nerves which pass through the sphenoidal fissure are usually associated with neurofibromatosis (Fig. 18). In the authors' experience, the erosion of the sphenoidal fissure associated with these lesions does not present the sharply defined, delicately etched margins so characteristically found with aneurysm or carotid-cavernous fistula.

Metastatic carcinoma, sarcoma or lymphoma may also produce osteolytic lesions of the bony orbit and widen the sphenoidal fissure. Usually, these lesions produce somewhat irregular margins, are rather

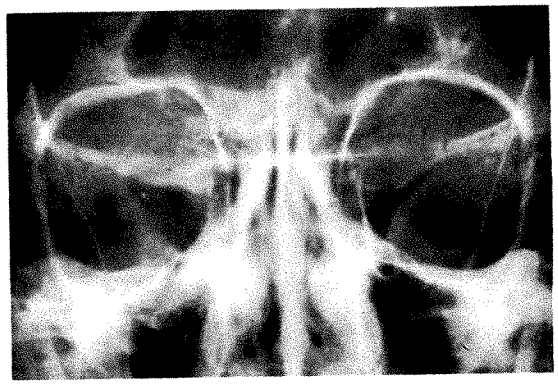


Fig. 17. Granuloma of unknown etiology involving the right orbit and contiguous dura in a 47 year old male. Note the widening of the ipsilateral sphenoidal fissure. Roentgenograms made 18 years previously demonstrated normal sphenoidal fissures bilaterally.

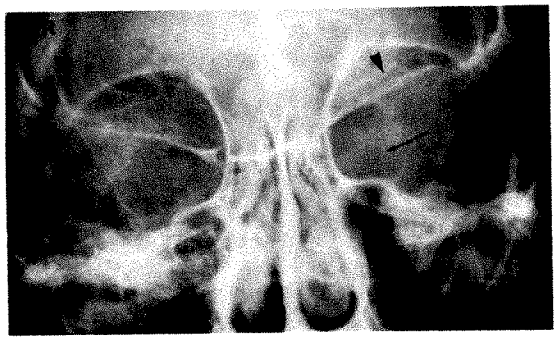


Fig. 16. Large meningioma arising from the inner third of the left lesser sphenoidal wing in a middle-aged female. Note the elevation of the lesser wing (arrowhead) and the extensive bone erosion producing marked widening of the sphenoidal fissure (arrow).

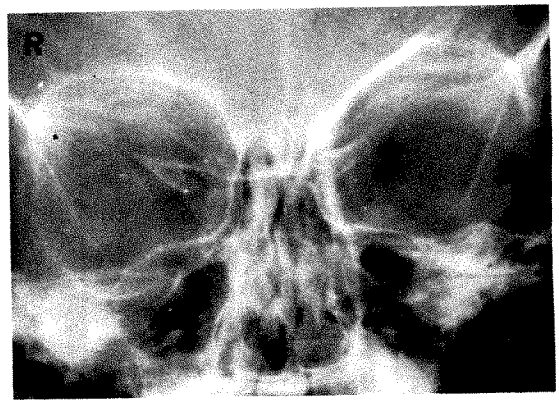



Fig. 18. Elevation of the left lesser sphenoidal wing and widening of the sphenoidal fissure in an 8 year old boy with neurofibromatosis. The widening of the fissure is due to neurolemmomas involving the extraocular motor nerves. Note that the bone margins of the dilated fissure are not so sharply defined as they are in vascular lesions.




FIG. 19. Extensive bone destruction of the right orbit due to metastatic carcinoma.

extensive, and present no appreciable difficulty in differential diagnosis (Fig. 19). Occasionally, chordomas may extend into the middle fossa to invade the sphenoidal wings and produce widening of the sphenoidal fissure. However, these patients frequently exhibit destruction of the base of the skull and the middle fossa.

E. *Reticuloendotheliosis.* Hand-Schüller-Christian's disease or Letterer-Siwe's disease may also involve the bony orbit and produce widening of the sphenoidal fissure (Fig. 20). Although the margins of the involved bone may be sharply defined, the systemic nature of the disease offers no difficulty in differential diagnosis.

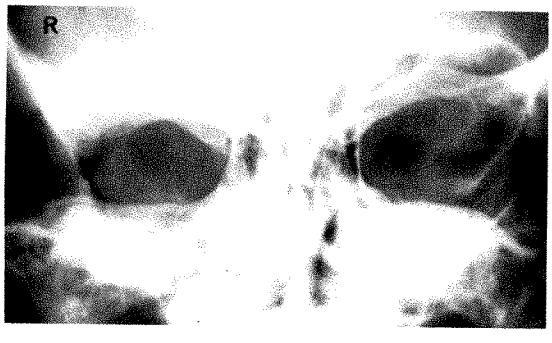



FIG. 20. Marked widening of the right sphenoidal fissure due to involvement of the orbit by Hand-Schüller-Christian's disease in a 5 year old girl. There were also large bilateral osteolytic lesions in the temporal and sphenoidal bones as well as a similar smaller lesion in the left mandible.

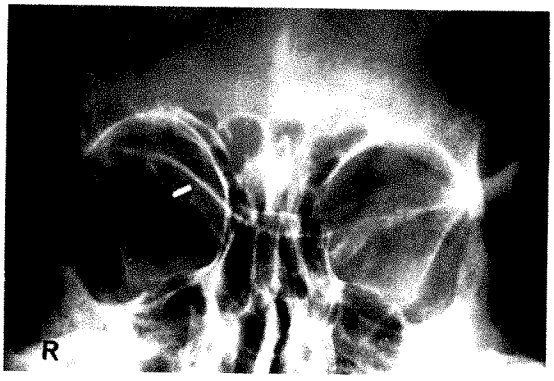



FIG. 21. Postoperative roentgenogram in the Caldwell projection showing elevation of the right lesser sphenoidal wing and enlargement of the ipsilateral sphenoidal fissure in a 39 year old female with a large right middle fossa arachnoid cyst associated with temporal lobe agenesis. The base view clearly demonstrated bulging of the osseous boundaries of the entire right middle fossa.

F. *Other diseases within the middle fossa.* Agenesis of the temporal lobe associated with an arachnoidal fluid "cyst" (Fig. 21), localized dilatation of the temporal horn, chronic subdural hematoma, and an occasionally slow-growing astrocytoma of the temporal lobe may each produce widening of the sphenoidal fissure. The ballooning of

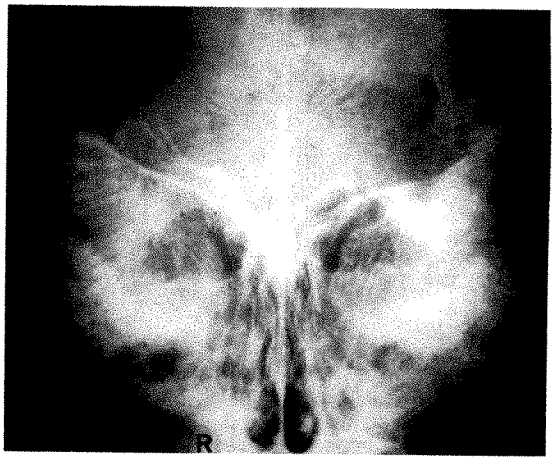



FIG. 22. Posteroanterior skull roentgenogram of an 8 year old girl with "leontiasis ossea" due to fibrous dysplasia. Note the narrowing of the right sphenoidal fissure as well as the extensive bone changes involving the entire calvarium and facial bones.

the involved middle fossa which is frequently associated with these lesions serves to differentiate this group from the preceding diagnostic categories.

Lombardi and his co-workers[12] have also reported widening of the sphenoidal fissure associated with mucocele of the sphenoid sinus.

II. NARROWING OF THE SPHENOIDAL FISSURE

A variety of diseases characterized by an overgrowth of bone may produce narrowing of the fissure.

A. *Fibrous dysplasia.* The sclerotic type of fibrous dysplasia may produce considerable thickening of the cranial vault, base or facial bones (Fig. 22). When the disease affects other portions of the skeleton, the diagnosis is relatively simple. However, localized fibrous dysplasia involving the lesser and greater sphenoidal wings may mimic the appearance of meningioma *en plaque*. Two important differential diagnostic features are the bizarre trabecular pattern and the disproportionate ground glass radiolucency in comparison with the over-all bony thickness.

B. *Paget's disease.* The thickened bone with a grossly abnormal mosaic, the widened diploë and the patchy, ill-defined areas of sclerosis and decreased density are characteristic of this disease.

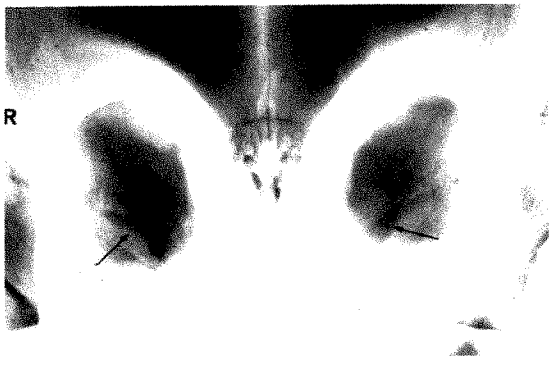



FIG. 23. Postmortem roentgenogram of the skull of a 32 month old girl showing marked slit-like narrowing of both sphenoidal fissures (arrows) by the abnormally dense bone. (Courtesy M. H. Wittenborg, M.D., Children's Hospital Medical Center, Boston, Mass.)



FIG. 24. Sclerosis and new bone formation in the medial half of the right lesser sphenoidal wing with slight narrowing of the sphenoidal fissure in a 42 year old female with a meningioma arising from the inner third of the lesser sphenoidal wing.

C. *Osteopetrosis.* This interesting abnormality, thought to be due to a congenital defect in bone resorption, usually involves the entire skeleton. The dense bone surrounding the sphenoidal fissure (and other cranial orifices) narrows the fissure and may result in neural compression (Fig. 23).

D. *Tumor.* Meningioma is the most common primary tumor which produces hyperostosis of the sphenoidal wings. The new bone, which may be very dense, frequently has fuzzy and ill-defined margins. Although the bony sclerosis may be extensive, narrowing of the sphenoidal fissure is relatively uncommon (Fig. 24).

Localized osteoblastic metastases, particularly from the breast and prostate, may uncommonly involve the sphenoidal wings and produce constriction of the sphenoidal fissure.[18] The metastatic lesions are rarely limited to the calvarium. The presence of similar sclerotic lesions in the facial bones, especially in elderly patients, should suggest the correct diagnosis.

E. *Thalassemia major (Mediterranean anemia).* The authors have observed a patient with thalassemia major with unilateral narrowing of the sphenoidal fissure

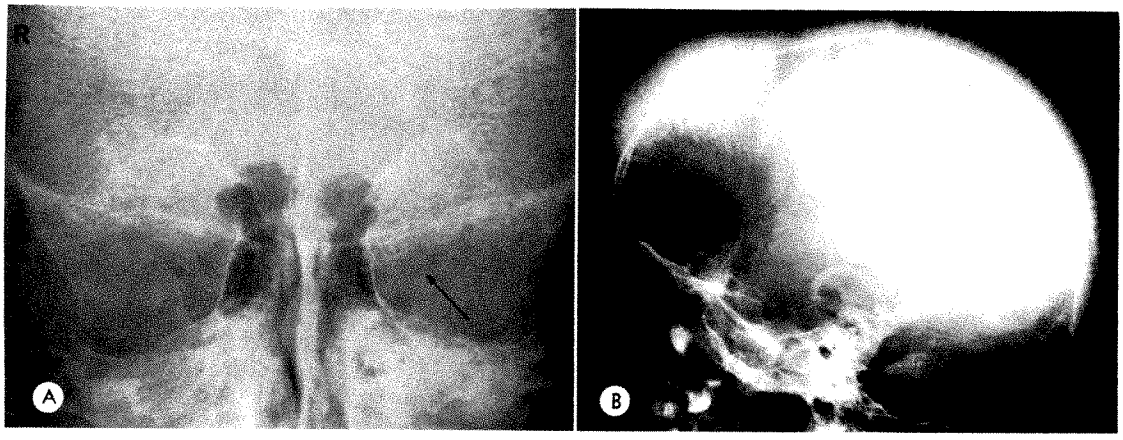



FIG. 25. (*A*) Caldwell projection demonstrating narrowing of the left sphenoidal fissure (arrow) in an 8 year old girl with thalassemia major. Roentgenograms of the skull were taken because of papilledema of the left fundus (? compression of ophthalmic veins by the expanded abnormal bone). Neurologic examination was otherwise entirely normal. (*B*) The lateral projection shows the typical "hair-on-end" appearance.

due to increased hematopoiesis and diploic expansion (Fig. 25, *A* and *B*).

## SUMMARY

1. The embryogenesis and anatomy of the sphenoidal fissure and adjacent structures are reviewed.

2. A discussion of the normal variations and congenital anomalies involving the fissure is presented.

3. Various pathologic processes producing dilatation and constriction of the fissure and their roentgen differential diagnosis are discussed in detail.

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# TOMOGRAPHY OF THE INNER EAR IN A CASE OF WAARDENBURG'S SYNDROME*

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IN 1951, Waardenburg[13] published the first extensive paper on a new syndrome, now commonly named after him.

The symptoms, listed in order of decreasing frequency, are, according to Waardenburg: (1) lateral displacement of the medial canthi and lacrimal points; (2) a hyperplastic, broad, high nasal root; (3) hyperplasia of the medial portions of the eyebrows; (4) partial or total heterochromia iridum; (5) congenital deafness or partial (unilateral) deafness; and (6) a white forelock.

Many other cases have been published in the intervening years, but in only 1 case so far was there an opportunity to make a histologic examination of the temporal bones in order to reveal the cause of the deafness.

In 1959, Fisch[3] reported the results of a histologic examination of the auditory pathway in a 3½ year old girl with the Waardenburg syndrome. This examination revealed an absence of the organ of Corti and atrophy of the spiral ganglion and nerve, but a fully developed osseous inner ear.

## THE INVESTIGATION

Included in a series of deaf children examined by tomography was a 15 year old boy with the Waardenburg syndrome. There were no cases of the syndrome in his family. The pregnancy and delivery were normal. His appearance is characteristic of the syndrome with a white forelock and depigmentation of the left eyebrow. The root of his nose is high and broad, and there is an increased distance between the medial canthi; furthermore, the patient was born deaf. His irides, however, have the same color (blue). The otologic examination revealed total deafness in both ears and the vestibular function was extinguished.

Tomography was performed with the Philips-Massiot Polytome using hypocycloid movements and 1 mm. cuts. The focal spot measured 0.3×0.3 mm., and the exposure time was 11.6 seconds (2 hypocycloid movements), allowing a kilovoltage of about 60 kvp. with 40 ma. No grid was used, but the Polytome was fitted with 3 corresponding diaphragms; one about the cone 11 cm. from the focus; a second at the lower end of the cone (the original); and a third just above the film. The size of each tomogram in the anteroposterior projection is 70 mm. in diameter and in the other projections 55 mm.

The projections used in this series of deaf children were the anteroposterior and the axial pyramidal. The combination of these 2 projections gives almost any required information regarding the inner ear,[5] with the exception of the posterior semicircular canal, the details of which do not visualize well. There was no sign of the posterior semicircular canal in the 2 projections; however, the examination was supplemented with cuts in the Stenvers projection, in which the canal normally is excellently outlined.

Figure 1, *A* and *B* shows the right ear in the anteroposterior projection. The lateral semicircular canal is noted as a protrusion from the vestibule without well defined crura. The vestibule is irregularly formed and no oval window can be seen. The superior semicircular canal is present but of a somewhat coarse shape. In Figure 2, *A* and *B*, which is an axial pyramidal view of the same ear, the cochlea is seen to be normal.

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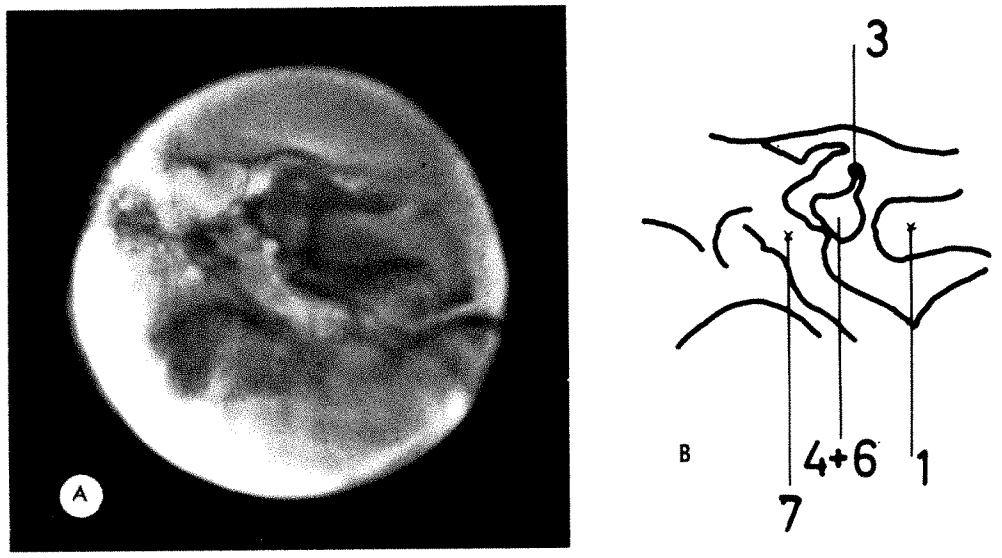



FIG. 1. (*A*) The patient's right ear in anteroposterior projection. The lateral semicircular canal is seen as a protrusion from the vestibule. There is absence of the oval window. (*B*) Sketch of *A*. 1. Internal auditory meatus; 2. cochlea; 3. superior semicircular canal; 4. lateral semicircular canal; 5. posterior semicircular canal; 6. the vestibule; 7. tympanic cavity. (This key applies to all subsequent figures.)

In the Stenvers projection (Fig. 3, *A* and *B*), there is no sign of the posterior semicircular canal, which can be seen in normal cases (Fig. 4, *A* and *B*).

Figure 5, *A* and *B* is an anteroposterior view of the left ear. The structures of the inner ear have a coarse appearance; the walls of the labyrinth are thickened, and the shape of the lateral and superior semicircular canal is not as slender as in normal

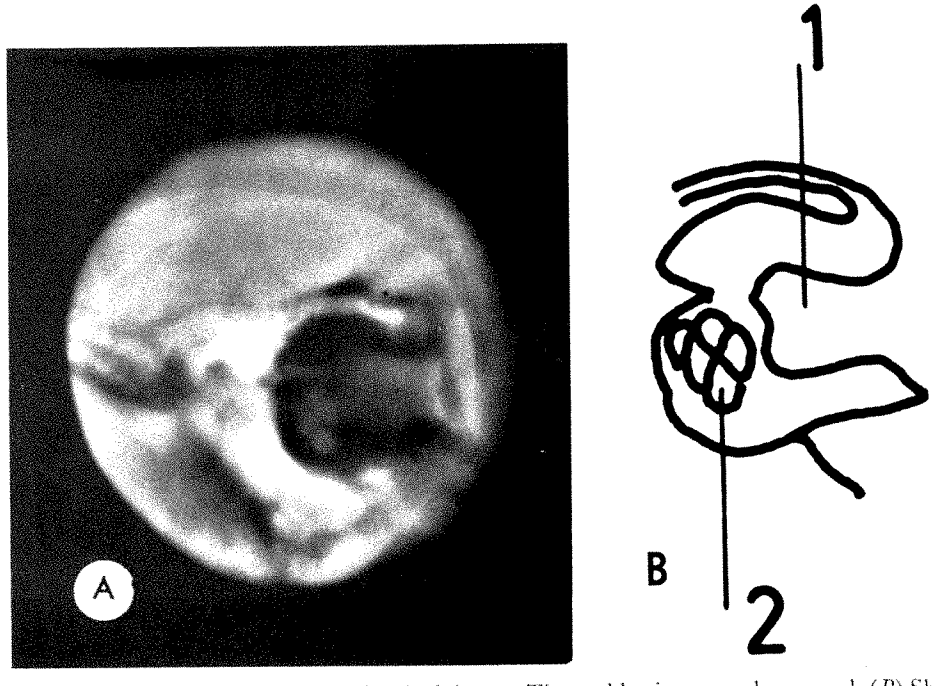



FIG. 2. (*A*) Axial pyramidal view of the patient's right ear. The cochlea is seen to be normal. (*B*) Sketch of *A*.

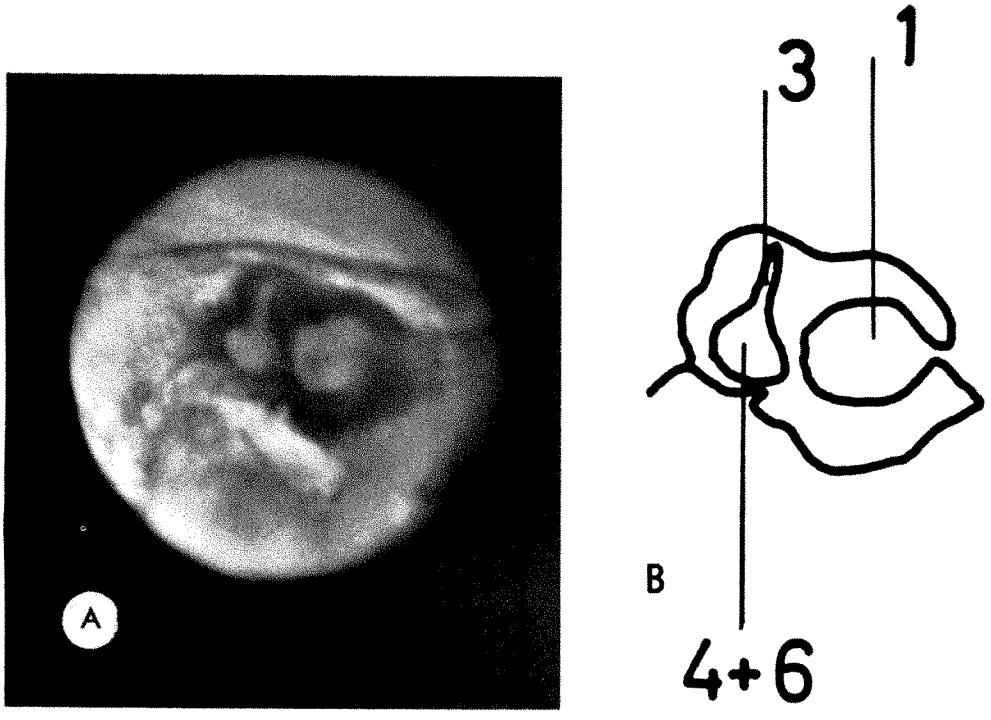



Fig. 3. (*A*) The patient's right ear in Stenvers' projection. There is no sign of the posterior semicircular canal. (*B*) Sketch of *A*.

cases (Fig. 6, *A* and *B*). On this side the oval window is present, but in none of the cuts was there any sign of the posterior semicircular canal. As regards the posterior semicircular canal and the cochlea, the appearance in the axial pyramidal and the Stenvers projections is similar to that noted on the right side (Fig. 2, *A* and *B*;

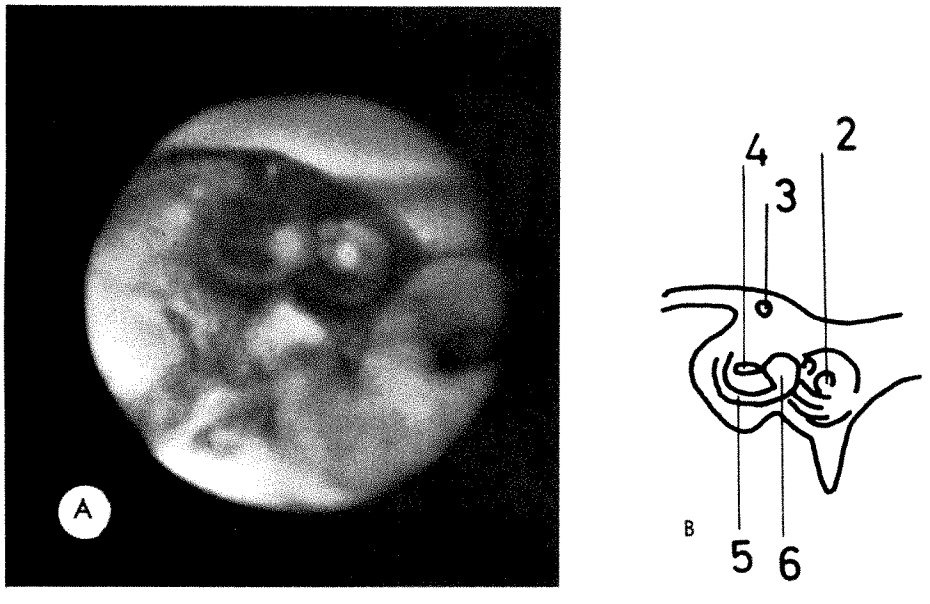



Fig. 4. (*A*) A normal ear in Stenvers' projection. (*B*) Sketch of *A*.

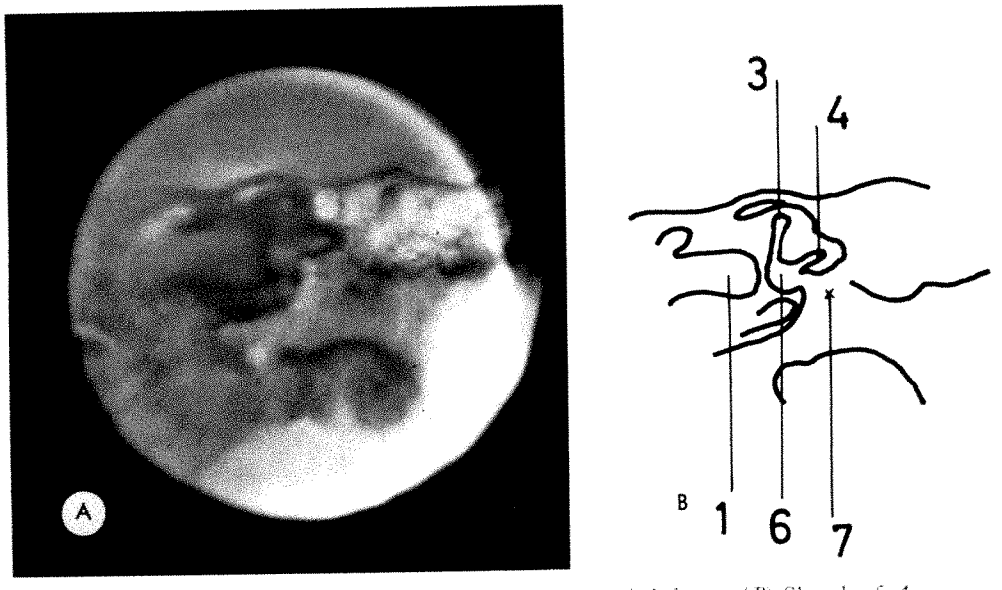



FIG. 5. (*A*) An anteroposterior view of the patient's left ear. (*B*) Sketch of *A*.

and 3, *A* and *B*). The internal auditory canal and the middle ear appear normal on both sides.

## DISCUSSION

The two reasons why the posterior semicircular canal may not be visualized in the tomograms are an osseous obliteration of the lumen or an aplasia of the canal.

Although complete or partial obliteration of the labyrinth most frequently is due to some acquired condition such as labyrinthitis, it may also be of congenital, noninfectious origin. Everberg[1] in his paper on

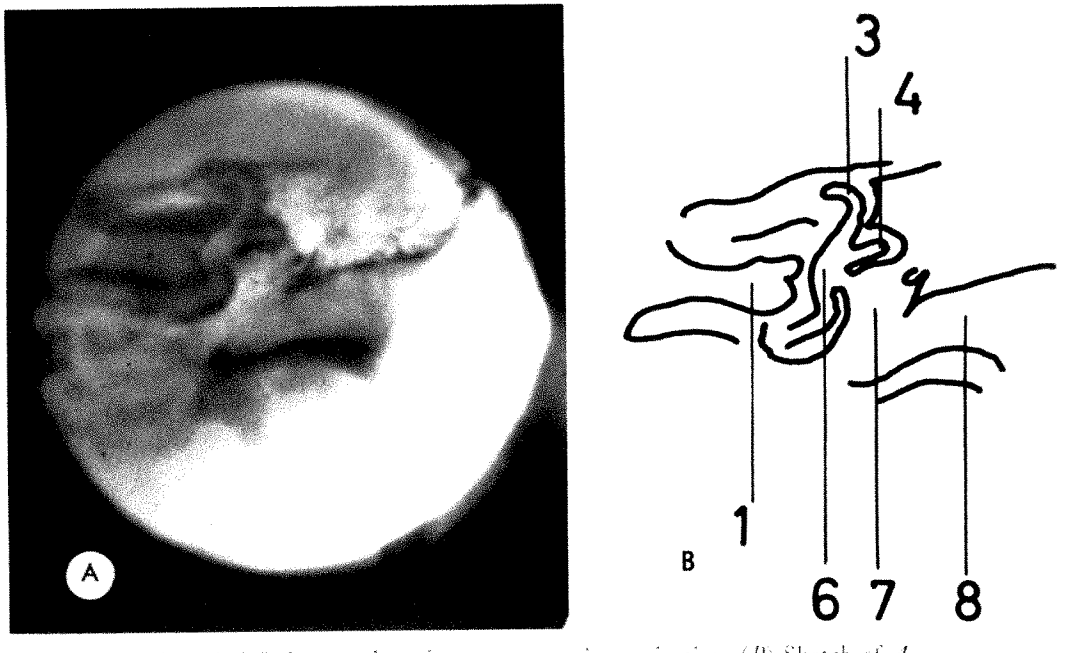



FIG. 6. (*A*) A normal ear in anteroposterior projection. (*B*) Sketch of *A*.

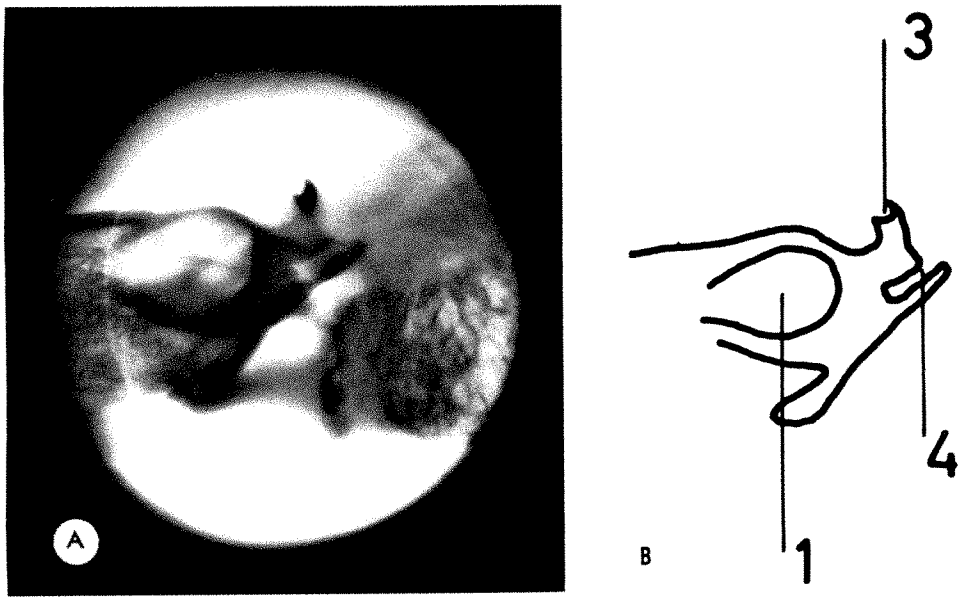



FIG. 7. (*A*) Stenvers' view of a temporal bone from the Ibsen-Mackeprang collection. There is aplasia of the posterior semicircular canal. The defects in the superior and lateral semicircular canals are due to the preparation. (*B*) Sketch of *A*.

unilateral deafness presents cases in which the tomograms show obliteration of at least the vestibule, and of the superior and lateral semicircular canals. In some of these cases there was no history of any infectious disease explaining the obliteration; therefore, it is assumed that the condition must be congenital. The outline of the labyrinth is normal, thus eliminating the possibility of aplasia.

In the present case, however, the Stenvers projection (Fig. 3, *A* and *B*) clearly shows that the posterior border of the labyrinth, which is formed by the posterior semicircular canal, is definitely more flattened than in normal cases (Fig. 4, *A* and *B*). It, therefore, seems justified to regard the case as an aplasia of the canal.

In his paper on ear malformations caused by thalidomide, Terrahe[12] noted that the inner ear malformations followed a certain pattern. In this connection it is interesting that the posterior semicircular canal was affected in the most severe cases, in which only remnants of the labyrinth were left.

Frey[3] describes a case of unilateral malformation of the middle ear and labyrinth, in which he found aplasia of both the lateral and posterior semicircular canals.

In a collection of 110 admirably prepared temporal bones from deaf children, dating from the beginning of the last century, and now kept in the Ear, Nose and Throat Department at the University Hospital (Rigshospitalet), Copenhagen, is one from a deaf-born child (another was lost, but was similar to the one available), showing aplasia of the posterior semicircular canal (Fig. 7, *A* and *B*). In this case the crura ampullaria of the remaining canals form a crus commune. In any other respect they are normal. The inner auditory canal is enlarged, but the cochlea and middle ear also appear normal.

In contradistinction to malformation of the posterior semicircular canal, reports of malformed lateral canals are not uncommon.[1,3,6,7,12] Most of the described cases resemble the present case.

Less common is the absence of the oval window. As a solitary malformation it has not been described radiologically, but only as a finding at operations for conductive hearing impairment. As part of more exten-

sive malformations, it has been demonstrated by means of tomography in a few cases by Frey[3] and Terrahe.[12]

The classification of ear malformations in 6 groups by Ormerod[8]—partly based on Siebenmann's observations—no longer is valid, since it does not include such malformations as the present one, and others published in recent years, mainly found by means of tomography. Between the Michel type (complete lack of development of the internal ear) and the Bing-Siebenmann type, as well as the subsequent types which concern only the membranous labyrinth, is only the Mondini type. However, in this type osseous malformation of the cochlea is also present, thus excluding quite a large group of inner ear malformations, including the present case.

SUMMARY

The tomographic findings of the inner ear in a patient with the Waardenburg syndrome are described.

This is the first published case with osseous malformation of the labyrinth in this syndrome.

The findings are discussed and compared with those of similar ear malformations published earlier.

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# THE INFERIOR ACCESSORY OSSICLE OF THE ANTERIOR ARCH OF THE ATLAS*

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KNOWLEDGE of the existence and appearance of accessory ossicles is of importance to the radiologist in order that he not confuse them with manifestations of pathologic conditions. This is particularly true of a rather large ossicle seen in association with the first cervical vertebra which might easily be mistaken for a traumatic or degenerative lesion. I have termed this "the inferior accessory ossicle of the anterior arch of the atlas."

## DESCRIPTION

The ossicle is a relatively large one, roughly triangular in shape with its base superiorly and its apex directed inferiorly. It is located in the midline, immediately below the anterior arch of the atlas (Fig. 1–4). There appears to be an articular space between the ossicle and the inferior aspect of the anterior arch of the atlas. In some of the cases illustrated here, there are additional tiny ossific nuclei in the areas of this articulation.

A review of the literature has shown only one notation of an ossicle located in this area. In Kohler and Zimmer's book "Borderlands of the Normal and Early Pathologic in Skeletal Roentgenology,"[1] Figure 668 shows a small osseous nucleus below the anterior border of the atlas, and it is stated that the authors have found it repeatedly. A similar small ossicle is illustrated in Figure 5. This small nucleus is not only of lesser magnitude than that described here, but also does not enter into articulation with the anterior arch of the atlas.

The midline location of the interior

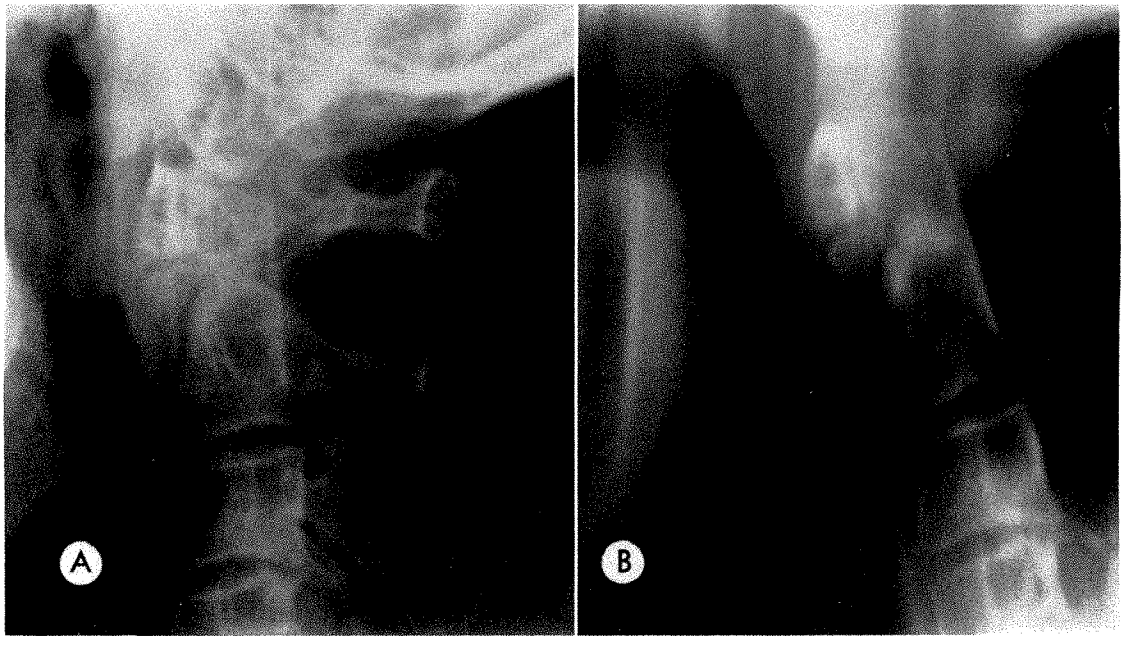



FIG. 1. (*A*) The ossicle is seen lying below the anterior arch of the atlas. (*B*) Midline laminagram demonstrates the ossicle to better advantage.

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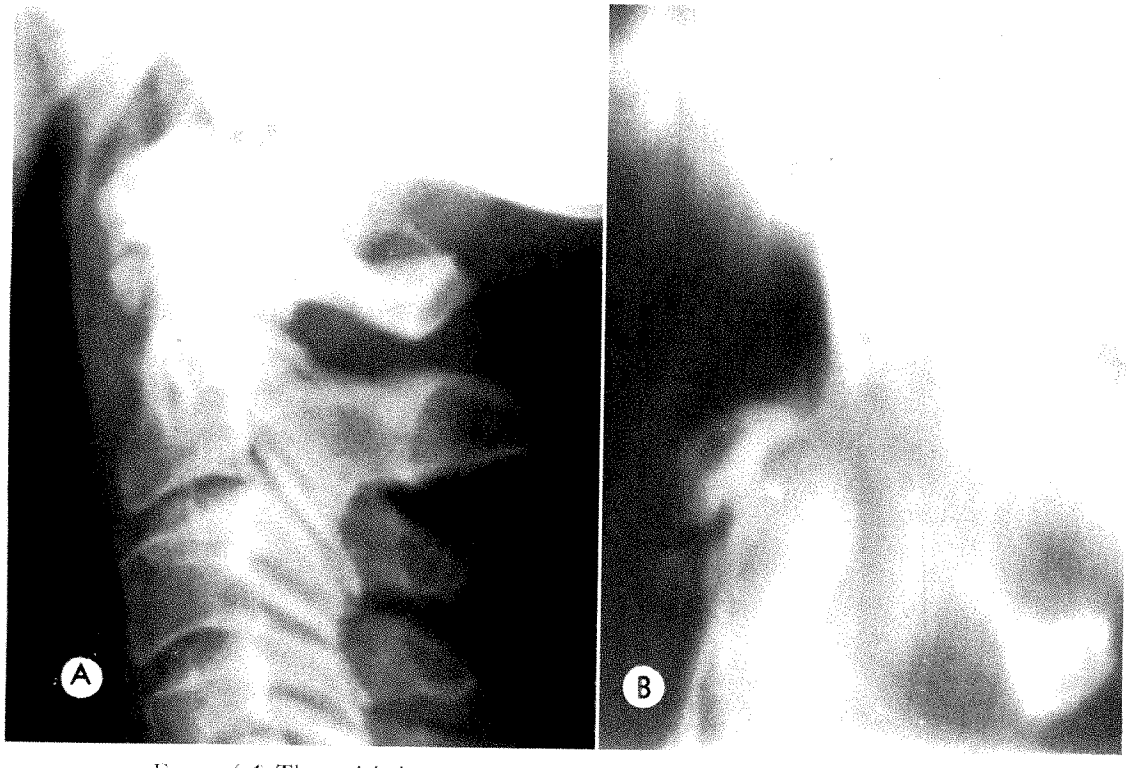



FIG. 2. (*A*) The ossicle in a second patient. (*B*) Midline laminagraphic section demonstrating the ossicle to better advantage.

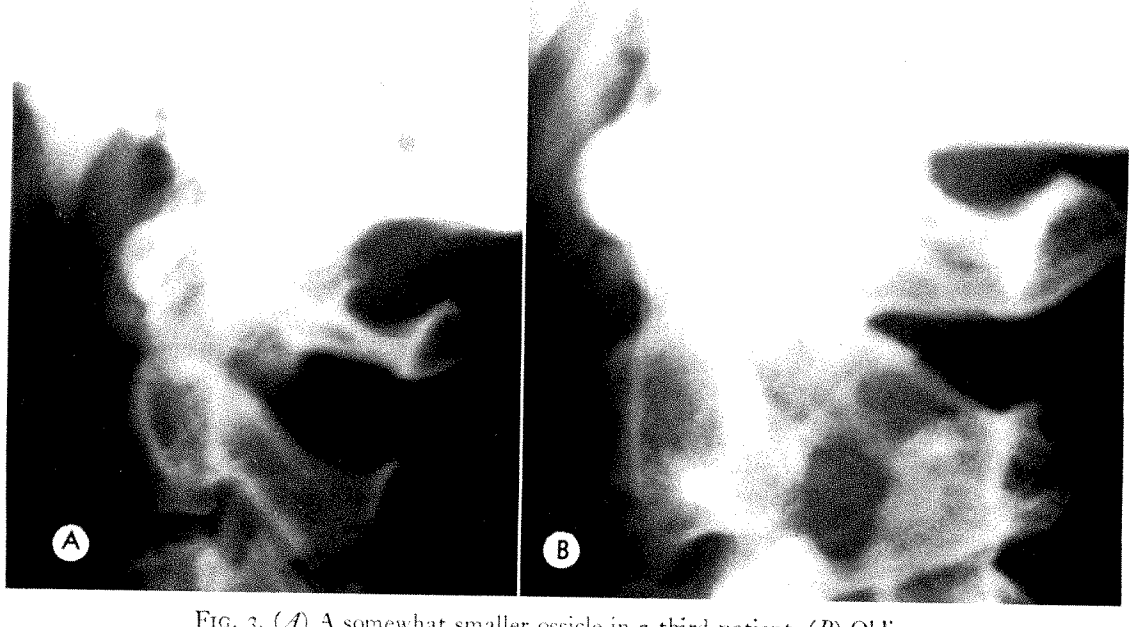



FIG. 3. (*A*) A somewhat smaller ossicle in a third patient. (*B*) Oblique projection shows articulation to better advantage.

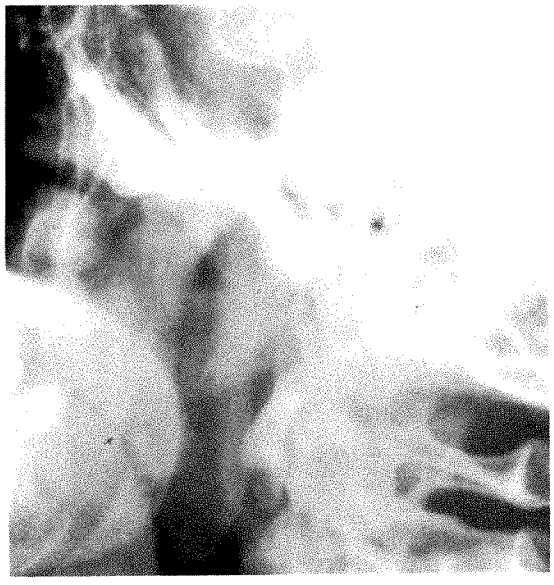


FIG. 4. Typical inferior accessory ossicle in a fourth patient.



FIG. 5. Small isolated osseous nucleus below the anterior border of the atlas, differing in appearance from those seen in Figures 1 to 4. The ossicle has been retouched for greater clarity. There is no articulation with the atlas. (Courtesy of Dr. J. F. Holt, University of Michigan.)

accessory ossicle was proven by laminagraphy, and it is suggested that the ossicle probably formed within the anterior longitudinal ligament, although there is no gross anatomic proof of this fact. Since the anterior arch of the atlas is intact, there is no reason to believe that the ossicle developed from a displacement of the early ossification center for that structure.

The discovery of these ossicles was incidental to the patients' problems, and the importance of their proper identification is only that they not be confused with acquired lesions. Our experience with only 4 such ossicles suggests that they are probably quite rare.

SUMMARY

A large accessory ossicle located below and articulating with the inferior aspect of the anterior arch of the atlas is described. This has been termed "the inferior accessory ossicle of the anterior arch of the atlas." Its importance lies in its recognition as only an anatomic variant.

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VOL. 101, NO. 4

# OSSIFIED ARYTENOID CARTILAGE MASQUERADING AS A FOREIGN BODY*

## A CASE REPORT

By CHARLES LOUIS MORREELS, JR., M.D., JERRIE CHERRY, M.D., and JACOB I. FABRIKANT, M.D.
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A KNOWLEDGE of the pattern of ossification of the arytenoid cartilages is essential for interpreting roentgenograms taken for localization of radiopaque foreign bodies in the laryngeal airway. This report is of interest since it illustrates the possible confusion which can arise between a unilateral ossified arytenoid cartilage and a foreign body and demonstrates how this may be resolved with polytomographic examinations.

### REPORT OF A CASE

A 57 year old female patient was referred with complaints of soreness and intermittent discomfort in the base of the tongue and in the lower part of the left side of the neck. Two months previously, she claimed to have inadvertently swallowed a piece of "hard material" while eating, but there had been no abnormal physical or roentgenographic findings. A roentgenogram of the soft tissues of the neck revealed a density suggestive of a foreign body within the pyriform sinus, but on direct laryngoscopy, none was found (Fig. 1). She returned to the clinic a number of times subsequently, complaining of a migratory but persistent discomfort in the neck, but on each occasion, no foreign body or mucosal disease was seen. Nine months later, her pain continued, and on the basis of the initial roentgenographic examination polytomographic studies were obtained (Fig. 2 and 3). These verified that the density was a unilateral ossified arytenoid cartilage, and reconfirmed that a radiopaque foreign body was not present. The patient was reassured of the findings, treated symptomatically and discharged.



FIG. 1. Density (arrow) overlying the thyroid cartilage and pyriform sinus region on the lateral soft tissue roentgenogram of the neck.

### DISCUSSION

As early as 1902, Scheier[6] had described the pattern of ossification of the cartilaginous skeleton of the larynx on postmortem roentgenographic and anatomic preparations. It was not until 1934, however, that Chamberlain and Young[1] reported on the roentgenographic appearance of ossified arytenoid cartilages. Since then a number of observers have emphasized the importance of the anatomic relationships of these structures.[3–5] Hately *et al.*[3] described the temporal pattern of ossification of the laryngeal cartilages of 516 patients. Ossification begins in late adolescence and by 20 years of age, almost all patients have some laryngeal cartilage ossification. The pattern is evidently genetically controlled

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FIG. 2. Polytomogram of the larynx in the coronal plane. The density represents the ossified arytenoid cartilage in normal juxtaposition to the partially ossified cricoid cartilage (arrow).



FIG. 3. Polytomogram of the larynx in the sagittal plane through the ossified arytenoid cartilage and cricoid cartilage (arrow).

to some degree since similar patterns were observed in identical twins.[7] The frequency and sex distribution of ossified arytenoid cartilages is not as well defined and varies widely in different series; however, it appears to be more prevalent in females.[3–5]

## CONCLUSION

From the case reported, it becomes readily apparent that routine lateral roentgenograms of the soft tissues may not be sufficient for adequate evaluation for a suspected foreign body in the upper air passages, particularly if superimposed ossified laryngeal cartilages are present. It may be necessary to elucidate both normal and abnormal anatomic structures with polytomographic examinations, initially in the coronal plane and possibly in the sagittal plane as well.

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# THE CORRUGATED VOCAL CORD*

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DURING an evaluation of positive contrast laryngography[1] it was necessary to obtain an appreciation of normal laryngographic appearances. Several patients with asymptomatic upper respiratory tracts were examined by positive contrast laryngography following bronchography. The examination of the larynx was carried out on the basis of the technique described by Powers *et al.*[2] An irregularity of the phonating vocal cords was occasionally encountered, and since no reference to this feature was found in the literature, a brief description is given.

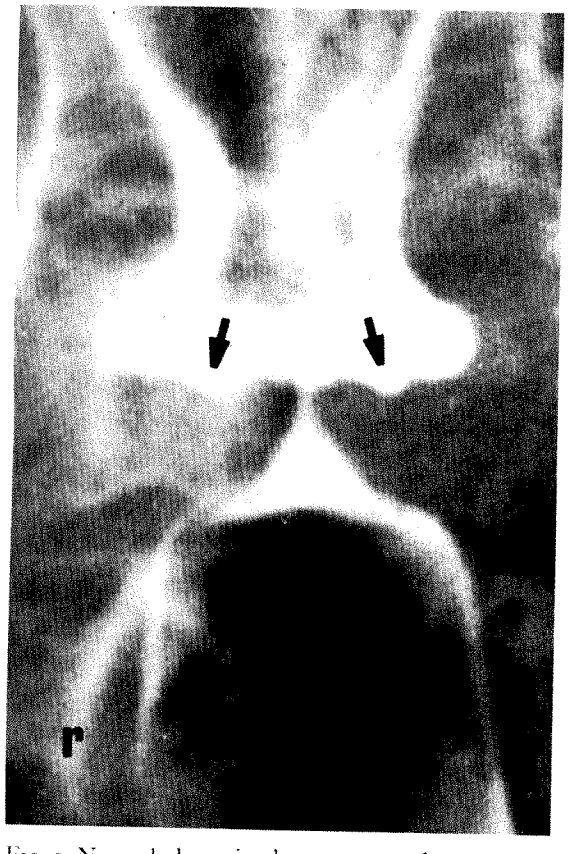



FIG. 1. Normal phonation laryngogram, frontal view, demonstrating bilateral corrugation of the superior aspect of the adducted true vocal cords (arrows).

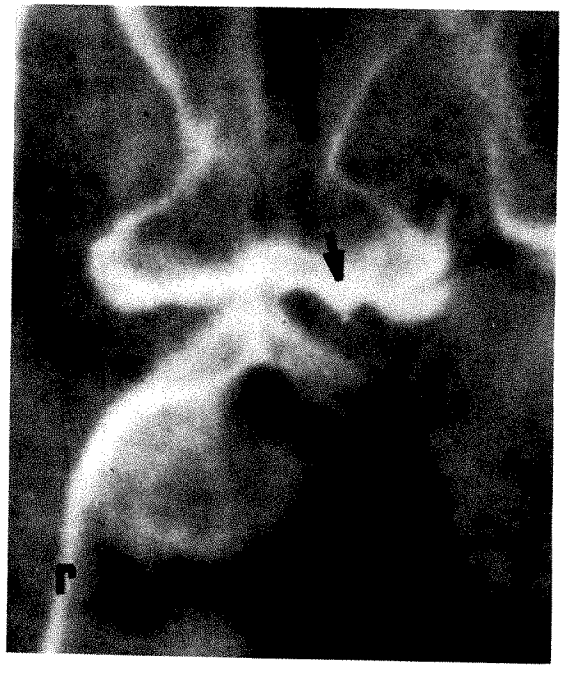



FIG. 2. Normal phonation laryngogram, frontal view. The corrugation is more apparent on the left (arrow) than on the right vocal cord.

The irregularity takes the form of a corrugation of the superior surface of the true vocal cords (Fig. 1) when visualized in the frontal projection during the phonation of a high pitched vowel sound. The corrugations are only demonstrated when the central roentgen ray is in exact alignment with the adducted cord. Minimal lack of alignment may result in the grooving being less well demonstrated on one side than on the other (Fig. 2). No abnormality is seen in the lateral view.

The cause of this normal corrugation is not immediately apparent. It seems unlikely that it is due to the rolling eversion of the free margin of the vibrating, phonating vocal cord since the frequency of this vibration is over 400 cycles per second and the spot laryngograms were obtained at

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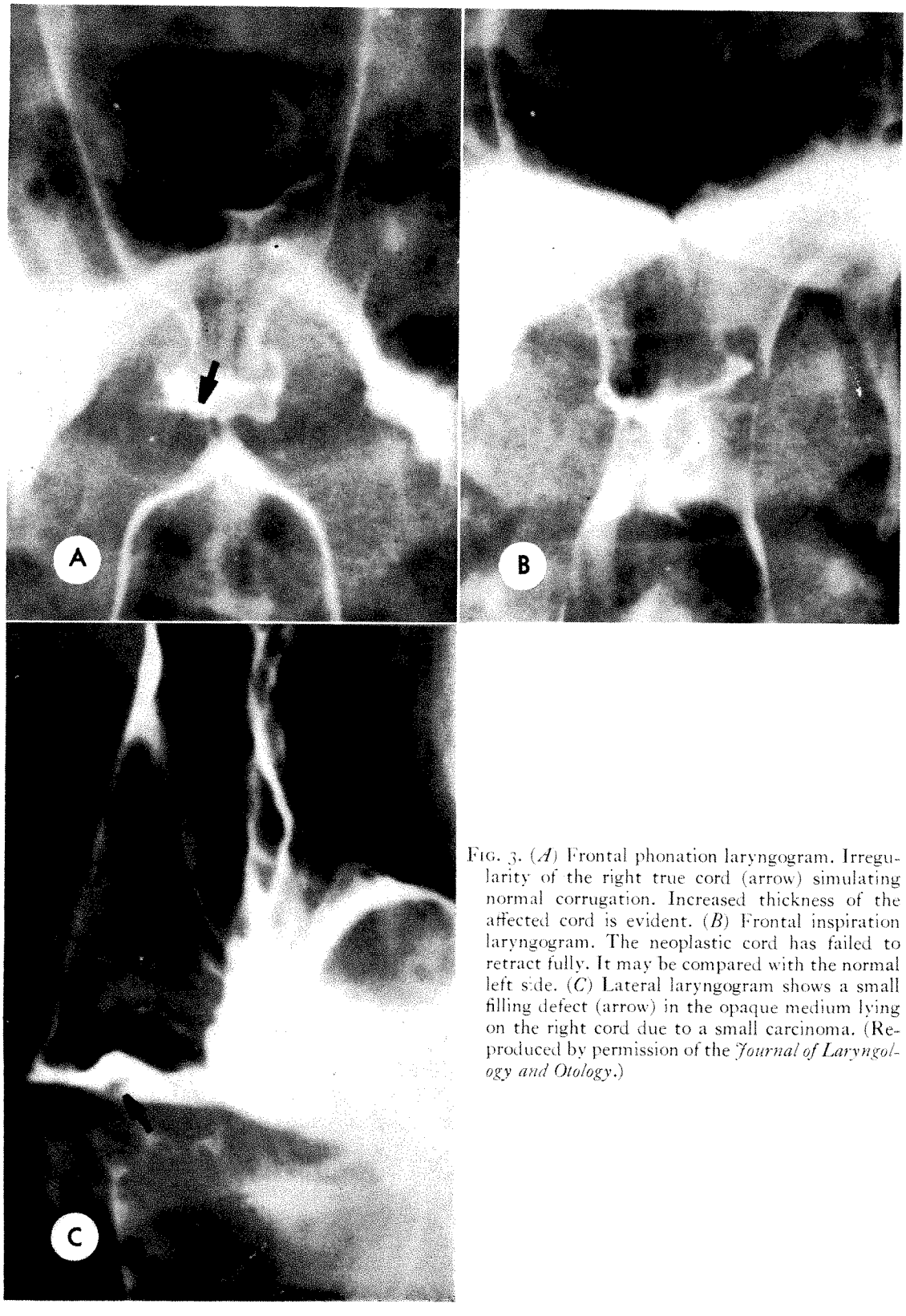



FIG. 3. (*A*) Frontal phonation laryngogram. Irregularity of the right true cord (arrow) simulating normal corrugation. Increased thickness of the affected cord is evident. (*B*) Frontal inspiration laryngogram. The neoplastic cord has failed to retract fully. It may be compared with the normal left side. (*C*) Lateral laryngogram shows a small filling defect (arrow) in the opaque medium lying on the right cord due to a small carcinoma. (Reproduced by permission of the *Journal of Laryngology and Otology*.)

0.04 second exposure times. Consequently, any vibratory effect would likely be obscured. The appearances may be the result of extreme tension of the true cord during phonation of an unusually high pitched sound. Thus the interaction between cricothyroid muscle, which increases the distance between the thyroid and arytenoid cartilages, and conus elasticus, which anchors the cord to the cricoid arch, might result in a depression above the line of insertion of the conus into the cord.

The importance of this corrugation of the vocal cord lies in its recognition as a normal variant which must be differentiated from an early glottic carcinoma. Such a tumor may cause a similar, unilateral irregularity (Fig. 3, *A–C*) but may be diagnosed by the increased bulk of the affected cord, with inability to retract fully on inspiration and by recognition of the tumor in the lateral view.

SUMMARY

The corrugated vocal cord, a normal variant, is described. Possible causes are suggested and the differentiation from carcinoma is discussed.

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# HEREDITARY OSTEO-ONYCHO-DYSPLASIA*

## THE NAIL-PATELLA SYNDROME

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HEREDITARY osteo-onycho-dysplasia, also known as the nail-patella syndrome, is a condition affecting the nails, the skeleton and occasionally the kidneys. A more descriptive term would be hereditary osteo-onycho-arthro-dysplasia.

A kindred affected with this disorder illustrates its autosomal dominant inheritance (Fig. 1), clinical, and roentgenographic manifestations.

### REVIEW OF THE LITERATURE

Skeletal and nail dysplasia was described for the first time in the nineteenth century by Chatelain[6] in a family with absence of thumb nails and patellae suggesting hereditary origin of this disorder. Little[22] in 1897, reported some cases of agenesis of patellae and thumb nails. Subsequently similar cases were reported by Wrede[35] in 1909 and by Firth[11] in 1912. Turner[34] in 1933, published detailed studies of cases with nail and bone dysplasias with flaring of the iliac crests and prominence of the anterosuperior iliac spines. Lester[21] in 1936, reported similar cases and noticed in some of them, that the ectodermal element of the irises was widened and darkened (Lester's sign[4]), causing a dark and irregularly widened pupillary border.

It was not until 1939 that Keiser,[17] noting iliac horns in similar cases, contributed to the now well defined clinical tetrad: dysplasia of nails, elbows, patellae, and presence of iliac horns. Senturia and Senturia[32] in 1944 and Nickerson[28] in 1945, reported some cases, but iliac horns were not mentioned. Fong[12] reported the first American case of iliac horns in 1946 that he had observed as an incidental finding in films of an excretory urography, consisting of conical bony projections arising bilaterally from the posterior surface of the iliac bones. In an addendum to Fong's article, Doub[7] described the iliac horns in a patient whom he had seen years earlier. Those cases were probably examples of osteo-onycho-dysplasia.

Mino, Mino, and Livingstone[26] writing in 1948, noted the relationship of the iliac horns with the triad of nail, knee, and elbow dysplasias. In their cases some other abnormalities were observed such as bilateral posterior dislocation of the deformed radial head, exostosis of the inner table of the frontal bone, thickening of the axillary border of the scapula and minor deformities of the wrist. Thompson, Walker, and Weens[33] a year later thought that the iliac horns occurred inconstantly in the syndrome.

Subsequent sporadic cases were reported by Love and Beiler,[23] Zimmerman,[36] and others,[1,10,13,16] who described some less frequent findings such as hypoplasia of the ilium, intrapelvic protrusion of the acetabula, coxa valga, excessive lordosis of the spine, genu valgum, weakness of the quadriceps femoris, thickening of the malleoli, hypoplasia of the scapula, subluxating clavicles, flattening of the humeral heads, and hypoplastic or deformed styloid processes of the ulna with and without dislocation. A review of the literature up to 1963 was made by Duncan and Souter[8] adding 3 cases of their own and describing some deformities of the feet.

Eighteen cases of 6 generations were reported in the same year by Duthie and Hecht.[9] Up to then the total number of fully documented cases in the world literature was about 300.

Lucas and Opitz[24] in a recent report of 5 kindreds found other unusual changes:

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scoliosis was observed in 3 cases and partial ankylosis of the distal interphalangeal joints of the fingers in others. Similar changes were found in 7 cases reported by Maini and Mittal[25] with flexion contraction of the distal interphalangeal joints of the fingers.

Disorder of the kidneys has been observed infrequently, but seems to be a potential fatal feature associated with osteo-onycho-dysplasia. Its presence is usually manifested by proteinuria and recurrent urinary infections. Six members of a family studied by Hawkins and Smith[15] had glomerulonephritis due to renal dysplasia, 2 of whom died of generalized edema. Brixey and Burke[3] reported a case where the patient died in uremia and the autopsy revealed glomerulonephritis, sparing the upper poles. Gibbs, Berczeller, and Hyman[14] reported a case of a 20 year old woman in whom albuminuria had been noted repeatedly for 10 years. A renal biopsy showed "focal glomerular necrosis" and diffuse hydropic degeneration of the tubules. The skin was also biopsied revealing elastic tissue alterations.

Muth[27] performed renal biopsy in a 17 year old patient with a history of persistent heavy proteinuria of at least 6 years' duration. He concluded that the changes did not justify the diagnosis of chronic glomerulonephritis, but were highly suggestive of an early phase. Recently Leahy[20] has reported a fatal case of hereditary nephropathy associated with osteo-onycho-dysplasia. Both the antemortem and postmortem examinations of the kidneys were indistinguishable from histologic phase of chronic glomerulonephritis. This case probably represents only the fourth documented death due to hereditary nephropathy of osteo-onycho-dysplasia.

Mental deficiency and psychosis have been described by Schröder,[31] and Barrett.[2] In one of the affected kindreds studied by Lucas and Opitz,[24] 8 members had mental disorders.

All of the cases reported up to date in the United States are Caucasians except for one, a Negro, reported by Carbonara and Alpert.[5] One Chinese family in Singapore was reported in 1965 by Pillay[29] which was probably the first report of osteo-onycho-dysplasia in the Mongolian Race. A report of an affected family from India has recently appeared in the literature.[25]

Osteo-onycho-dysplasia is transmitted as a mendelian dominant trait. The gene is fully penetrant; both sexes are affected in an equal number and pass on the gene with equal probability to half of their sons and half of their daughters. This condition was found linked to another gene, namely ABO locus. The knowledge of the genetic

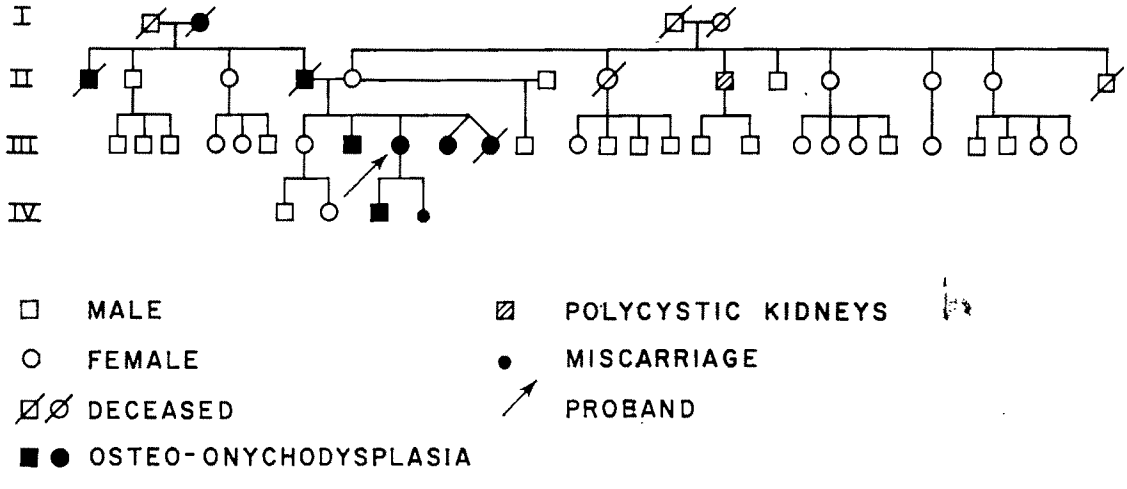



FIG. 1. J. R. Kindred.

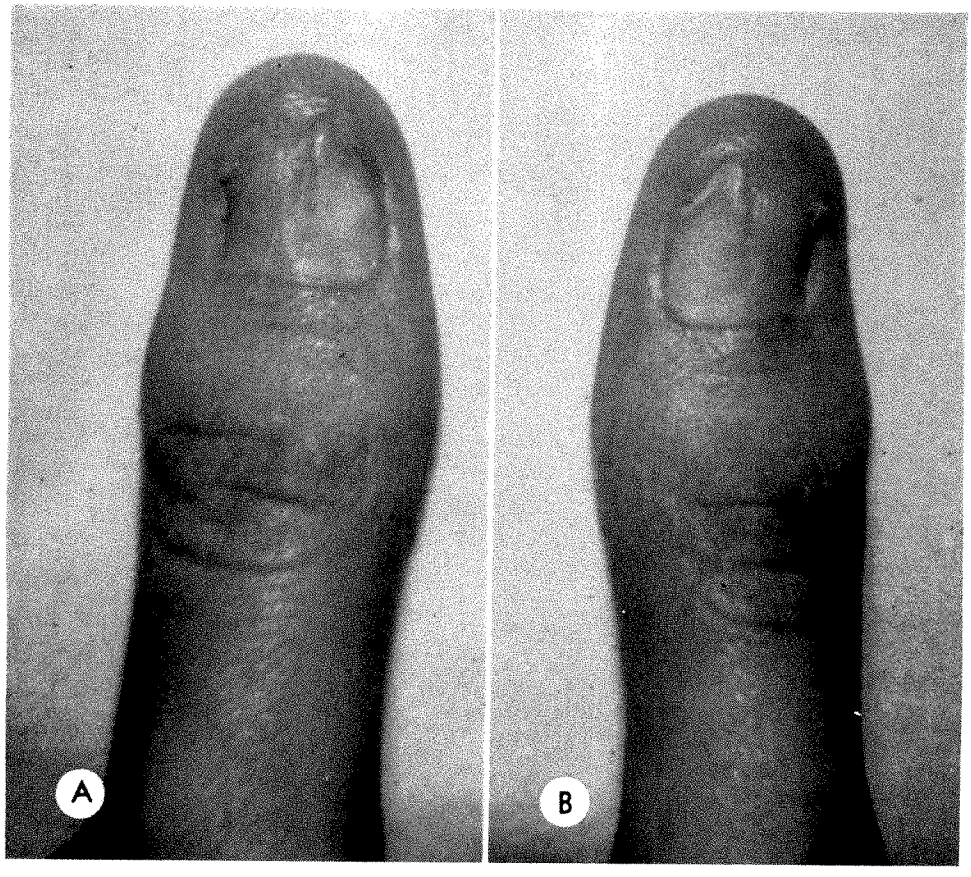



FIG. 2. (*A* and *B*) Thumb nails of Case 1 showing the classical changes of nail dysplasia.

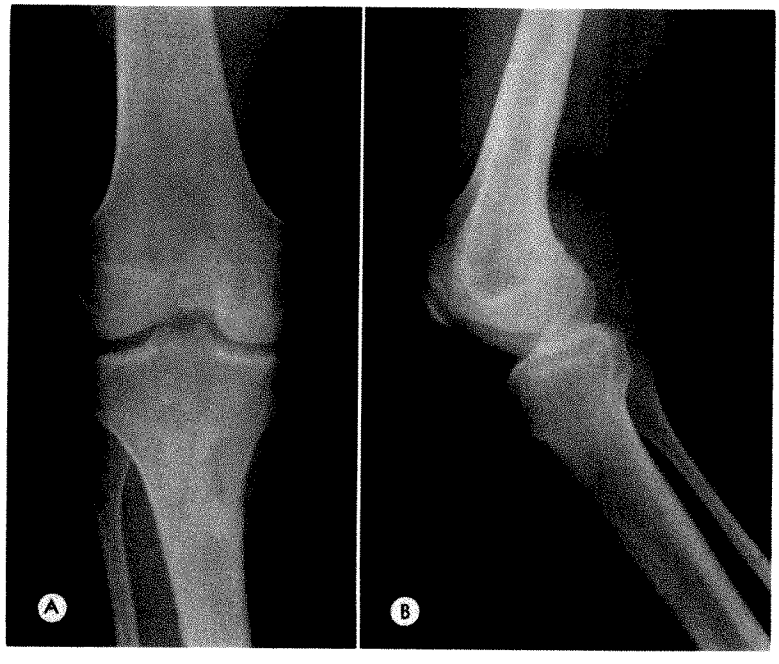



FIG. 3. (*A*) Anteroposterior and (*B*) lateral views of the knee of Case 1 revealing a hypoplastic patella with flattening of the lateral femoral condyle. Note the postoperative changes in the tibia.

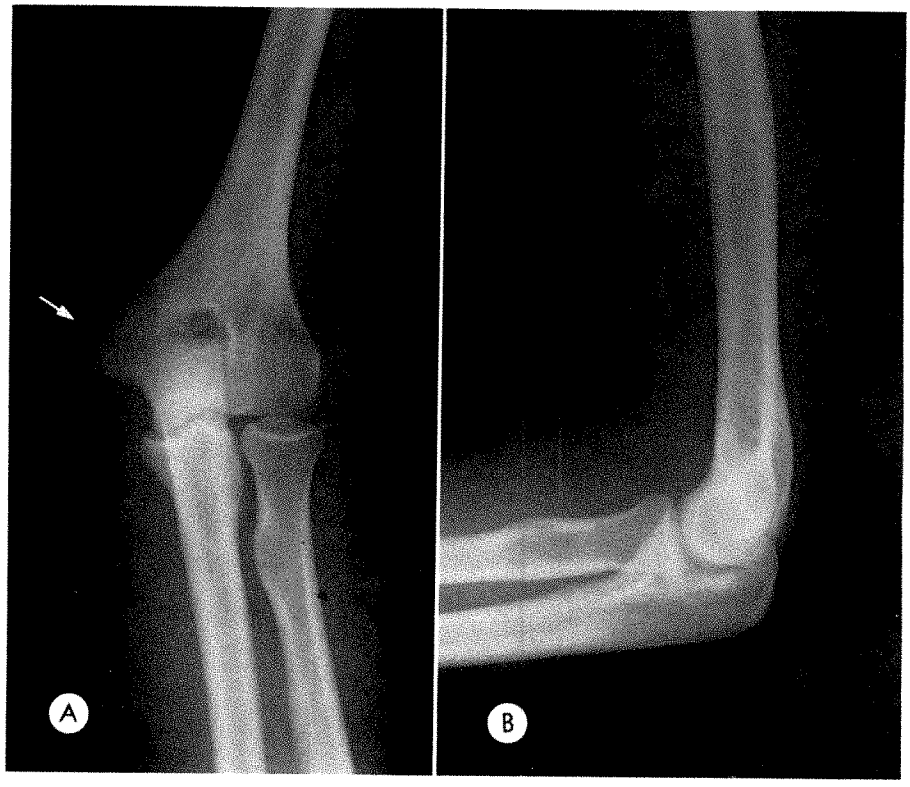



Fig. 4. (*A*) Anteroposterior and (*B*) lateral views of the elbow of Case 1 showing prominence of the medial epicondyle (arrow) and increased carrying angle.

behavior of this syndrome has been mainly the result of linkage studies by Renwick, Lawler, Wildervanck, Hauge and Mosbech.[18,19,30] These studies have shown that the chromosome carrying the gene of osteo-onycho-dysplasia is also the vehicle transporting the ABO blood group locus. This has been confirmed by Duthie and Hecht[9] in 1963.

## REPORT OF CASES

Case 1. Jo R. was a 22 year old female housewife, seen at the Orthopedic Clinic of the Uni-

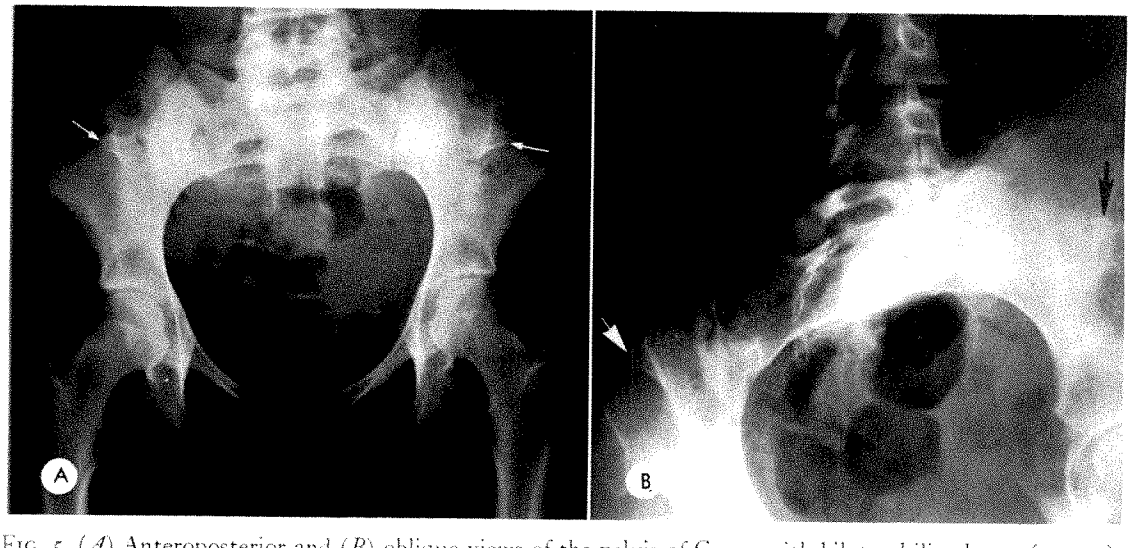



Fig. 5. (*A*) Anteroposterior and (*B*) oblique views of the pelvis of Case 1 with bilateral iliac horns (arrows).

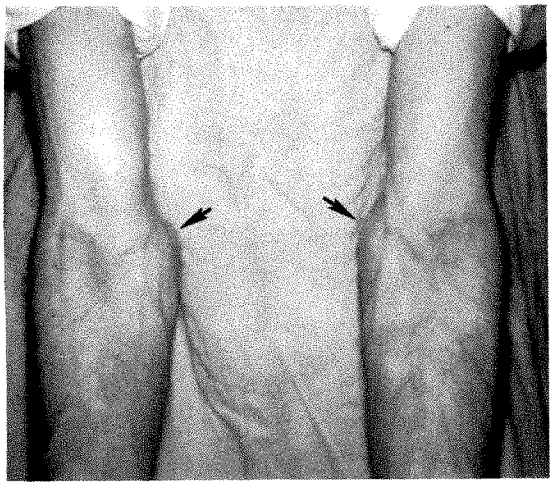


FIG. 6. Elbows of Case II showing increased carrying angle and prominence of the medial epicondyles (arrows).

versity of Kansas Medical Center because of recurrent dislocation of the patellae for a period of 3 years. This had become more frequent and painful, causing her to fall because of instability of the knees.

Physical examination revealed lateral dislocation of the patellae upon flexion of the knees with crepitation, tenderness, and swelling. The elbows showed slight limitation on motion and increased carrying angle. Deformity of the thumb nails was noted (Fig. 2, *A* and *B*).

She stated that several members of her family were affected with the same condition. Her previous record showed bouts of urinary infection and chronic proteinuria.

Roentgenographic examinations of the knees revealed hypoplastic patellae with flattening of the lateral femoral condyles (Fig. 3, *A* and *B*) producing lateral patellar dislocation on flexion. A Houser procedure was carried out on both knees to correct the patellar dislocation, with satisfactory results.

The skeletal findings which appeared to be familial led us to further investigation in this patient and her family (Fig. 1).

The rest of the roentgenographic examination on this patient disclosed prominence of the medial epicondyle of the humeri and small radial heads bilaterally (Fig. 4, *A* and *B*) with increased carrying angle. The pelvis showed bilateral iliac horns which in retrospect were clinically palpable (Fig. 5, *A* and *B*).

CASE II. Glen P., a 20 year old male mechanic, brother of Jo R., had been rejected from military duty because of deformities of the elbows and patellar dislocation. There was also history of recurrent urinary infection.

Physical examination showed marked limitation on pronation, supination, and extension of both elbows with increased carrying angle (Fig. 6). There were small patellae which dislocated upon flexion of the knees (Fig. 7). Deformities of the thumb (Fig. 8) and toe nails were present. The deformities did not produce serious disabilities.

On roentgenographic examination of the skeleton there were bilateral posterior subluxation of the radial heads which appeared to be deformed, prominence of the medial epicondyles and bilateral flattening of the capitellum (Fig. 9, *A* and *B*). The knees showed hypoplastic and fragmented patellae with anterolateral



FIG. 7. Case II with knees in flexion producing lateral dislocation of the patellae (arrows).



FIG. 8. Dysplastic thumb nails in Case II.

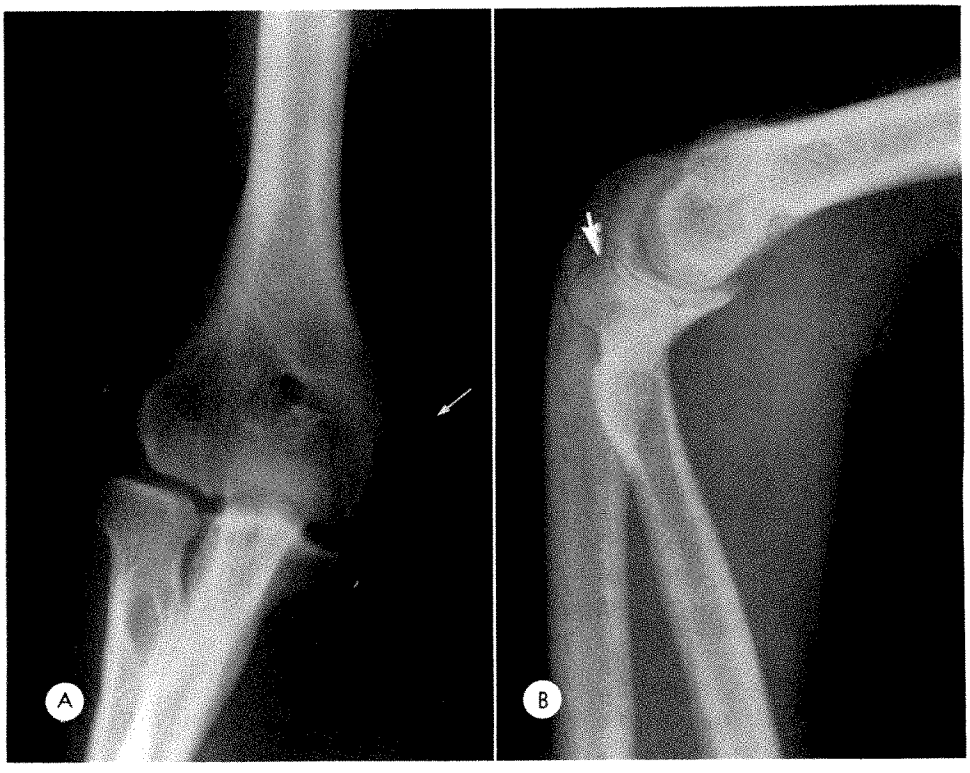



FIG. 9. (*A*) Anteroposterior and (*B*) lateral views of the elbows in Case II with flattening of the capitellum and hypoplastic radial head with posterior subluxation (arrow in *B*). Note prominence of the medial epicondyle (arrow in *A*).

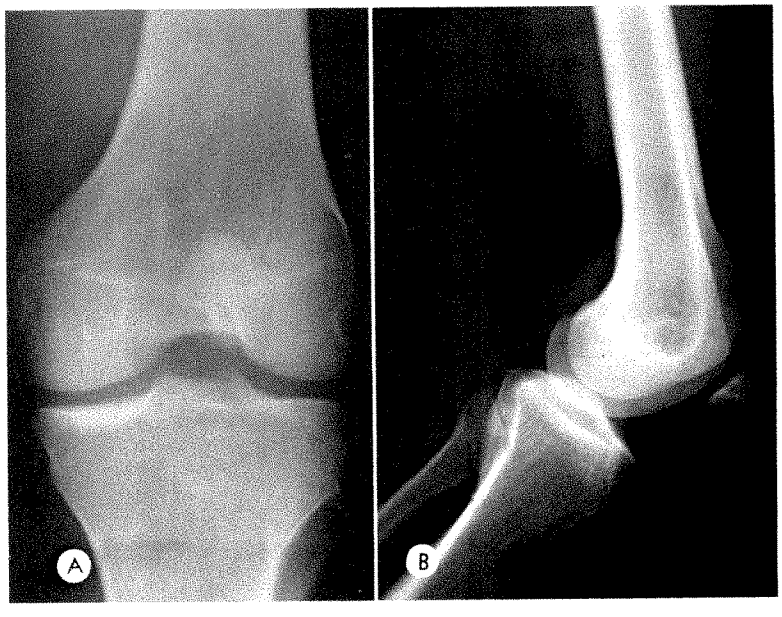



FIG. 10. (*A*) Anteroposterior and (*B*) lateral views of the knee of Case II disclosing hypoplastic and fragmented patella.



FIG. 11. Pelvis of Case II revealing bilateral iliac horns (arrows).

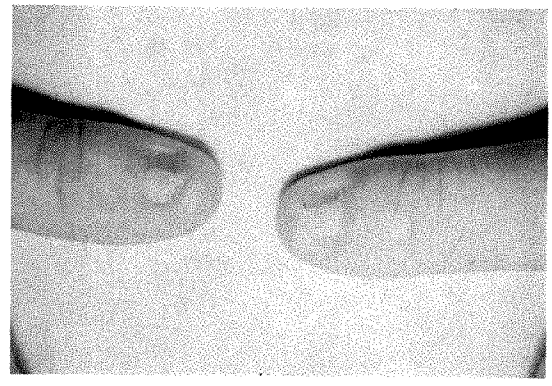


FIG. 13. Dysplastic thumb nails of Case IV.

flattening of the lateral femoral condyles (Fig. 10, *A* and *B*). The pelvis disclosed bilateral iliac horns which were clinically palpable (Fig. 11).

CASE III. Karen P. was an 18 year old female, sister of Jo R., who had been having orthopedic treatment for scoliosis. She also had deformities of the elbows and recurrent dislocation of the patellae. Some deformity of the thumb nails was present. Roentgenographically, scoliosis of the spine with excessive lordosis of the lumbosacral angle and presence of iliac horns were the main features (Fig. 12, *A* and *B*).

CASE IV. Kavin R. was a 4 year old male, son of Jo R., with no symptoms. On physical examination increased carrying angle of the elbows and dysplastic thumb nails (Fig. 13) were found. Roentgenographically, prominence of the medial epicondyles of the elbows was noted (Fig. 14, *A* and *B*).

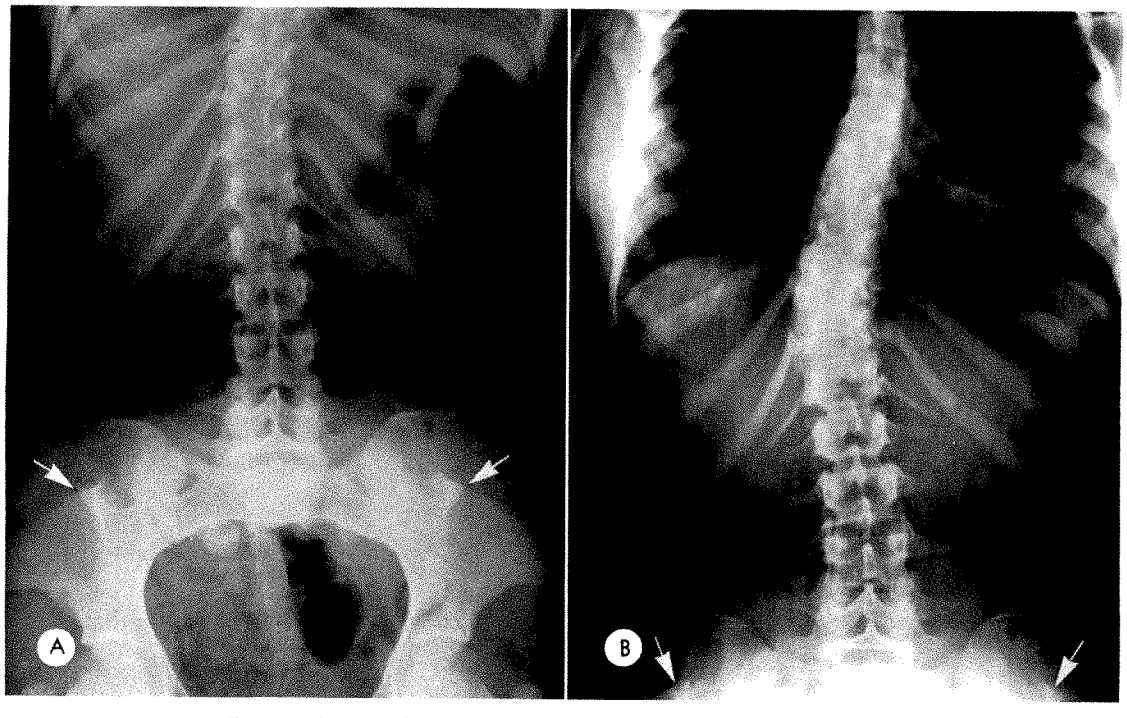



FIG. 12. (*A* and *B*) Scoliosis and iliac horns (arrows) of Case III.

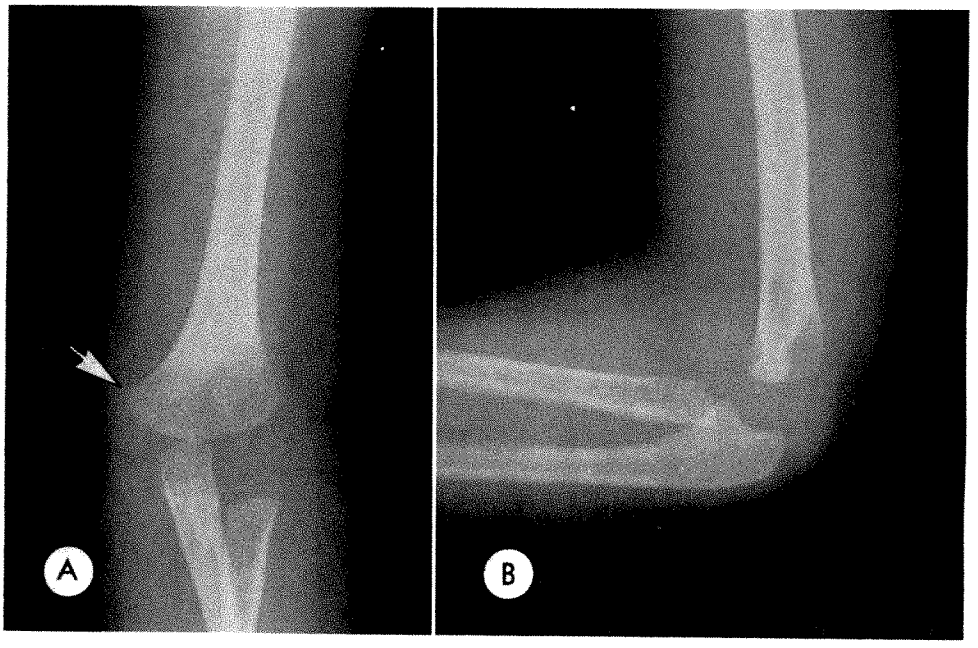



Fig. 14. (*A*) Anteroposterior and (*B*) lateral views of the elbow of Case IV with prominence of the medial epicondyle (arrow) and increased carrying angle. This was more evident clinically.

## SUMMARY AND CONCLUSIONS

Hereditary osteo-onycho-dysplasia, a disorder inherited in an autosomal dominant manner, is manifested by dysplasia of the knees, elbows, and nails. The presence of iliac horns, bony projections arising from the posterior aspect of the iliac bones, seems to be pathognomonic of this condition. Although a completely penetrant syndrome, it exhibits extremely variable expression not only of the components of the tetrad but also of a number of other anomalies of the skeleton. Occasionally, the kidneys and eyes are also affected.

The incidence of major involvement in patients with hereditary osteo-onycho-dysplasia in previously reported cases is as follows: nails 98 per cent; elbows 90 per cent; knees 92 per cent (agenesis of the patellae 11 per cent, hypoplastic patellae 79 per cent, patellar dislocation 29 per cent) and iliac horns 81 per cent (70 per cent are clinically palpable).

A review of the literature, and the clinical and roentgenographic observations of a family affected with the classic tetrad of osteo-onycho-arthro-dysplasia in which, in addition, there were manifestations of renal dysplasia, are presented.

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# SPONDYLO-EPIPHYSEAL DYSPLASIA TARDA*

## A CAUSE OF PREMATURE OSTEOARTHRITIS

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SPONDYLO-EPIPHYSEAL dysplasia tarda is a hereditary chondrodysplasia, which was described in the European literature by Maroteaux *et al.*[7] It occurs in males, presenting in adolescence or adult life with hip or back pain, caused by premature osteoarthritis. The patient's short stature first becomes evident in late childhood and is a result of decrease in spinal height; the extremities are normal in length. There are no visceral abnormalities; the mental capability of these patients is normal.

### REPORT OF CASES

CASE I. A 39 year old Negro male was admitted to Jackson Memorial Hospital for correction of his right hip disability. He stated that he was well until one year prior to admission, when he began to have pain in his right hip. This became increasingly severe over the next 6 months, until he was unable to continue his trade as a roofer.

The patient stated that he had been aware of his short stature since childhood. His mother and father were of normal height; there was no history of consanguinity. The patient stated that he had 4 brothers and 2 sisters. One brother was smaller than the patient and had the same type of short trunk. This brother lived outside the state of Florida and was unavailable for examination. The other siblings were reported as normal in appearance.

Physical examination revealed a muscular, middle-aged, Negro male whose extremities were of normal length, but who had a markedly shortened trunk and a shortened neck. His height was 5 feet, 2½ inches. Considerable pain and limitation of motion were found on examination of the right hip. The remainder of the joints of the extremities were clinically normal.

Roentgenograms of the entire spine revealed flattening of all the vertebral bodies, with a bulging of the middle and posterior thirds of the bodies, producing convex superior and inferior surfaces. The anterior thirds of the vertebral bodies were severely narrowed, with concave superior and inferior surfaces. The disk spaces were markedly thinned, especially in their middle and posterior thirds (Fig. 2). Normal pediculate measurements were noted. Inspection of the cervical spine showed slight hypoplasia of the odontoid process.

The ribs were wide and flaring in their axillary and anterior portions. Posteriorly, the ribs were crowded by the shortening of the dorsal spine. The pelvis exhibited slight hypoplasia of the iliac bones, principally in their cephalic portions, without abnormality of the sciatic notches. The sacral alae were also small, and the sacrum articulated low on the iliac bones. The right hip revealed narrowing of the joint space, most severe in the superior and lateral portions. There were cystic and sclerotic changes in the subchondral bone of the acetabulum and of the femoral head. The left hip joint had slight irregularity of the bony articular surfaces and slight hypertrophic bone change.

The shoulders showed minimal flattening of the articular surfaces of the humeral heads and slight irregularity of the glenoid fossae. Roentgenograms of the knees revealed mild flattening and depression of the medial tibial plateaus. No abnormalities were seen in the skull or mandible. The hands were normal; there was slight flattening of the distal articular surfaces of both first metacarpals in the feet.

At surgery, the right femoral head and acetabulum were visibly roughened and pitted, with obvious cyst formation. Bony spurring was present along the periphery of the femoral head. A cup arthroplasty was performed on the right hip.

CASE II. A 38 year old Negro male school custodial worker complained of pain and stiffness in both hips, especially the left hip, for

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the past 2 years. He had marked stiffness in the hips upon awakening in the morning. The hip pain occurred on prolonged standing or walking, and was aggravated by motion. He had occasional joint aches, especially in the hips and the knees, for over 20 years.

The patient stated that his 5 brothers were all short and were similarly affected. His 2 sisters were of normal appearance and were tall in height. His parents were not affected, while one paternal grandfather was said to be short in stature. Two of the brothers were available for roentgenographic study (Cases III and IV).

Physical examination revealed that the patient had a short trunk and relatively long extremities; his fingertips reached his knees in the erect position. His height was 5 feet $2\frac{1}{2}$ inches. He walked with a bilateral hip limp, more severe on the left.

Roentgenograms of the entire spine revealed flattening of all the vertebral bodies (Fig. 1*A*). The bulging of the posterior two-thirds of the vertebral bodies seen in Case I was present in some of the cervical and dorsal vertebrae. Most of the vertebral bodies were uniformly flattened; some were slightly wedged anteriorly or slightly biconcave. In frontal projection, the vertebral bodies had a widened transverse diameter. The disk spaces appeared normal in the dorsal spine, but widened in the lumbar spine. The odontoid process was moderately hypoplastic. There was a mild lumbar scoliosis.

The chest had a normal anteroposterior diameter, but a wide transverse diameter; the ribs were unremarkable. The segments of the body of the sternum were incompletely fused. The configuration of the pelvis was unremarkable, but the iliac bones were slightly small. The skull and mandible were normal.

In the hips, the femoral heads were severely flattened and deformed, with irregularity of their articular surfaces. Prominent sclerotic and cystic bone changes were present in both femoral heads. The hip joint spaces were moderately narrowed, especially centrally and superiorly. The acetabula had slight bone sclerosis and irregularity. No hypertrophic bone changes were evident. The femoral necks were short. Several ossified bodies were seen medial to and overlying both femoral necks, apparently in the hip joints (Fig. 4*A*).

The knees showed broadening of the ends of the femora and the tibiae. There was slight irregularity of the articular surfaces, including the patellae; the femoral surfaces were flattened and the tibial surfaces were depressed. The knee joint spaces were slightly narrowed (Fig. 5, *A* and *B*). Similar changes were present in

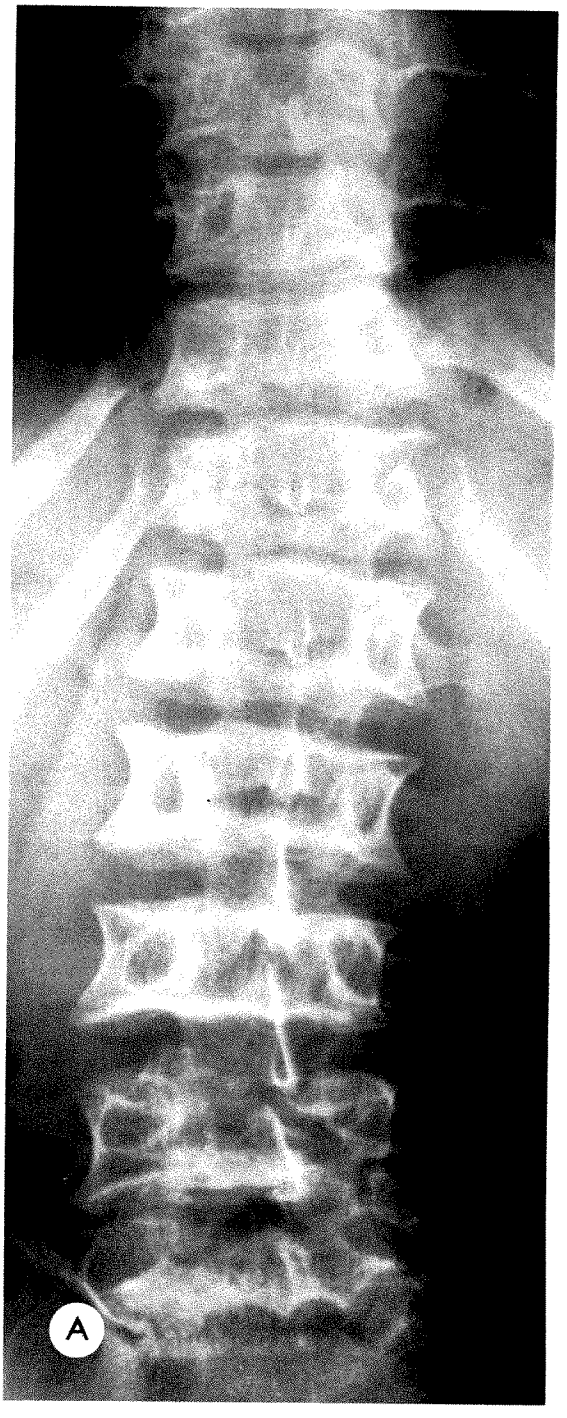



FIG. 1. (*A*) Frontal roentgenogram demonstrates flattening of the vertebral bodies of the dorsolumbar spine.

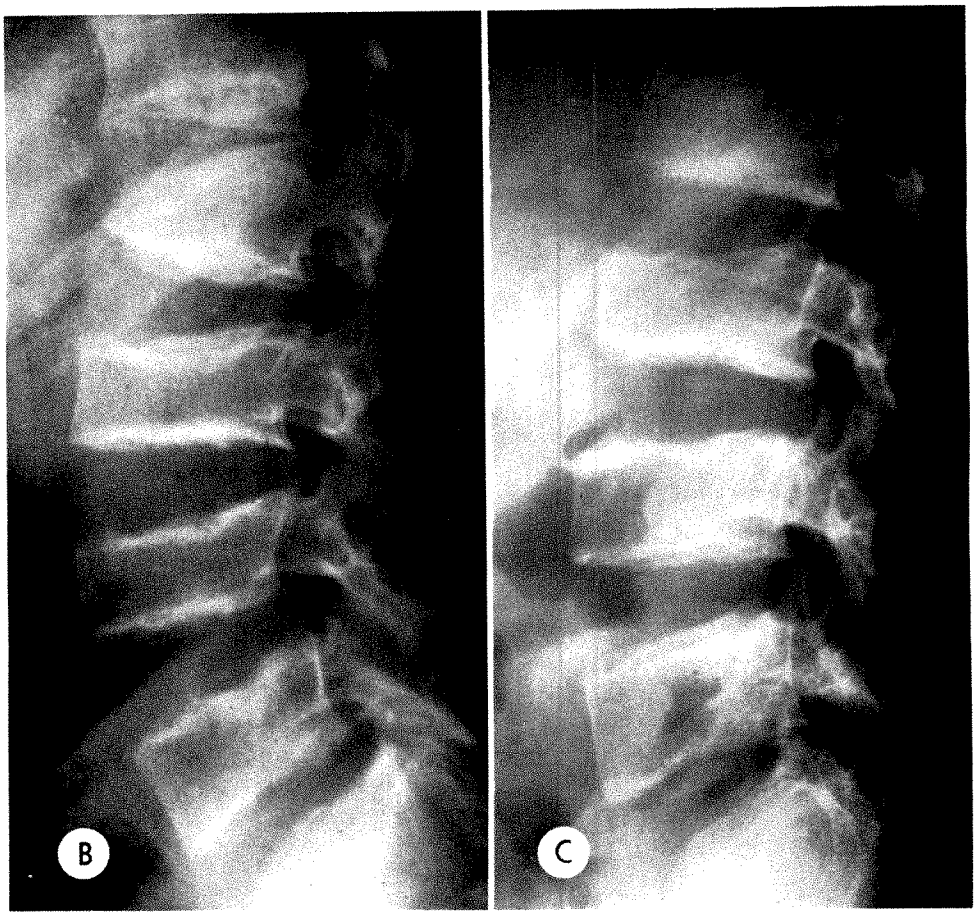



FIG. 1. (*B* and *C*) Lateral roentgenograms of 2 other patients (Cases III and IV) reveal flattening of all of the vertebral bodies. There is some variation in the shapes of the bodies, but most are rectangularly flattened. The disk spaces are wide.

both ankles, with the deformity being more severe in the talus than in the tibia.

Roentgenograms of the shoulders revealed flat and shallow glenoid fossae, with irregularity of the articular surfaces and slight subchondral cyst formation. The humeral articular surfaces were normal. The elbows showed moderate flattening of the articular surfaces of the distal humeri. The wrists had slight flattening of the articular ends of the radii; the distal ulnae were short. The navicular and lunate bones were small, flat, and severely deformed; the radial articular surfaces of the navicular bones showed irregularity and fragmentation.

In the hands, the only major changes were in the metacarpophalangeal joints: the metacarpal heads were broad, flat, and irregular; the proximal phalanges were also flat, and the joint spaces were moderately narrowed. In the feet, there were similar, less severe, changes in the metatarsophalangeal joints. Flattening of the articular surfaces of all of the tarsal bones was also noted.

At surgery, the left femoral head was broadened and flattened. There was considerable degeneration of the joint cartilage. Several loose bodies were present. A vitallium cup arthroplasty was performed on the left hip.

CASE III. The 38 year old twin brother of the patient described as Case II had occasional pains in the hips, knees, and ankles for many years. His height was 5 feet, 5 inches. Roentgenograms of the spine, skull, pelvis, and joints showed remarkably similar findings to those of Case II (Fig. 1*B*; 3*A*; and 8).

CASE IV. This 36 year old male sibling of the patients described as Cases II and III had occasional pains in the hips and ankles for many

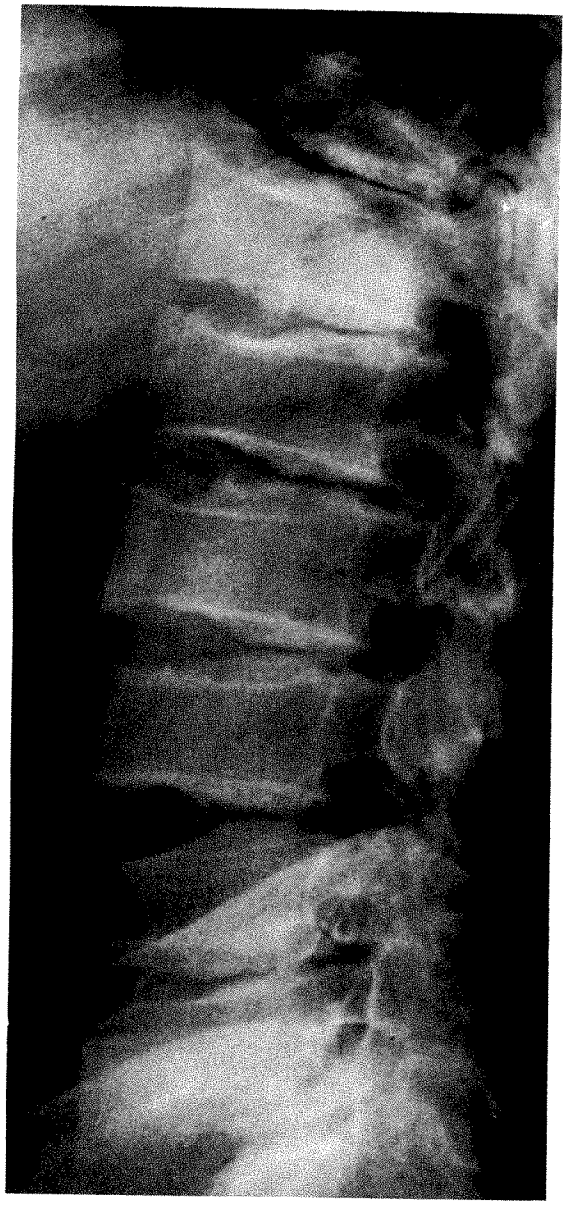


FIG. 2. Lateral roentgenogram shows bulging of the posterior portions of the vertebral bodies, and severe narrowing of the anterior portions of the bodies. Marked loss of disk spaces is noted, especially posteriorly (Case 1).

years. His height was 5 feet, 2 inches. Again, the roentgenographic examination was similar to that of Case II (Fig. 1*C*; 3*B*; and 4*B*). The shoulders were, however, more severely affected than in the other cases: the humeral articular surfaces were markedly deformed, flattened, and irregular; the glenoid fossae were flattened and hypoplastic, and there were several ossified bodies in the shoulder joint spaces (Fig. 7).

## CLINICAL AND ROENTGENOLOGIC FINDINGS

After an asymptomatic childhood, the patient with spondylo-epiphyseal dysplasia tarda begins to complain of pain in the hips, and less often of pain in other major joints of the extremities. Physical examination reveals shortening of the trunk, with normal length of the extremities. The involved joints often have limited or painful motion.

The principal roentgenographic findings are in the spine and in the major joints. Universal vertebra plana is the roentgen hallmark of the disease, appearing in the dorsolumbar spine as well as in the cervical spine (Fig. 1, *A–C*; and 3, *A* and *B*). The vertebral bodies may show either uniform flattening, or anterior wedging (Fig. 1, *B* and *C*). An unusual bulging of the posterior and central portions of the upper and lower vertebral end plates, with anterior narrowing, has been described.[4,7] This finding resembles the central "tongue" of Morquio's disease. It was prominent in Case 1, but was seen in only some of the vertebrae in our other cases (Fig. 2).

The roentgen changes in the spine have been described in patients of 5 and 6 years of age,[7,13] but usually become evident during adolescence. The intervertebral disk spaces are normal or wide (Fig. 1, *B* and *C*), but are eventually often narrowed by the progression of the disease (Fig. 2). Moderate dorsal kyphosis is not uncommon; mild scoliosis is occasionally found. The odontoid process also participates in the spinal underdevelopment and is hypoplastic, as in Morquio's disease.[5,15]

Premature or precocious osteoarthritis of major joints of the extremities almost always appears in adult life.[6] The degenerative arthritis is secondary to the developmental deformity of the articular bony surfaces and the developmental abnormality of the joint cartilages. The arthritis is most severe in the hips (Fig. 4, *A* and *B*), where surgical intervention may be required. The other major joints, especially

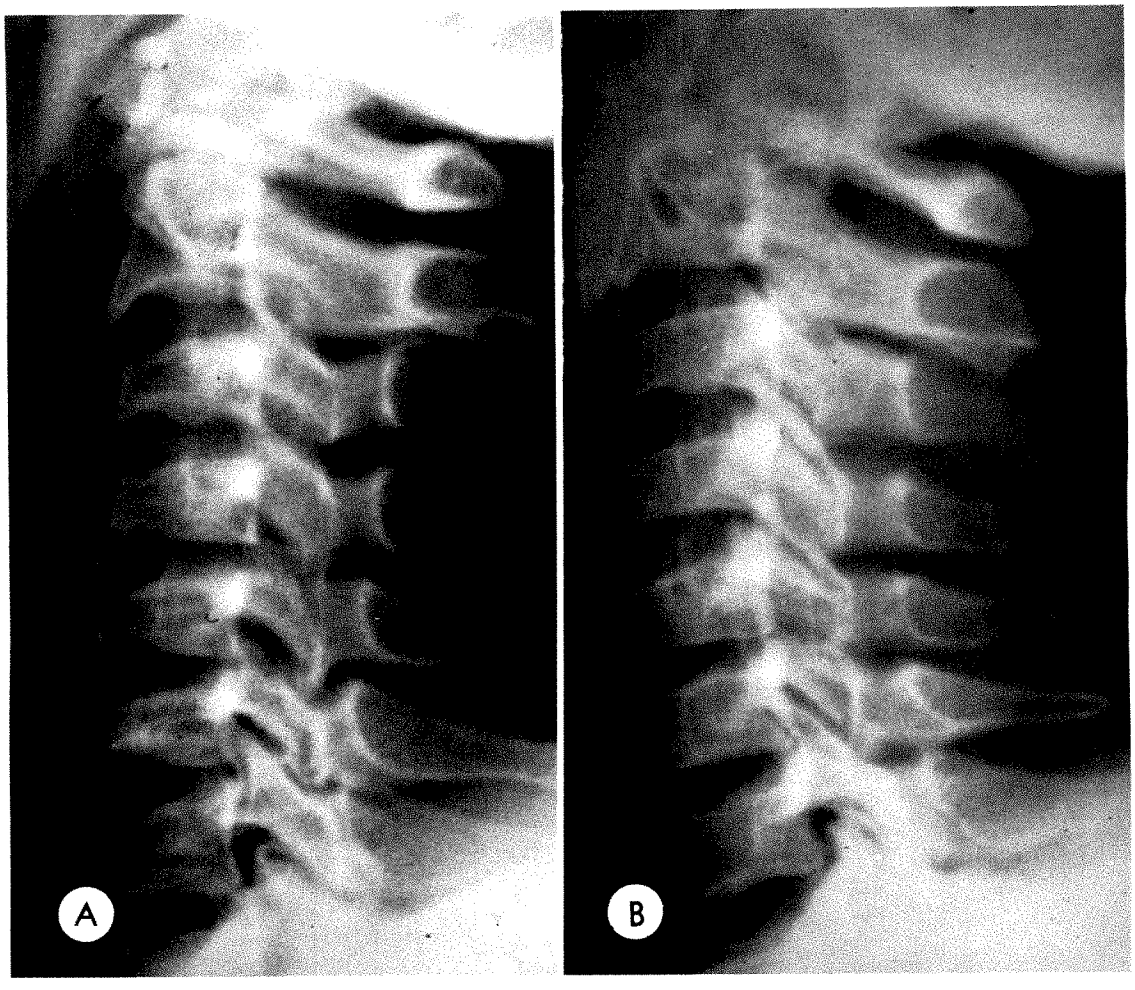



FIG. 3. (*A* and *B*) Lateral roentgenograms of the cervical spine of Cases III and IV disclose changes similar to those seen in Figure 1, *B* and *C*, but of less severity. Hypoplasia of the odontoid process is present, but could not be easily reproduced.

the other weight-bearing joints, the knees and the ankles, are commonly involved; changes in the shoulders are not unusual. The joint abnormalities are manifest roentgenologically by flattening and irregularity of the articular surfaces and by narrowing of the joint spaces (Fig. 5, *A* and *B*; 6, *A* and *B*; and 7).

The long bones are normal in length and in their metaphyseal and diaphyseal segments. The bones of the hands and feet are minimally involved in most cases, but significant findings were present in 3 of our 4 patients (Fig. 8). As in Morquio's disease, there were hypoplastic changes in the carpal bones.[5,8] The ribs are often widened in their lateral aspects and crowded together posteriorly by the shortened spine. The pelvis may be normal or may have reduced dimensions, especially the iliac bones; the greater sciatic notches are normal, and there is no resemblance to achondroplasia.

## DISCUSSION

The frequent presentation of cases of spondylo-epiphyseal dysplasia tarda as premature osteoarthritis of a major joint should be emphasized. Such cases of osteoarthritis at an early age, especially of the hips, should have a roentgenologic survey of the skeleton. The finding of universal vertebra plana in the spine will reveal the

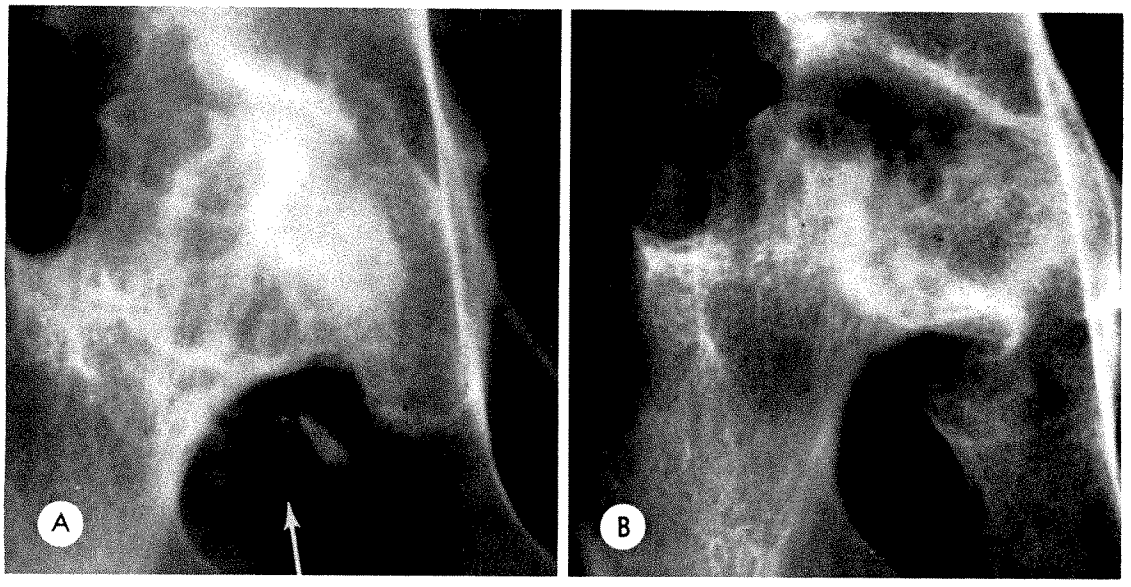



FIG. 4. (*A* and *B*) Frontal roentgenograms of hips of Cases II and IV show severe deformity of the femoral heads, with sclerotic and cystic bone changes. Ossified joint bodies are present (arrow). The femoral necks are shortened and widened.

true nature of the disease, and will identify other susceptible family members. Spondylo-epiphyseal dysplasia tarda has thus far been definitely identified only in males, and has been shown to have a recessive, sex-linked genetic transmission.[7] Both Negroes and whites are affected by this hereditary disease.

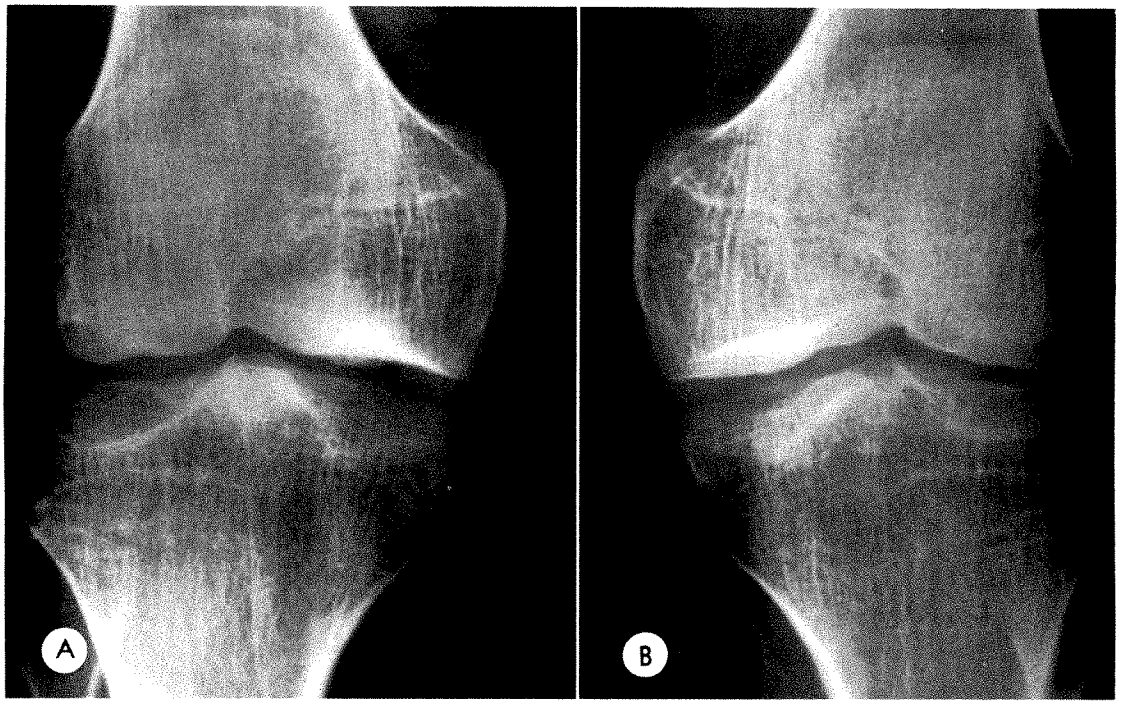



FIG. 5. (*A* and *B*) Knee roentgenograms reveal flattening of the femoral articular bony surfaces and depression of the tibial plateaus.

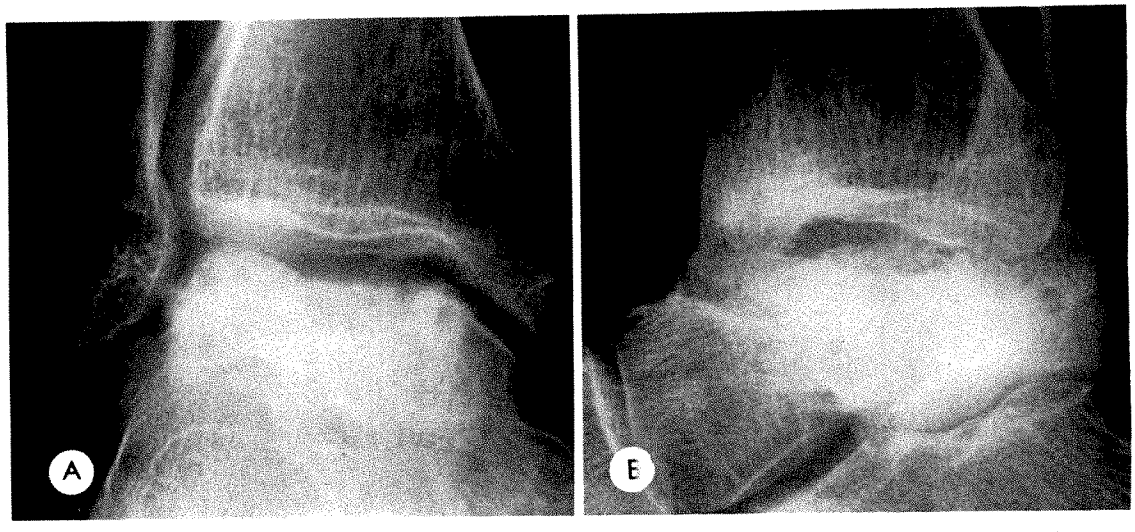



FIG. 6. (*A* and *B*) Frontal and lateral roentgenograms of the ankle demonstrate marked deformity and sclerosis of the superior aspect of the talus, with depression and irregularity of the articular surface.

Premature osteoarthritis of multiple joints occurs principally in two other diseases: multiple epiphyseal dysplasia and ochronosis. In multiple epiphyseal dysplasia tarda, the roentgenograms of the joints in the adult resemble those of the adult with spondylo-epiphyseal dysplasia tarda. Universal vertebra plana, however, is absent and the dwarfism is of the short-limb type.[3] Universal vertebra plana is also absent in ochronosis, a disease caused by the deposition of ochronotic pigment in connective tissues, including articular and intervertebral cartilages. Patients with ochronosis exhibit characteristic calcification in the intervertebral disks and excrete homogentisic acid in the urine.



FIG. 7. Frontal roentgenogram of the shoulder (Case IV) shows irregularity and flattening of the humeral articular surface. The glenoid fossa is shallow. Several ossified joint bodies are visualized (arrows).

The universal vertebra plana, or platyspondyly, found in spondylo-epiphyseal dysplasia tarda occurs in only a few diseases. Some cases of achondroplasia have mild flattening of the vertebral bodies, especially in the lumbar spine, but have accompanying characteristic changes in the metaphyses of the extremities, in the spinal canal, in the pelvis and in the skull.[1] Patients with the pseudoachondroplastic form of spondylo-epiphyseal dysplasia often show slight platyspondyly, but the cardinal abnormalities are in the extremities. The limbs are shortened and the epiphyses and metaphyses are markedly irregular, fragmented and deformed; the changes in the extremities may resemble advanced achondroplasia, but the differentiation from achondroplasia has been delineated by Ford *et al.*[2] and by Rubin.[14]

Rubin[15] has separated dysplastic errors in epiphyseal development into (1) failure of development of articular cartilage and bone, and (2) failure of ossification of the epiphy-



FIG. 8. Roentgenogram of the hand and wrist reveals hypoplasia and deformity of the navicular and lunate bones, with fragmentation of the navicular. The radial articular surface is flattened. The metacarpal heads are enlarged, flattened, and irregular. The adjacent articular surfaces of the proximal phalanges are also flattened, and the metacarpophalangeal joint spaces are narrowed. Only very minimal changes are present at the interphalangeal joints.

seal center. The latter is represented by multiple epiphyseal dysplasia, which has both congenita and tarda forms. The failure of development of articular cartilage and bone is divided into Morquio's disease and spondylo-epiphyseal dysplasia tarda, being severe and mild expressions of the same developmental error.

It is Morquio's disease,[8,11,12] with its universal vertebra plana, that most clearly resembles the over-all appearance of spondylo-epiphyseal dysplasia tarda. Morquio's disease, or spondylo-epiphyseal dysplasia congenita, represents the severe defect in the formation of articular cartilage and bone. The interference with the development of articular bone leads to irregularly shaped, flattened, and deformed epiphyses, and rapid evolution to degenerative arthritis. The same defect of articular cartilage and bone in the vertebral bodies produces platyspondyly. These severe deformities are noted soon after the child begins to walk, and cause increasing disability. The hips and knees, the major weight-bearing joints, are worst affected.

In Morquio's disease, as in spondylo-epiphyseal dysplasia tarda, the extremities are of approximately normal length, since the articular bone is not involved in bone length. In some severe cases of Morquio's disease, the defective epiphyses allow secondary injury of the growth plates by normal joint stresses, thus shortening the extremities. Even in such cases, however, the spine is more markedly shortened than the extremities. The joint deformities in Morquio's disease, aggravated by muscular weakness and relaxation of ligaments, result in progressively crippled and sickly children. In contrast, spondylo-epiphyseal dysplasia tarda appears as degenerative joint disease of varying severity and disability in young and middle-aged adults.

The clinical and roentgenologic approach to such a systemic disease as spondylo-epiphyseal dysplasia tarda remains vital until a basic biochemical defect can be found. Hurler's disease, and more recently Morquio's disease, have been discovered to show characteristic abnormalities of the urinary acid mucopolysaccharides.[5,9,10] The mucopolysaccharides are important constituents of connective tissue, especially cartilage. It is hoped that the goal of biochemical definition of spondylo-epiphyseal dysplasia tarda, like all genetic diseases, will be attained.

## SUMMARY

Spondylo-epiphyseal dysplasia tarda is a hereditary chondrodysplasia which appears in adolescence or adult life as premature osteoarthritis of the spine or of major joints of the extremities. It appears to be the mild form of the same abnormal-

ity present in Morquio's disease. The characteristic findings are universal vertebra plana in the spine and flattening and irregularity of the articular surfaces of the major points. More cases will be uncovered by carefully examining the skeleton in all patients with premature osteoarthritis.

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# REITER'S SYNDROME AND PSORIATIC ARTHRITIS*

## THEIR ROENTGEN SPECTRA AND SOME INTERESTING SIMILARITIES

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REITER'S syndrome and psoriasis may both be manifest in joint pathology. Ordinarily the diagnosis of these two diseases is rather straightforward, clinically and roentgenographically. However, in some cases, the clinical findings, the roentgenographic findings, or both may coalesce, causing diagnostic, if not therapeutic, confusion. The coexistence of these two diseases, or the evolution of one into the other, has been mentioned previously by Weinberger *et al.*[17] in 1962 and Wright and Reed[21] in 1964. The latter reported 12 cases in which the two diseases were intimately associated.

In this paper, the roentgen manifestations of each of these diseases will be set forth and the interesting area of overlap between the two will be considered.

All roentgenograms from 32 cases of Reiter's syndrome and 39 cases of psoriatic arthritis seen at the National Institutes of Health between 1953 and 1962 were studied.

### REITER'S SYNDROME

Reiter's syndrome was named after Hans Reiter,[14] who reported a case of urethritis, arthritis and conjunctivitis, following an episode of bloody diarrhea in 1916. Urethritis, arthritis and conjunctivitis have subsequently remained as the essential components of this syndrome, but some feel that the eye involvement is so often minimal or insignificant that it need not necessarily be present to make the diagnosis.[15,18,19,21] Several other manifestations are inconsistently associated with this syndrome; these are: (a) skin involvement (keratodermia blennorrhagica); (b) nail involvement; (c) balanitis circinata; (d) oral mucosal lesions; (e) prostatitis; (f) iritis; and (g) diarrhea (as in Reiter's original case). The skin lesions are of particular interest because they are considered by most experts to be indistinguishable from pustular psoriasis.

Etiologically, Reiter's syndrome remains an enigma. The latest opinion[16,17,19,21] favors an infectious origin perhaps with a hypersensitivity component.[21] Some have postulated a viral etiology, but Weinberger's group found evidence tending to incriminate pleuropneumonia-like organisms.[16] Indiscrete sexual exposure is a very frequent precursor of symptomatology.

Several descriptions of the roentgenographic manifestations of Reiter's syndrome have appeared in the literature, the most recent in the radiologic literature being Weldon and Scalettar's report from Walter Reed General Hospital in Washington, D. C., in 1961.[18] Murray, Oates and Young,[12] and Reynolds and Csonka,[15] authored articles on the subject in 1958, and Weinberger *et al.*,[17] Kulka,[8] and Weinberger[16] discussed the results of an extremely thorough long-term study of 16 patients in 1962. Our experience with the disease parallels that of previous authors.

In selecting case material for our evaluation, we elected, as have several others in the past,[15,18,19,21] to include cases of venereal arthritis without clear-cut conjunctivitis, although the majority of our patients did exhibit eye symptoms at some time. No difference in the roentgenographic features was found between those patients with eye signs and symptoms and those without.

### DISTRIBUTION OF LESIONS

As has been the experience of other authors, we found the highest incidence of Reiter's arthropathy in the joints of the lower extremities. Involvement of the sacroiliac joints and the joints of the upper

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extremities was less common but not infrequent (Table 1). Knee involvement was most common in our experience (80 per cent of all cases), and ankle and foot involvement was the next most common (60 per cent and 53 per cent, respectively). Positive roentgen features were found about the hands and wrists nearly one-third of the time, and this is a significantly higher incidence than the 15 per cent reported by Weldon and Scalettar.[18] Our low incidence of sacroiliac involvement (24 per cent) is best explained by the fact that 22 of our 32 patients had a history of less than 1 year of symptoms. Other workers have indicated that the incidence of sacroiliac involvement in Reiter's syndrome increases dramatically as the disease becomes more chronic. Mason and associates[11] reported an incidence of 8 per cent in patients whose history was less than 5 years, and an incidence of 54 per cent in patients with a history longer than 5 years. Of 7 patients in our series with sacroiliac abnormalities (Fig. 1), 4 had long (greater than 1 year) histories. Only 1 had a short (2 months) history and 2 were entirely lacking in low back symptoms.

Typical calcaneal spurs (*i.e.*, spurs with definite erosions and/or wooly periosteal proliferation) were likewise found infrequently in our series, undoubtedly because we had relatively few cases with long histories. Of our 10 chronic cases, however, 4 or 40 per cent had typical calcaneal spurs (Fig. 2, *A* and *B*). Elbows, shoulders and hips were infrequently involved, and only in patients with histories in excess of 5 years.

TABLE I

FREQUENCY AND DISTRIBUTION OF JOINT INVOLVEMENT IN REITER'S SYNDROME

| Locus | Cases Evaluated | No. of Cases Involved | Per Cent |
|---|---|---|---|
| Hands and wrists | 32 | 9 | 29 |
| Elbows | 9 | 2 | 22 |
| Shoulders | 10 | 2 | 20 |
| Feet | 32 | 17 | 53 |
| Ankles | 30 | 18 | 60 |
| Knees | 28 | 22 | 80 |
| Hips | 29 | 2 | 7 |
| Sacroiliacs | 29 | 7 | 24 |

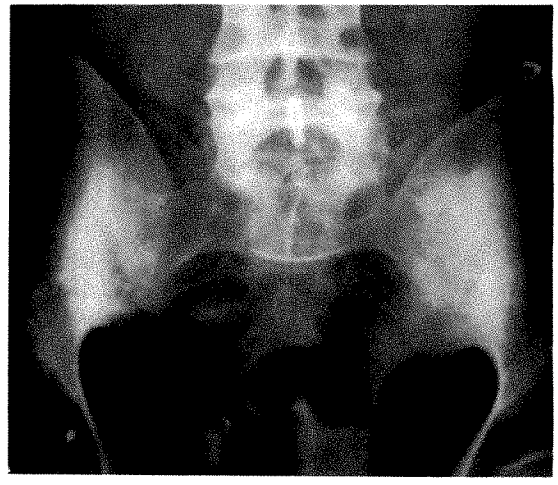


FIG. 1. An anteroposterior roentgenogram of the sacroiliac joints in this patient with several years of intermittent back pain shows nearly total obliteration of the joint spaces bilaterally, with extensive associated juxta-articular sclerosis.

ROENTGEN MANIFESTATIONS

A. *Periostitis.* Because of the interest expressed by previous authors in periostitis as a more or less characteristic finding in Reiter's syndrome, we searched our cases very carefully for this manifestation. We were interested to find that less than one-fourth of the involved joints (19 of 81) showed periostitis, either of the linear or of the fluffy, wooly variety. We found linear periosteal new bone (Fig. 3, *A* and *B*) largely in acute cases (a few weeks' to a few months' history), whereas fluffy, wooly periosteal new bone proliferation (Fig. 2, *A* and *B*; and 4) were not seen unless a long history was elicited (this was particularly true of the calcaneal spurs). Mason *et al.*,[11] in 1959 found a somewhat higher incidence of periostitis, particularly about the mal-

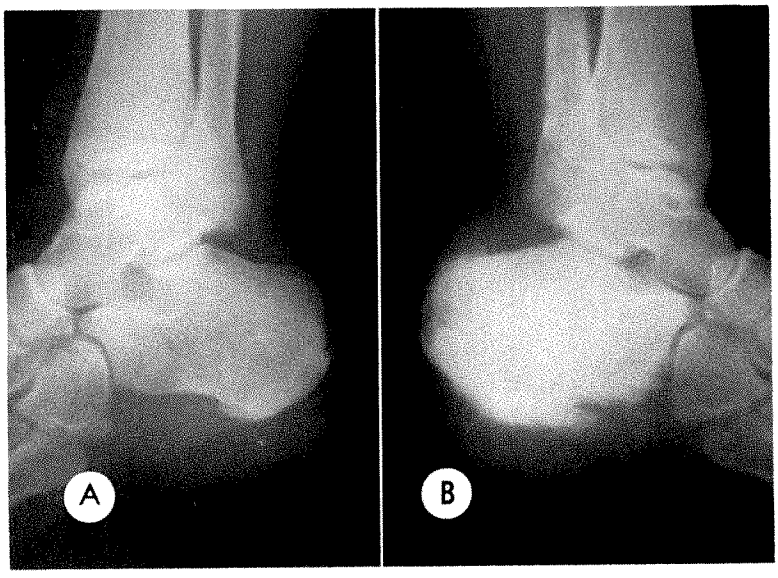



FIG. 2. (*A* and *B*) Lateral os calcis views on this patient with long-standing heel pain show unilateral, exuberant, irregular, wooly periosteal new bone proliferation resulting in a striking increase in size and density of the os calcis on one side. Previous authors have considered this appearance essentially pathognomonic of Reiter's syndrome.

leoli. Interestingly, they also indicated that periostitis is to be found nearly as frequently in rheumatoid arthritis as in Reiter's syndrome. Patients with psoriatic arthritis seemed to exhibit this feature occasionally,[5] although not as frequently as those with Reiter's syndrome.

In evaluating a pathologic articulation exhibiting periostitis, one should think of rheumatoid arthritis, Reiter's syndrome, infectious arthritis, and also psoriatic arthritis. The differentiation between these must and can be made on the basis of other clinical and roentgenographic features.

B. *Osteoporosis.* We also looked carefully for localized osteoporosis, since some disagreement seems to have existed in the past regarding its incidence. Mason *et al.*,[11] declined to evaluate their subjects for osteoporosis, referring to Kellgren and Lawrence who suggested in 1957 that observer error could arise in assessing lesser degrees of osteoporosis.

Others[16,17,18] have felt that bone atrophy was a useful diagnostic feature. Our experience supports the latter view. Over 50 per cent of all joints involved in our 32 patients showed definite *localized* osteoporosis (Fig. 5, *A* and *B*), (*i.e.*, involving parts of 2 phalanges or 2 carpal bones on each side of an involved joint, with all other osseous structures appearing normal). The incidence rises to greater than 75 per cent if only the acute cases are evaluated. As one would expect, the well localized zone of osteoporosis correlates 100 per cent with the location of clinical involvement (pain and usually swelling and heat). This very localized osteoporosis is more like what one would expect with an infectious arthritis than with the collagen arthritides. Osteoporosis usually is regional and related, at least largely, to disuse in rheumatoid arthritis. Osteoporosis, either localized or generalized, is uncommon with psoriatic arthritis.

C. *Thickened periarticular tissues.* This very nonspecific feature (Fig. 6) was the most common encountered in our cases, representing an incidence of 69 per cent over-all and greater than 80 per cent in cases with acute symptoms. The correlation of this finding with local pain, swelling and heat was very high. However, even in

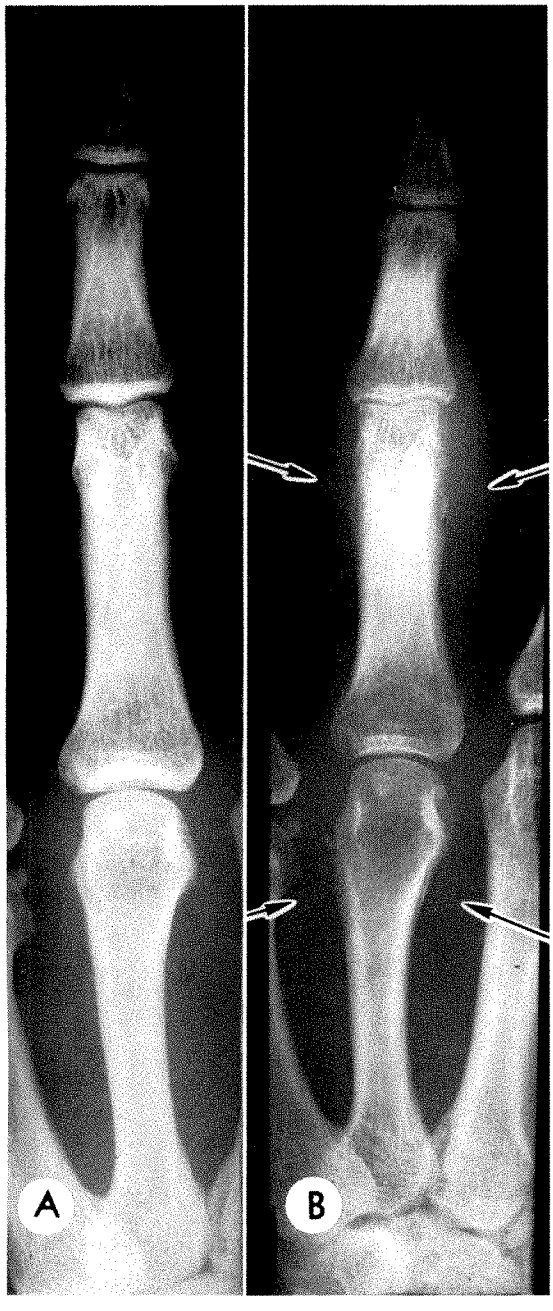



FIG. 3. (*A*) An anteroposterior roentgenogram of the third finger before clinical Reiter's involvement appears normal. (*B*) An anteroposterior roentgenogram of the same finger several weeks after the onset of local pain, warmth and swelling. Both pure linear (lower right arrow) and somewhat fluffy-appearing (lower left arrow) periostitis are apparent along with the local soft tissue swelling (upper arrows) and equivocal joint space narrowing.

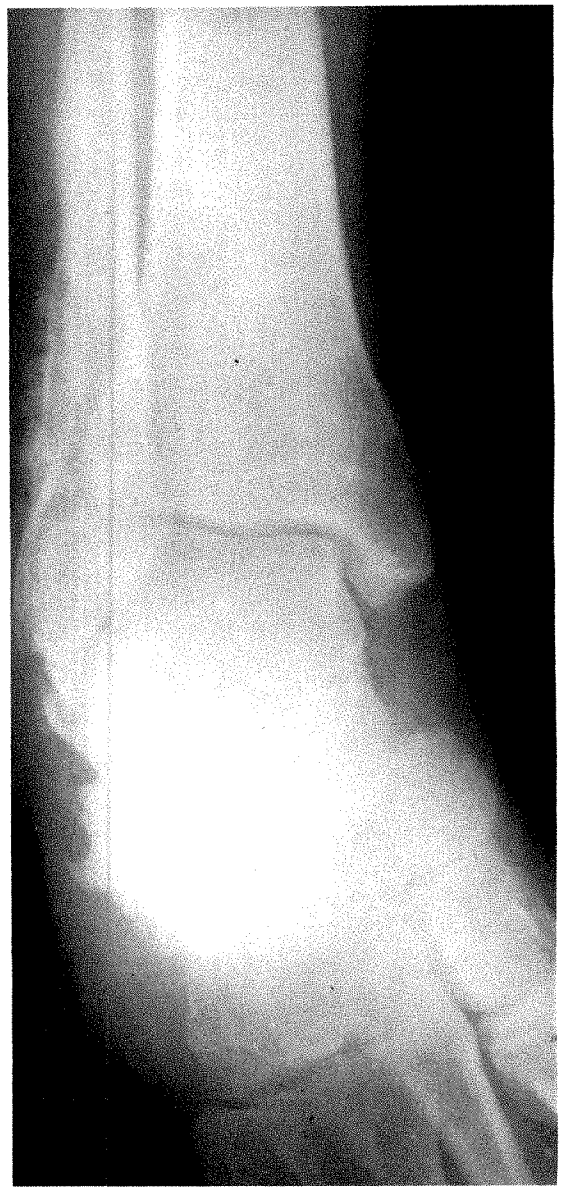


FIG. 4. An anteroposterior roentgenogram of the ankle of this patient with a long history of local symptoms shows exuberant wooly periostitis of the lateral malleolus, astragalus and calcaneus. The ankle joint spaces are intact.

patients with long histories and clinically rather quiescent joints, some thickening of soft tissues was frequent. Mild osteoporosis and thickened periarticular tissues were usually the only roentgen features about sites of knee involvement.

D. *Joint narrowing or destruction.* An

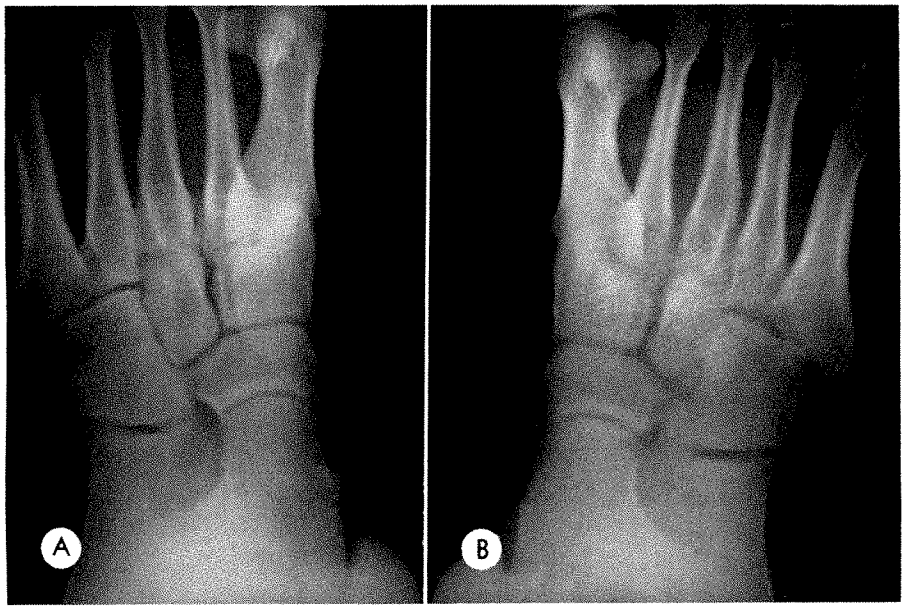



FIG. 5. (*A* and *B*) Oblique roentgenograms of both ankles show no abnormality on the clinically asymptomatic side (*A*) and local tarsal osteoporosis on the side manifesting local pain (*B*). This subcortical atrophy simulates joint involvement because the cortices appear much less distinct than on the opposite normal side.

evaluation of the 81 anatomic areas involved by Reiter's syndrome in our 32 cases revealed narrowing or destruction of one or more joints in only 18 of these areas, or an incidence of 22 per cent. Many joints superficially appeared to be narrowed because of the local juxta-articular bone atrophy on both sides of an involved joint. Of the 18 involved areas, 7 were the sacroiliac joints. However, of all other joints involved, joint narrowing or destruction was found in a location other than the hands and feet (including intercarpal and intertarsal joints) only once (narrowing of the knee joint space). Involvement of the knees and ankles was frequent, but the findings usually were limited to soft tissue swelling, osteoporosis and occasional periostitis. The smaller joints of the tarsus, carpus, hands and feet were involved somewhat less frequently than the knees and ankles, but, when involved, showed a much higher incidence of joint narrowing or destruction.

Surprisingly, roentgenographic abnormality of the joint space was as frequent in our acute cases (Fig. 6), (less than 1 year—in most cases, a few weeks to 5 or 6 months) as in the chronic cases (over 1 year—in most cases, several years). Of all abnormal peripheral joints involved in our chronic cases, 43 per cent showed narrowing or destruction of the joint space. A similar evaluation of all of our cases (two-thirds of which were acute) revealed an incidence of 40 per cent. This would certainly suggest that, although many peripheral joint abnormalities are reversible in acute Reiter's syndrome, if a peripheral joint space is going to be irreversibly damaged, it is as likely to happen in the relatively acute case as in the chronic case.

Permanent damage to the joint in Reiter's syndrome has been underestimated by some in the past, but our findings tend to agree with the experience of Weinberger *et al.*,[17] who found, in a long term follow-up study of 16 cases, that more than 50 per cent end up with permanent joint damage.

E. *Tendon calcification-ossification.* Reynolds and Csonka[15] reported the occasional incidence of tendon calcification-ossification among their 60 patients (a Pellegrini-Stieda type, around the knee). We found this manifestation 7 times, representing an

incidence of less than 10 per cent of all involved joints. The knee was involved 3 times (Fig. 7), the shoulders twice, the elbow once and the foot once. This calcification and/or ossification appeared to be within or surrounding tendinous insertions and was found exclusively in cases with longstanding disease.

F. *Calcaneal spurs.* Of 30 patients having roentgen studies of the os calcis, only 4 (14 per cent) showed the characteristic wooly periosteal new bone and/or erosion of well developed calcaneal spurs (Fig. 2, *A* and *B*). All 4 were plantar spurs. This incidence is low compared with the 30 per cent reported by others.[11,12,18] However, if we look only at our 10 chronic cases, our incidence becomes approximately that of previous authors.

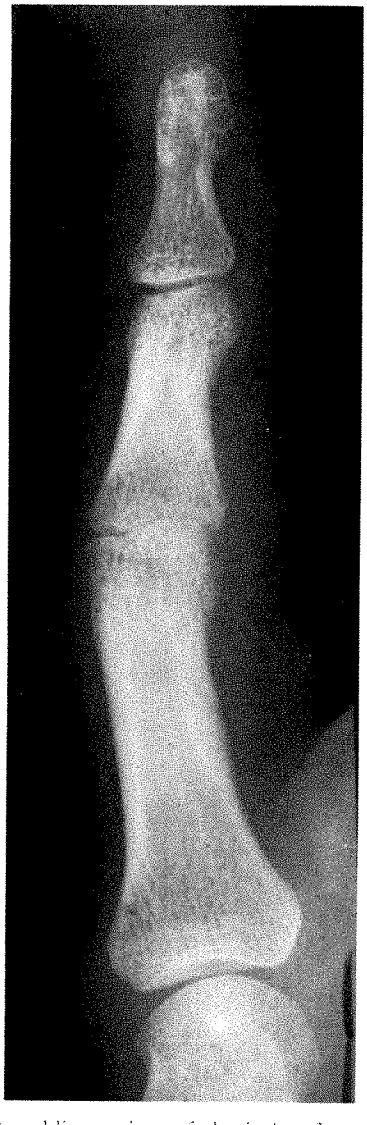


FIG. 6. An oblique view of the index finger on this patient with a 2 month history of local pain and swelling shows striking soft tissue swelling, definite proximal interphalangeal joint narrowing, and minimal juxta-articular osteoporosis.

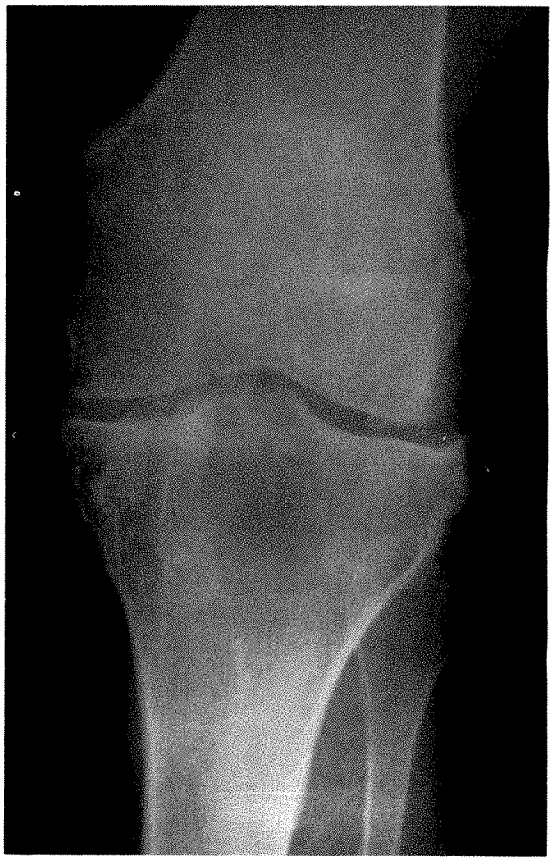


FIG. 7. An anteroposterior roentgenogram of the knee on this patient with a long history of local symptoms shows equivocal narrowing of the knee joint space and extensive calcification and ossification of tendinous insertions about the knee.

G. *Paravertebral ossification.* Paravertebral ossification has not been mentioned as a manifestation of Reiter's syndrome alone. Bywaters and Dixon,[5] in 1965, reported 4 cases of progressive paravertebral ossification, different from ankylosing spondylitis and senile ankylosing hyperostosis, in psoriatic arthritis, and 3 of their cases also had stigmata of Reiter's syndrome. We

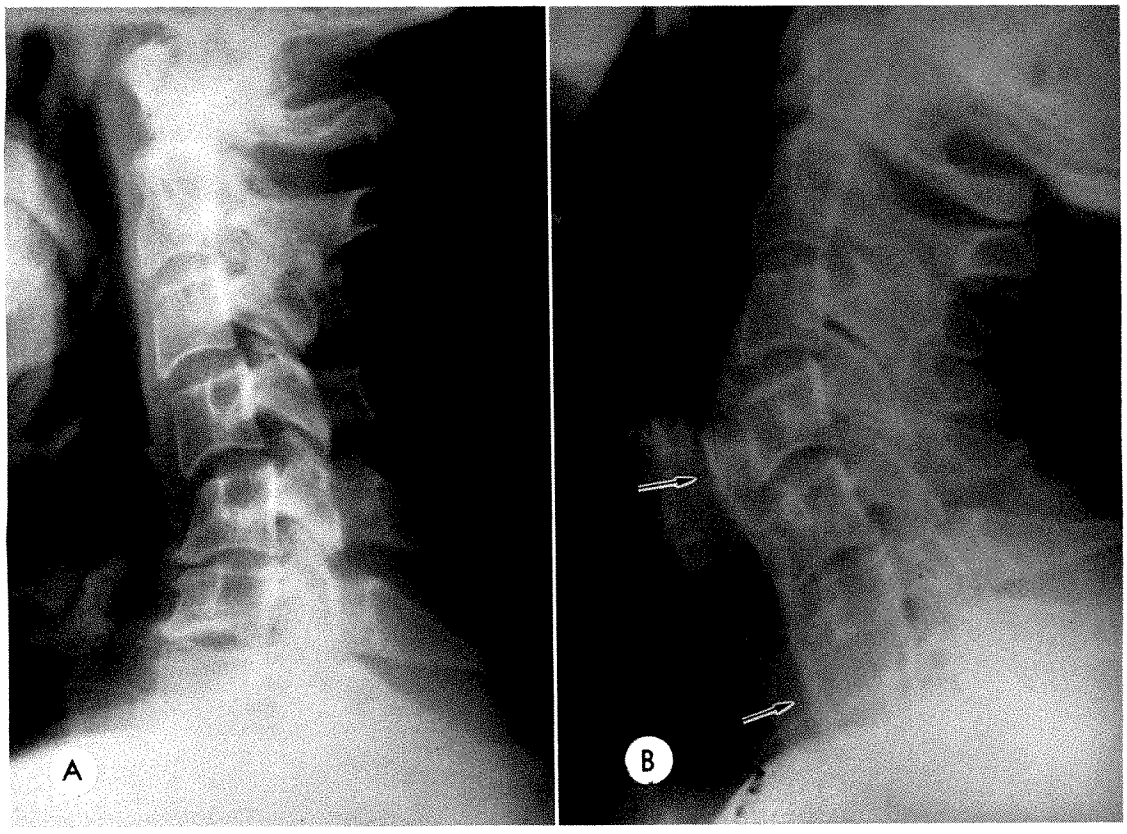



FIG. 8. (*A*) A lateral roentgenogram of the cervical spine shows diskogenic degenerative disease only. (*B*) A lateral view of the same spine less than 1 year later. In the interim the patient developed severe Reiter's arthropathy, which later seemed to evolve into psoriatic arthritis. Now a heavy anterior cloak of bone is noted (arrows). This patient had normal sacroiliac joints and no evidence of ankylosing spondylitis in the thoracolumbar spine.

were interested to find 3 cases in our series in which similar progressive paravertebral ossification was present, 2 in the cervical anterior prevertebral region (Fig. 8, *A* and *B*), and one in the lumbar region laterally. In 2 of these 3 cases, Reiter's syndrome appeared to evolve into or coexist with psoriasis.

## PSORIATIC ARTHRITIS

The existence of psoriatic arthritis as a specific entity has been stressed in the literature.[1,2,20] It is stated that psoriatics with arthritis, who are seronegative for rheumatoid factor, either exhibit changes of rheumatoid arthritis or a distinct arthropathy characteristically involving the distal interphalangeal (D.I.P.) joints. It may be associated with bony proliferation at the base of the great toe, while causing various degrees of destruction of many of the other joint surfaces, as well as joint fusion.[1,7,20] In many ways, our findings parallel those earlier described. However, we have had some difficulty in distinguishing psoriatic arthritis from the arthritis of Reiter's syndrome and, indeed, even in separating the "rheumatoid" and "non-rheumatoid" type of psoriatic arthritis. Our findings are summarized below.

### DISTRIBUTION OF LESIONS

Cervical spine studies were available on 27 patients, 5 (18 per cent) of whom had erosive changes of the apophyseal joints and 4 (16 per cent) of whom had abnormal atlantoaxial separation (Fig. 9). Those patients with abnormal mobility of the atlan-

toaxial joint all had roentgen evidence of systemic rheumatoid arthritis. Erosion at the apophyseal joints associated occasionally with dense paravertebral ligamentous calcification has been previously noted.[5,7]

Articular erosions were seen in 5 of 28 (18 per cent) lumbar spines reviewed. (One case of associated ankylosing spondylitis was observed.)

Arthritic changes in the sacroiliac joints were seen in 10 of 28 (36 per cent) cases, a similar incidence having been previously noted.[2,6,20] Erosion of the greater and lesser femoral trochanters bilaterally was seen in one case.

Ten of 30 (33 per cent) cases showed rheumatoid arthritic changes in the knee with joint space narrowing, articular destruction, and juxta-articular osteoporosis. Tendinous and/or peritendinous calcification was observed in 3 of 30 (10 per cent) cases. Periostitis was seen in 2 (7 per cent) patients.

Rheumatoid changes in the ankles were seen in 8 of 27 (30 per cent) cases, with peritendinous calcification or ossification in 4 (15 per cent). Dense periostitis was also seen in 4 patients (Fig. 10).

Changes of rheumatoid arthritis with no involvement of the D.I.P. joints of the feet were present in 9 of 32 (28 per cent) pa-

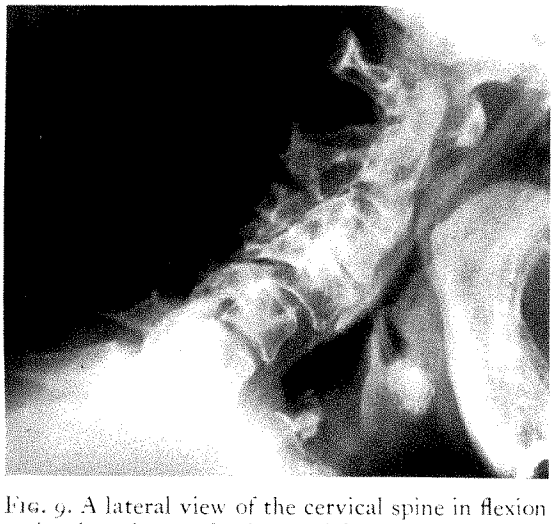


Fig. 9. A lateral view of the cervical spine in flexion showing abnormal atlantoaxial separation as well as subluxation of $C_4$ on $C_5$.



Fig. 10. An anteroposterior view of the ankle with dense periostitis of the medial malleolus.



Fig. 11. An anteroposterior view of the toes with bony proliferation along the phalangeal shafts and arthritis of the proximal interphalangeal joint of the great toe.

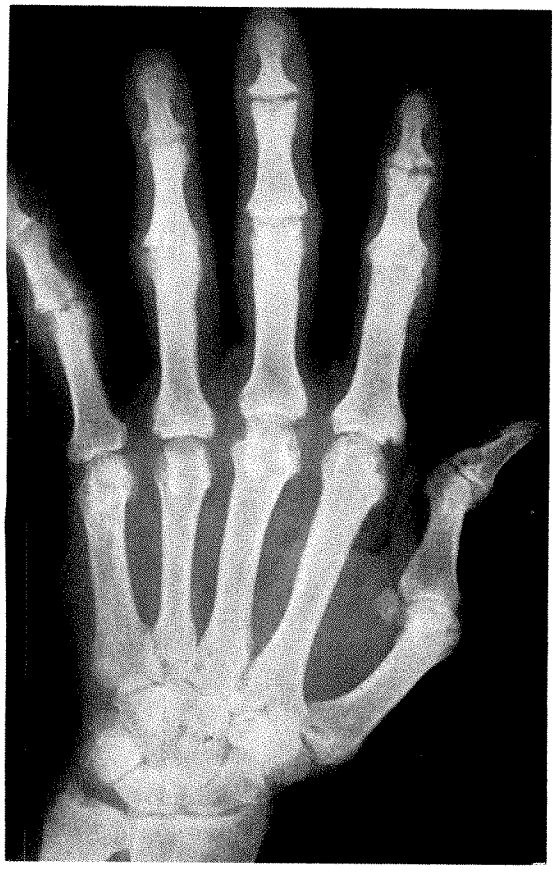


FIG. 12. An anteroposterior roentgenogram of the hand shows arthritic disease of the ulna, carpal, metacarpophalangeal and interphalangeal joints. Note D.I.P. joint widening.

tients. Proximal interphalangeal joint erosion of the great toe associated with D.I.P. joint erosion of the remaining digits was present in 10 of 32 (32 per cent) patients. Frequently, bony proliferation along the phalangeal shafts as well as tarsal and metatarsal joint destruction was present in this second group (Fig. 11). Metatarsal and phalangeal periostitis was observed in 4 of 32 (13 per cent) patients, all with chronic psoriatic arthritis. No definite correlation between psoriatic nail changes and D.I.P. joint disease could be established. This coincides with the experience of Baker *et al.*[2]

Five of 10 patients had articular destruction of the humerus. Two of these patients, both with systemic rheumatoid changes, had distal clavicular erosions.

Nine of 34 (26 per cent) patients had normal roentgen studies of the hands. Twelve (35 per cent) had changes indistinguishable from rheumatoid arthritis, while 13 (38 per cent) had destructive arthritis of the D.I.P. joints, frequently associated



FIG. 13. An oblique roentgenogram of the fifth digit shows linear periostitis of the metacarpal and phalangeal shaft.

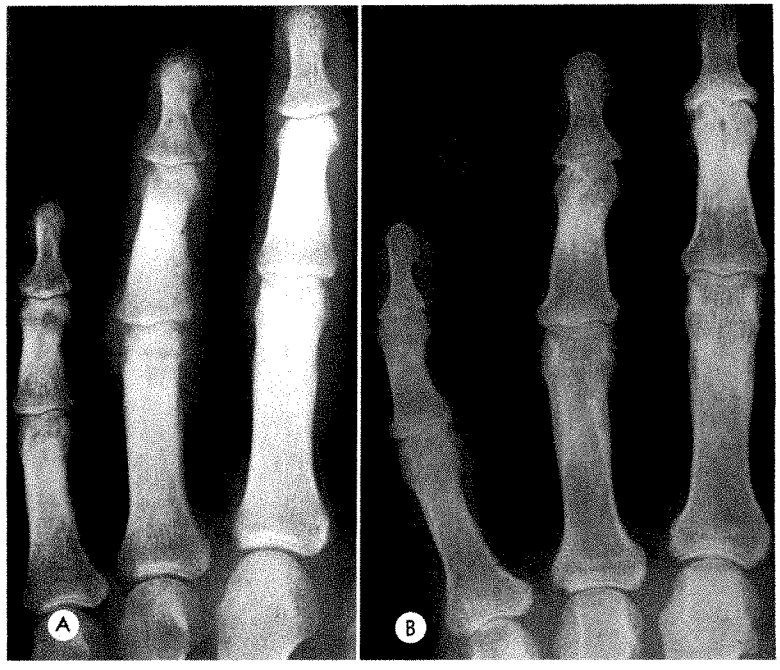



FIG. 14. (*A*) An anteroposterior roentgenogram of the hand shows swelling and juxta-articular osteoporosis of the proximal interphalangeal joint of the fourth digit. (*B*) A similar examination of the same patient 2 years later shows preservation of the proximal interphalangeal joint, but now demonstrates distal interphalangeal joint arthritis of the third digit and fusion of the distal interphalangeal joint of the fourth and fifth digits.

with carpal, metacarpal and proximal phalangeal arthritis (Fig. 12). Periostitis was present in 3 (9 per cent) patients (Fig. 13).

In our series the incidence, in the joints of the hands and feet, of several "characteristic" features of psoriatic arthritis (distal interphalangeal joint involvement 35 per cent, interphalangeal joint ankylosis 15 per cent, widened joint spaces 35 per cent, great toe involvement 35 per cent, distal tuft resorption 22 per cent) was comparable to the incidence of these features mentioned by Avila *et al.*[1]

*Comments.* The spectrum of psoriatic arthritis is confusing. Patients may present at one time with changes of acute rheumatoid arthritis; then improve, only to go on and develop stigmata of psoriatic arthritis with D.I.P. arthritis and joint fusion (Fig. 14, *A* and *B*). The opposite course was seen in 2 other patients who presented with monoarticular D.I.P. arthritis of the hand, only to rapidly develop an arthritis mutilans of both hands and feet as well as generalized charges of rheumatoid arthritis (Fig. 15, *A* and *B*). Although characteristic roentgen charges of psoriatic arthritis are frequently seen,[1,2,5,7,9,10] confusion with other arthritides may lead to difficulty in its roentgen diagnosis.

## DISCUSSION

In evaluating the roentgen features of Reiter's syndrome, we were interested to discover that 2 cases which displayed the severest arthritic changes had been clinically reassessed and diagnosed as pustular psoriasis, after having been classified for several years as chronic recurrent Reiter's syndrome with keratodermia blennorrhagica. This stimulated us to also evaluate 39 cases of psoriatic arthritis to see if there was a significant zone of overlap between Reiter's syndrome and psoriatic arthritis. We found that the overlap was limited but not insignificant.

The important prerequisite for confusion




FIG. 15. (*A*) An anteroposterior roentgenogram of both hands in a patient with arthritis mutilans. (*B*) A photograph of the same patient's hands.

is the presence of a cutaneous lesion. Reiter's arthritis without keratodermia blennorrhagica could not be confused with psoriatic arthritis, since the latter does not exist without skin involvement.

Given skin lesions, the causes for possible confusion are numerous: (1) Both diseases may have associated similar nail lesions; (2) both diseases may manifest progressive articular destruction in the peripheral joints of the extremities; (3) both diseases rather frequently show sacroiliac involvement (these changes are indistinguishable in the 2 diseases); (4) periostitis associated with arthritis is most frequent in Reiter's syndrome and nearly as common in rheumatoid arthritis, but it is not uncommon in psoriatic arthritis; (5) paravertebral ossification, apparently a rarity whatever the predisposing disease, occurs occasionally in psoriatics with stigmata of Reiter's syndrome; (6) peritendinous calcification-ossification is found in both diseases with a similar incidence (approximately 10 per cent); (7) etiologically, the similarity is at best conjectural (it is known that psoriatic arthritis exacerbates with streptococcal infections and perhaps with certain viral infections, and many have suggested an infectious etiology for Reiter's syndrome); and (8) both diseases are seronegative for rheumatoid arthritis.

These similarities are certainly of interest, but it must be mentioned that more

often than not the two diseases are quite distinct, even given cutaneous manifestations. Psoriatic arthritis is not associated with urethritis. The roentgenographic joint lesions in psoriasis usually do not resolve, and joint destruction and deformity are frequent. Arthritis mutilans may evolve. Reiter's syndrome, however, always requires the presence of urethritis for diagnosis, and joint destruction and deformity are much less common. Arthritis mutilans does not occur in Reiter's syndrome.

SUMMARY

Although Reiter's syndrome and psoriatic arthritis are ordinarily two rather discrete entities, there is a significant zone of diagnostic overlap in patients with cutaneous lesions. Both diseases may coexist in the same patient or Reiter's syndrome seemingly may evolve into psoriatic arthritis.

The separate roentgenographic spectra of both diseases are set forth and the causes for diagnostic confusion of these two diseases are discussed.

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# BONE LESIONS IN PRIMARY AMYLOIDOSIS*

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AMYLOIDOSIS is a relatively uncommon disease which is manifested by diffuse and focal infiltration of numerous organ systems with a homogeneous, amorphous eosinophilic substance having an affinity for various distinctive staining techniques. Metachromasia and congophilia are basic characteristics.

Classification into primary and secondary amyloidosis is generally accepted so that differentiation of the former is made upon specific staining characteristics, absence of associated chronic disease, and predilection for specific sites such as mesenchymal tissue.[3] Common areas of involvement include muscle, vessels, heart, lungs, skin and the tongue. Although infiltration of the liver and spleen are usually seen in secondary amyloidosis, numerous cases in the primary form of the disease have been reported.[9]

Amyloidosis is represented by a derangement of protein metabolism. Atypical serum protein patterns are noted, usually including a decrease in albumin and an increase in alpha$_2$ globulin as well as alteration of various glycoproteins.[6] The similarity to serum protein patterns in multiple myeloma has long been recognized and some investigators have suggested an intimate relationship between these disease entities.[1]

The amyloid infiltrates and replaces normal tissue causing atrophy of adjacent structures. This process results in widespread involvement with enlargement of diseased organs and subsequent interference with normal physiologic activity. This type of pathogenesis is well exemplified by the large number of amyloid cases with cardiac involvement (50 per cent), with death commonly due to cardiac failure.[2]

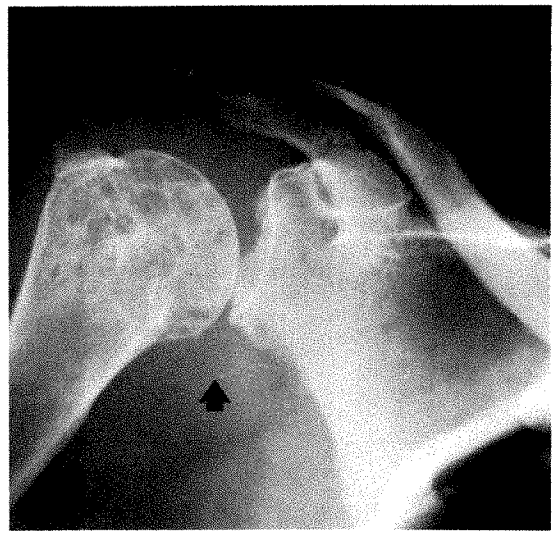


FIG. 1. Left shoulder. Note the increased soft tissue density surrounding the joint (arrows). Lytic changes are also seen in the head of the humerus.

Macroglossia, present in approximately 30 per cent of the cases of primary amyloidosis, also indicates the tendency for diffuse infiltration and organ enlargement.[8]

## REPORT OF A CASE

A 67 year old Negro female entered the hospital complaining of a progressive enlargement of her tongue. She had been hospitalized previously for pyelonephritis and also had a history of arteriosclerotic cardiovascular disease. Physical examination revealed a chronically ill female appearing older than her stated age. Blood pressure was 180/100, pulse 70 /min. A thick tongue deeply furrowed with white discoloration was noted. Auscultation of the heart and lungs disclosed a Grade II/VI systolic murmur and scattered bilateral basilar rales. Percussion revealed cardiomegaly and moderate abdominal distention was observed. The extremities showed fusiform swelling of the elbows and knees.

*Laboratory Findings.* Alkaline phosphatase

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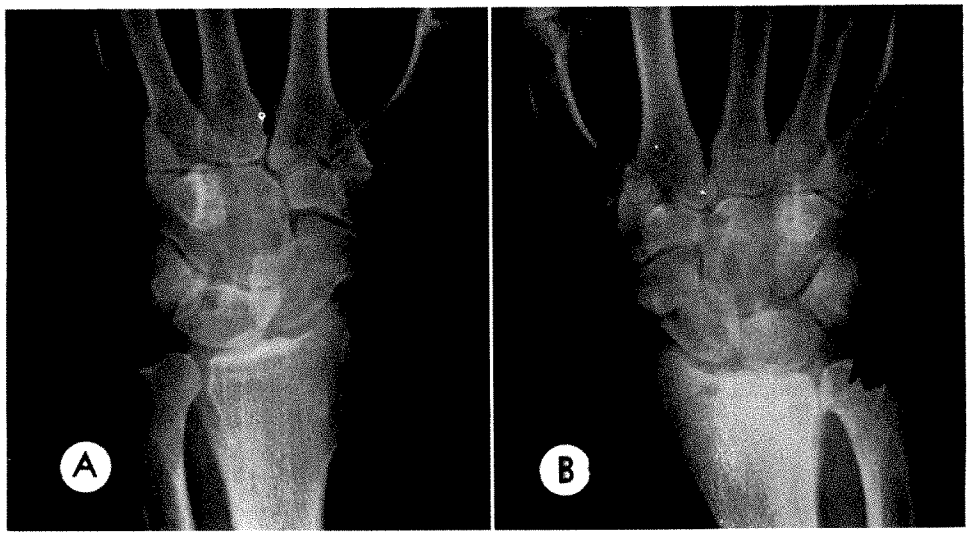



FIG. 2. (*A* and *B*) Increased soft tissue density surrounds the wrist joints representing capsular and pericapsular infiltration w th amyloid (arrow in *B*).

17 Bodansky units, blood urea nitrogen 48 mg. per cent, calcium 9.5 mg. per cent, phosphorus 3.1 mg. per cent, sodium 135 mg. per cent, potassium 5.9 mg. per cent, $CO_2$ 21, chloride 104 mg. per cent and uric acid 7 mg. per cent. Latex agglutination was negative, but Bence Jones proteins were found in the urine. Serum electrophoresis revealed an increase in alpha$_2$ globulin. Bone marrow examination failed to demonstrate myeloma cells. A tongue biopsy showed amyloidosis.

*Roentgenographic Examination.* Cardiomegaly and diffuse pulmonary infiltration were found (Fig. 3). Lytic areas were noted at the proximal humeri and femoral necks. A pathologic fracture was seen through the right humerus (Fig. 7).

*Clinical Course.* The patient's condition remained stable for a month, after which she was re-admitted to the hospital complaining of abdominal pain. Roentgen and clinical examina-

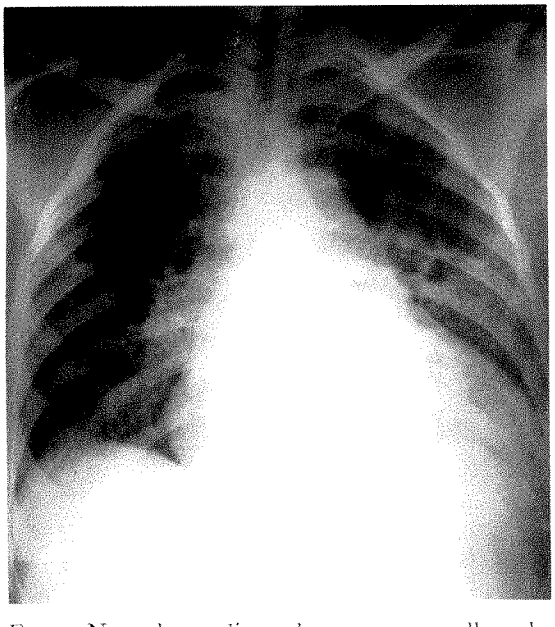


FIG. 3. Note the cardiac enlargement as well as the bilateral infiltrative changes representing severe diffuse myocardial and alveolar infiltration by amyloid.

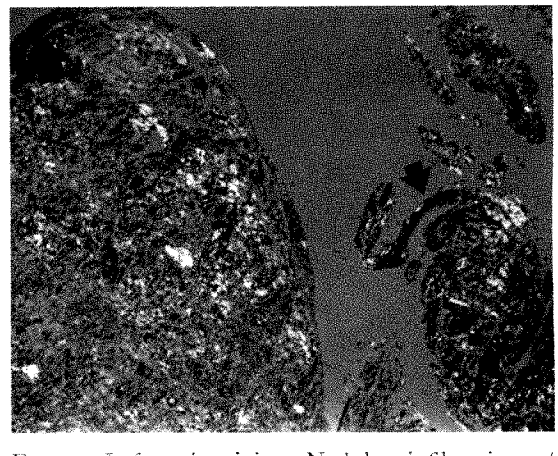


FIG. 4. Left wrist joint. Nodular infiltration of synovial membrane by amyloid. Congo red stain; photographed with polarized light. Birefringent amyloid varies from white to gray in the photograph. Note the strips of normal synovium (arrows). Original magnification ×35.

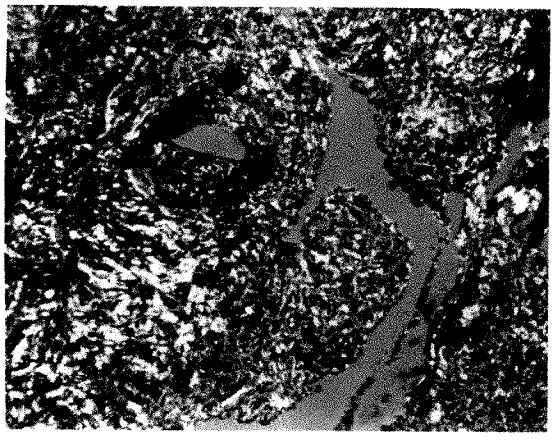


FIG. 5. Periarticular tissues of left hip joint. Congo red stain; photographed with polarized light. Birefringent amyloid varies from white to gray. Note the heavy infiltration of connective tissue and mild infiltration of the artery (arrow). Original magnification ×100.

tion suggested ileus with no evidence of intestinal obstruction.

Conservative management was followed including tube decompression with fluid and electrolyte replacement. Her abdominal symptoms improved; however, atrial fibrillation developed and the venous pressure rose. Cardiac failure progressed and did not respond to treatment. The patient's course continued to deteriorate and several days later she succumbed to severe cardiac failure.

*Postmortem Examination.* Diffuse and focal infiltration of the tongue, myocardium, gingiva, lips, adrenals and alveoli was noted. Diffuse involvement of numerous small vessels was also seen causing occlusion and decreased caliber of the coronary and bronchial arteries. Diffuse infiltration of the small arteries of the mesentery was present. The liver showed rare amyloid deposits within the Kupffer cells and a few amyloid filled follicles were noted in the spleen. Moderate diffuse and focal amyloid was found in the glomeruli of the left kidney. The opposite renal artery was stenotic and showed amyloid infiltration. The thoracic duct was literally occluded with amyloid. The synovial membranes of the wrists, knees and hips were markedly enlarged and displaced and massive amounts of amyloid filled the joint spaces and invaded adjacent connective tissue, muscle and tendons.

## COMMENT

The distribution of amyloid in the above case overlaps the usual classification of amyloidosis in the primary and secondary forms. Nevertheless, it is proper to regard this case as the primary disease because the abnormal protein that infiltrated the tissues varies in its congophilia and metachromasia, and, moreover, the most significant deposits by far were found in the mesenchymal organs such as cardiac muscle, smooth muscle of vessels (Fig. 5) of the

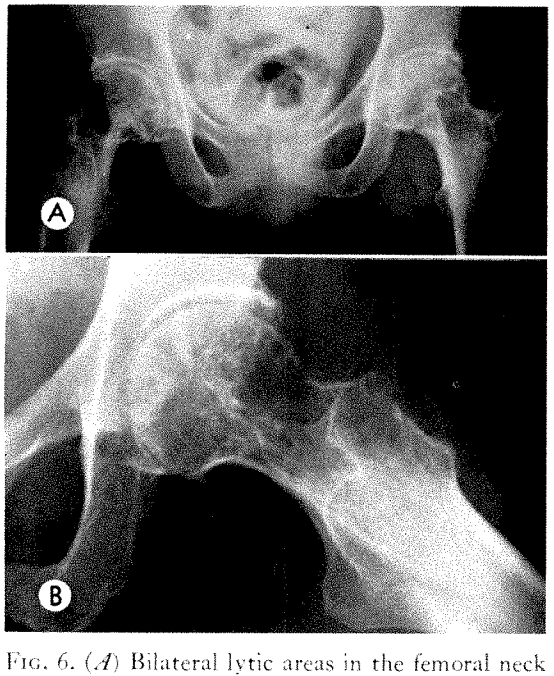



FIG. 6. (*A*) Bilateral lytic areas in the femoral neck and head are noted. Capsular and pericapsular thickening is also seen. (*B*) Left hip showing lytic changes in the femur.



FIG. 7. Right shoulder. A pathologic fracture is seen through the proximal right humerus.

gastrointestinal tract and the striated muscle of the tongue and extremities. The variable staining reactions of primary amyloidosis have been well described.[7]

The amyloid infiltration of the synovial membranes and periarticular fascia was a conspicuous feature of this case that during life led to the clinical impression that the patient manifested rheumatoid disease with secondary amyloidosis. Conversely, classical roentgen findings associated with soft tissue joint enlargement of rheumatoid disease were absent; this was confirmed by gross and microscopic examination of the joints post mortem.

## ROENTGEN FINDINGS

Primary systemic amyloidosis can manifest itself in numerous ways on roentgen examination. The diffuse joint enlargement is shown as an increase in the soft tissue density surrounding the involved joints.

Soft tissue density representing pericapsular and capsular infiltration was noted in the shoulder and wrist area (Fig. 1; and 2, *A* and *B*). Displacement of the synovial membrane with extensive deposition of amyloid in the adjacent joint space is demonstrated on histologic section taken from the left wrist joint (Fig. 4).

Examples of bone lesions in these cases are exceedingly few. The above patient showed lytic areas, representing massive infiltration by amyloid, in the proximal humeri and the femoral necks (Fig. 6, *A* and *B*). These findings have a striking similarity to a case reported by Gardner,[4] which also showed bone involvement with predilection for these sites.

Koletsky and Stecher[5] reported a case showing bone lesions having a similar predilection in location and also showing involvement of the olecranon and coronoid process.

## SUMMARY AND CONCLUSIONS

Reports of primary systemic amyloidosis affecting bone are rare. Those that have been recognized showed a striking predilection for involvement of the proximal humerus and the femoral neck. The roentgen changes consisted of destructive lesions caused by replacement of bone by massive deposits of amyloid. Increased soft tissue density representing capsular and pericapsular infiltration was uniformly noted at major joints.

A review of primary amyloidosis is presented including discussion of the pathogenesis. A case is reported which represents one of the rare examples of bone lesions in the primary systemic disease. Clinical and postmortem details are given.

Although the diagnosis of primary systemic amyloidosis is usually made before striking bone findings are seen on roentgen examination, the lytic osseous changes represent an interesting addition to the large group of differential or skeletal changes in systemic diseases.

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# SKELETAL DEVELOPMENT IN GONADAL DYSGENESIS, FEMALE IN PHENOTYPE*

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THE term "gonadal dysgenesis" in a subject with female phenotype denotes a congenital gonadal anomaly which may be of a variable degree because the gonads can be either absent or rudimental. In the absence of the gonads and consequently of sex differentiation, the ducts and external genitalia develop in a feminine direction, whatever the gonosomic pattern of the subject.

On the basis of clinical, pathologic and cytogenetic characteristics, the gonadal dysgenesis, female in phenotype, can be divided into two fundamental varieties: the "Turner's syndrome" in a wide sense, and the so-called "pure gonadal dysgenesis."[11]

In *Turner's syndrome* we find a short stature (varying between 120–145 cm. and therefore approaching nanism) and a series of malformations involving several organs and systems, among which the skeletal, cutaneous, urinary and ocular are the most affected. Peculiar to the syndrome is the diffuse involvement of the mesenchymal structures. Determination of sex chromatin in accordance with Barr's and Davidson's methods, has revealed the presence of negative chromatin in about 75 per cent of the cases.[11] The study of karyotype has shown that over 95 per cent of the cases present abnormalities of number and/or structure of sex heterochromosomes.

Patients with *pure gonadal dysgenesis* are distinguished from those with Turner's syndrome by a normal or even tall stature (of eunuchoid type), the lowest limit being 153 cm.; and by an absolute lack of the so-called Turnerian malformations, while in the above mentioned form we find a more or less complete sexual infantilism. Determination of sex chromatin has demonstrated the presence of positive chromatin in the majority of cases. The study of karyotype has shown a high incidence of mosaicism, very often comprising cells of XO type. The existing literature indicates mosaicism in 31 per cent of the patients investigated.[11]

## MATERIAL AND METHOD

Among the 71 patients affected by gonadal dysgenesis seen at the University of Florence Endocrinological Center we have had the opportunity to investigate roentgenographically, the skeletons of 50 subjects: 37 affected by *Turner's syndrome*, and 13 by *pure gonadal dysgenesis*. The age of the patients varied from 11.6 to 28.7 years with the majority in the 16–21 age group.

The case material includes 35 patients who had received no previous treatment, 11 patients who had received treatment, and 4 patients presenting the malformation pattern of Turner's syndrome with spontaneous menstruations, cyclic in 1 and scanty and irregular in the other 3.

In 15 patients, 10 of whom were affected with Turner's syndrome and 5 with pure gonadal dysgenesis, we examined the skeletons after substitutive hormonal treatment had been given during a period varying from a minimum of 12 months to a maximum of 6 years. This therapy was based on the cyclic administration of estrogens (10,000 units every 2 days for 3 weeks) and of progesterone (10 mg. every 2 days for 1 week).

We studied the osseous development assessing the maturation degree and consequently the bone age; the latter, in both groups of patients, was compared with stature.

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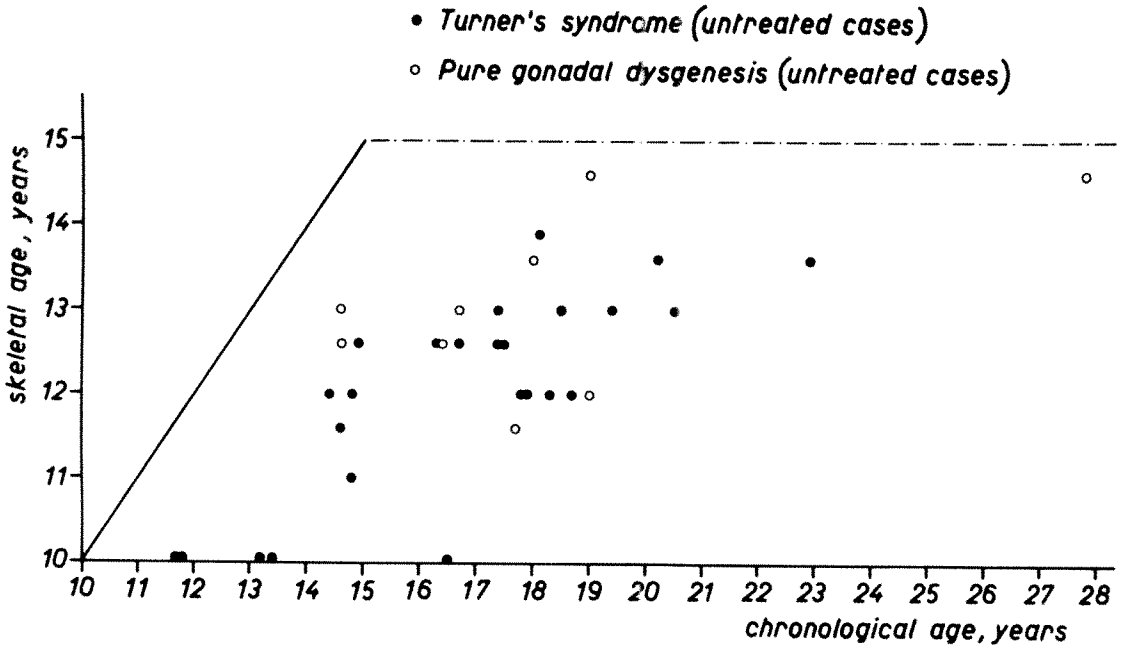



FIG. 1. Untreated patients with Turner's syndrome and with pure gonadal dysgenesis. Evaluation of skeletal age, assessed according to Greulich and Pyle standard, against chronologic age.

## RESULTS AND DISCUSSION

As we have not had the opportunity to collect data on patients under 11 years of age, it is impossible to state whether the ossification centers have appeared regularly or not. Leszczynski and Kosowicz[15] reported the appearance of ossification centers at the expected time in patients of the 8–10 year age group.

In the 50 subjects examined by us, assessment of the wrist and hand bone age was made in those not previously treated: a group of 35 patients aged 11.6–22.10 years. Their roentgenograms were assessed on the basis of the Greulich and Pyle standard.[12] The results are shown in Figure 1. It may be seen that both in patients with Turner's syndrome and in those with pure gonadal dysgenesis, there is a delay in skeletal maturation affecting subjects of any age; this delay, slight for younger patients, increases with age. The skeletal ages studied appeared between 10 and 13.6 years, this being the highest or oldest skeletal age in our series. We can state that the skeletal maturation between these two bone ages occurs during a period of about 10 years. It is reasonable to assume, therefore, that initially there is a slowing down in the bone maturation, followed by an arrest at the age of puberty.[2,15]

Such behavior of skeletal maturation is undoubtedly related to a lack of gonadal steroids due to absence of the gonads which is almost always anatomic but which occasionally can also be functional. A confirmation of the altered maturation due to lack of steroids of gonadal origin, can be found in Figure 2, which shows the skeletal age of patients who underwent substitutive hormonal treatment before or after we made the diagnosis of gonadal dysgenesis. Following therapy all have developed secondary sexual characteristics as well as menstrual periods. This Figure shows that complete maturation of the hands has occurred in all patients.

The skeletal maturation of the foot has been assessed by the Hoerr, Pyle and Francis standard.[14] No difference in the skeletal age has been found between hand and foot.

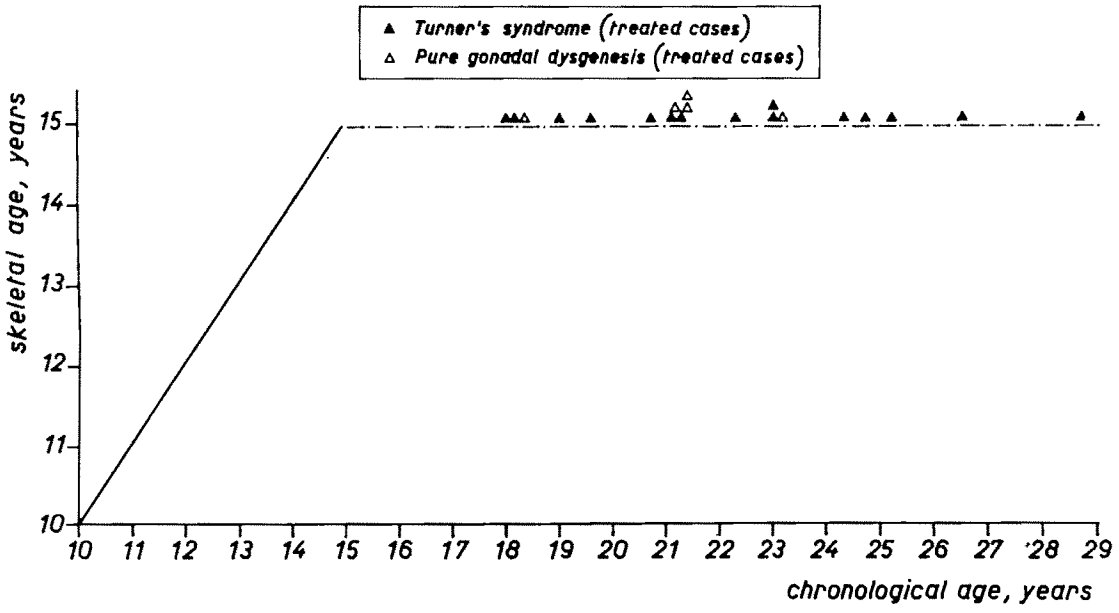



FIG. 2. Patients with Turner's syndrome and pure gonadal dysgenesis, who underwent substitutive hormonal therapy. Evaluation of skeletal age, assessed according to Greulich and Pyle standard, against chronologic age.

Assessment of the skeletal maturation in the hip and pelvic region was then made on the basis of the Oxford method.[1]

Apart from the standard deviation, no significant differences were noted in the skeletal age in 2 anatomic regions (hand-foot, hand-pelvic region) evaluated by means of 2 different methods (Table I).

In most patients having hand skeletal ages between 12 and 13.6 years, when the growth cartilage plate is still present, a process of osseous invasion of the growth cartilage plate has been observed—sometimes in an advanced stage in the bones of the elbow, in the proximal and in the distal femur, in that order of frequency.

The epiphyseal union appearing advanced relative to the hand and pelvic skeletal age, was, however, retarded in relation to the chronologic age of the patient.

The discrepancy in maturation between different anatomic areas can be explained on the basis of accelerated bone growth, which occurs at puberty but does not affect all growth cartilage plates at the same time and to the same extent. According to Hewitt and Acheson,[18] for example, the skeletal maturation of the pelvis is far more responsive to the hormonal changes of puberty than is that of the femur. Thus, it is conceivable that the maturation delay should be more pronounced in those parts of the skeleton whose maturity is subject to a more powerful acceleration at puberty; *i.e.* in the bones of the pelvis, hand and of the proximal tibia.

However, it must be pointed out that discrepancies of 1 or 2 years in the epiphyseal union of segments whose maturation generally proceeds simultaneously, may be related, according to Garn *et al.*[10] to genetic

TABLE I

COMPARISON BETWEEN SKELETAL AGE OF TWO DIFFERENT AREAS

| | | |
|---|---|---|
| Hand and foot | equal or ±6 months | 23 cases |
| | ± 6 months | 8 cases |
| Hand and pelvic region | equal or ±6 months | 18 cases |
| | ±6–12 months | 15 cases |
| | ±12 months | 6 cases |

Hand=Greulich and Pyle standard; foot=Hoerr, Pyle and Francis standards; pelvic region=Oxford method.

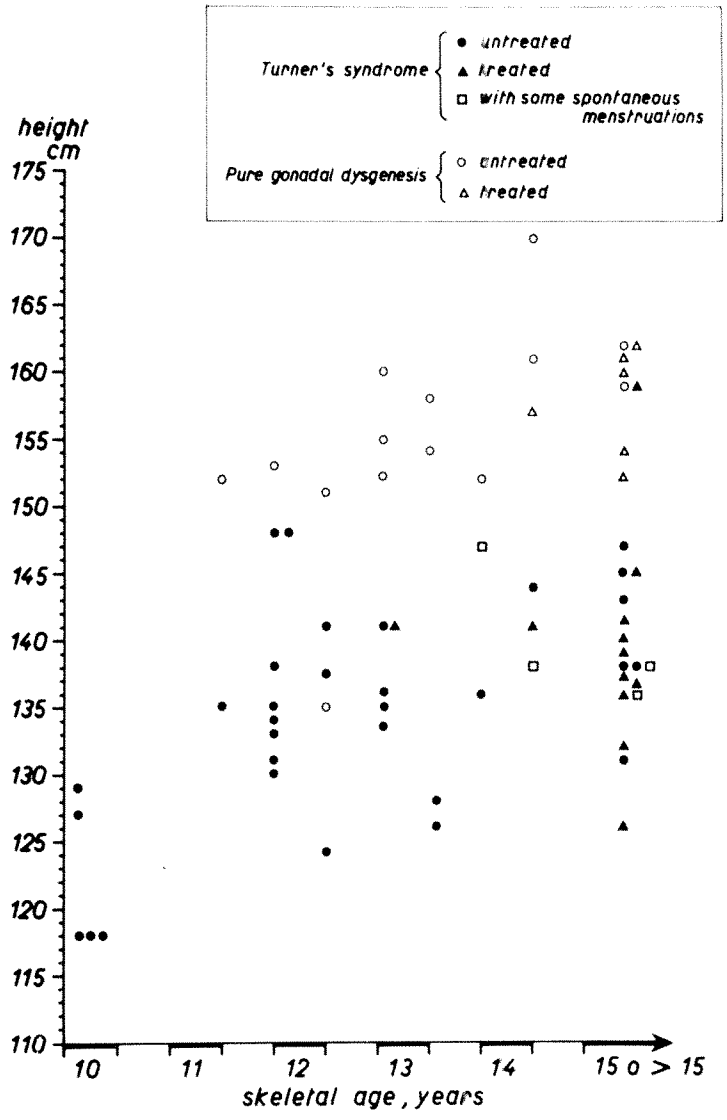



Fig. 3. Evaluation of skeletal age against height in patients with Turner's syndrome and with pure gonadal dysgenesis. The treated cases and the untreated cases are marked by different symbols.

determinants. A genetic factor, therefore, must also be considered in discussing the phenomenon observed in our patients.

The appearance of the epiphysis of the iliac crest seems to be a sufficiently reliable index of sexual maturity. It is well known that the ossification of this epiphysis occurs at puberty, during the year of maximum growth or within the year preceding or following it.[4] In order to assess this index of maturation we have examined the pelvis of 30 patients who never had menstrual periods; of these, 4 presented ossification of the iliac crest. The 26 patients with ossification still absent were all, except two, 13.9 years of skeletal age or under. Thus there can be no doubt that, with few exceptions, no ossification of this epiphysis occurs in prepubertal skeletons. On the other hand, the fact that such epiphysis was present in 12 out of 14 patients having spontaneous or therapeutically induced menstrual periods, proves that its appearance is an index of hormonal modifications peculiar to pu-

berty. Moreover, this correlation is confirmed by the fact that in 10 out of 13 patients not having this epiphysis, we observed its appearance after substitutive hormonal treatment.

To evaluate the stature of our patients, some treated and some untreated, taking into account the influence of therapy, we have correlated height with bone age and therefore with skeletal maturity. Figure 3 shows not only the values obtained when the diagnosis was made but also those found after a certain period of treatment or a period of observation of those cases that had received no treatment.

First of all there is a well defined difference of height at any age between patients affected with Turner's syndrome in a wide sense and those with pure gonadal dysgenesis. While the latter are between 151–170 cm. in height, patients with Turner's syndrome, with 1 exception, are 118–148 cm. tall.

Examining the stature of patients with Turner's syndrome, it appears that in any age group there is a difference of at least 10 cm. between extreme cases. Significant are the values in patients with already completed growth. The difference between minimum and maximum heights, without considering the exception, is 21 cm.; in other words in this group of patients with short stature there is a variability of about 20 cm.—not very different, therefore, from the one found among normal subjects. A lesser variability is found in the heights of patients with pure gonadal dysgenesis, but this group is far less numerous.

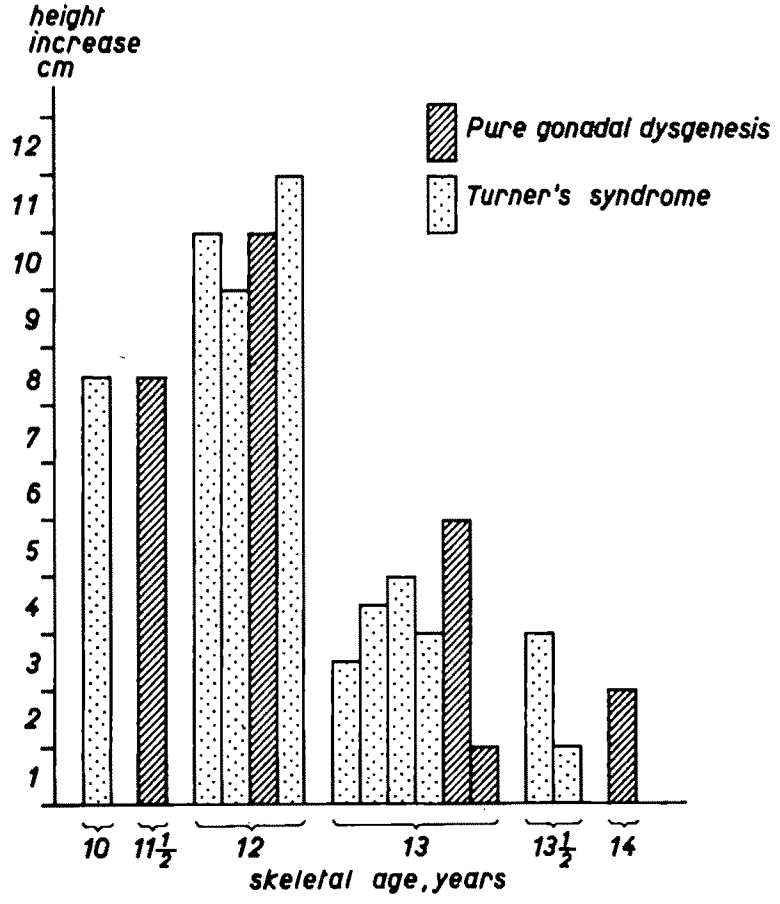



FIG. 4. Evaluation of increase in height against the skeletal age of 15 patients who underwent substitutive hormonal therapy until completion of skeletal maturation.

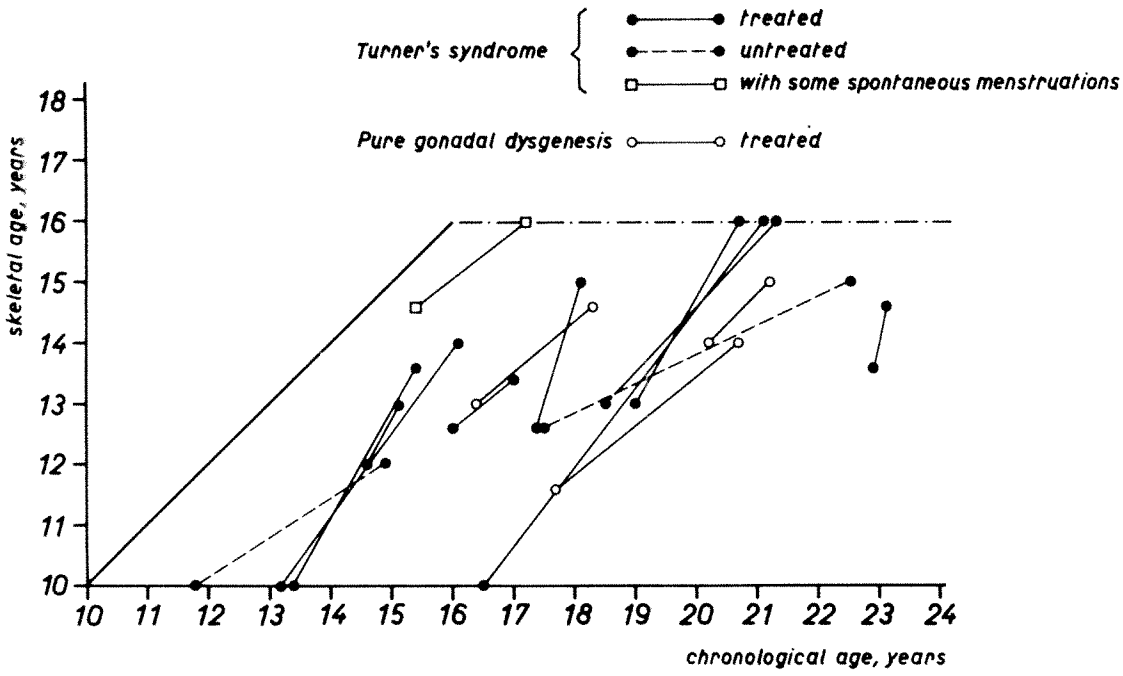



FIG. 5. Variations of skeletal age compared to chronologic age in 15 patients with Turner's syndrome and with pure gonadal dysgenesis. The treated cases and the untreated cases are marked by different symbols.

Comparison of the heights of treated and untreated patients shows a similar distribution.

The distribution of the heights of patients with different skeletal ages exhibits an upward slope which points to an appreciable growth process, both in patients with Turner's syndrome and in those affected by pure gonadal dysgenesis.

The amount of height variation during the period of skeletal growth has been studied in 20 patients given substitutive hormonal therapy; of these, 15 cases were treated at different bone ages until skeletal growth ceased.

The results of assessments after treatment are shown in Figure 4. It clearly appears that greater growth (8–11 cm.) occurs when the bone age is 10–12 years; shortly after that the height increase is generally slight. No difference is noted between patients of the two groups.

In order to assess the maturation process during therapy we have tabulated the variations of skeletal age against those of chronologic age. In Figure 5, where these variations are given, a standard development curve of normal subjects according to Greulich and Pyle[12] may be seen.

At this point mention should be made of the fact that the curves referring to our patients are merely the joining tract of 2 points representing respectively the initial and final assessments, and have therefore no meaning as maturity curves.

Figure 5 also shows the variations found in the only untreated case. While in this case bone maturation proceeded slowly as compared to normal, in patients undergoing treatment, maturation usually proceeds in an analogous or faster way.

Accelerated growth has not affected the increase in height; this increase depended exclusively on the degree of skeletal age before therapy.

Several hypotheses have been put forward to explain the shortness of stature in patients with Turner's syndrome. At present only two of these are considered plausible; *i.e.* that short stature is related (1) either to a defect of tissue receptivity of growth cartilage plate or (2) to

anomalies of the structure and consequently of the activity of the endogenous growth hormone. This latter hypothesis seems to be rather doubtful in the light of recent studies on metabolic and statural variations found in some patients with Turner's syndrome treated with physiologic doses of human growth hormone (HGH).[8]

The first hypothesis is based on the fact that in the skeleton of patients with Turner's syndrome there are alterations of both the growth cartilages, in which characteristic lesions of the condrodystrophy type have been found,[5,17] and of the bone structure, whose trabeculae appear coarse, thick and normally calcified.[7,15]

In our opinion also the hypothesis of the altered tissue receptivity is debatable. The roentgenologic findings in our cases have shown that the bone deformities,[6,16] resembling a chondrodystrophic condition, even if occuring very frequently, do not affect a certain number of patients. Furthermore, we have found no proportionality between the degree of the bone deformity and the short stature. Similar remarks can be made concerning the bone structure; the latter actually has appeared altered in fewer cases.

An altered tissue receptivity of growth cartilage plate is to be regarded as doubtful also as regards bone growth. If height variations are analyzed in relation to age, one can note that, based on the data of Zurli *et al.*,[18] there is an absolute difference in height between normal subjects and patients with Turner's syndrome, but there is no difference in the range of growth between the two groups during the period of growing. On the other hand, the growth cartilages of these patients have appeared responsive not only to the administration of androgens and growth stimulating hormone[3,9] but also to estrogen[8] treatment, as proved by our data. It is worth mentioning that there have been no differences in height variations after hormone therapy between patients with Turner's syndrome and pure gonadal dysgenesis.

We have therefore good reason to believe that short stature in patients with Turner's syndrome is determined by a genetic factor; in short, this clinical finding would be, as other malformations are, related to a genetic abnormality. Obviously environmental and, above all, hormonal factors combine to aggravate the clinical picture. The lack of gonadal steroids accounts for a stature inferior to the definitive one as long as the state of prepubertal immaturity persists. The administration of estrogens enhances the maturation process and is responsible for the spurt in height typical of puberty.

Therefore, we can conclude that if skeletal maturation in the two varieties of gonadal dysgenesis is analogous as a consequence of the fact that the gonadal situation is also analogous, the height, instead, is different. The short stature of the subjects with Turner's syndrome, never observed in those with pure gonadal dysgenesis, must be regarded as malformative in origin and related to a genetic aberration, as are all the other malformations of these patients.

## SUMMARY

1. The skeletal development has been investigated roentgenographically in 50 patients affected by gonadal dysgenesis, of whom 37 had *Turner's syndrome* and 13 had *pure gonadal dysgenesis*. In 15 patients the skeletal development has been controlled after substitutive hormonal therapy.

2. Both in patients with Turner's syndrome and those with pure gonadal dysgenesis there is a delay in skeletal maturation affecting subjects of any age; the skeletal ages comprised those between 10 and 13.6 years. This behavior is referred to as a lack of gonadal steroids.

3. All the patients who underwent substitutive hormonal treatment showed maturation of the examined bones.

4. Discrepancy in maturation delay between different anatomic areas has been found.

5. The appearance of the epiphysis of the iliac crest seems to be a sufficiently reliable index of sexual maturity. In 26 out of 30 patients, whose iliac crest has been ex-

amined, this epiphysis was absent and it appeared in most of the cases in whom menstruation was induced therapeutically.

6. The skeletal growth was altered differently in patients with pure gonadal dysgenesis and those with Turner's syndrome; only the latter had a short stature which is regarded as malformative in origin.

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# LIPOATROPHIC DIABETES MELLITUS (GENERALIZED LIPODYSTROPHY): ROENTGEN FINDINGS IN TWO BROTHERS WITH CONGENITAL DISEASE*

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ALTHOUGH the clinical and metabolic aspects of congenital lipoatrophic diabetes have been well documented[3,15-18,21,23] the roentgenographic changes have not been reviewed in detail. This lack is lamentable, as the striking bone and soft tissue abnormalities seen on roentgenograms are characteristic.

The syndrome of lipoatrophic diabetes, also known as generalized or total lipodystrophy, when fully developed consists of several abnormalities. These include almost complete absence of adipose tissue, accentuated muscularity, hepatomegaly with cirrhosis, insulin-resistant diabetes mellitus not associated with ketosis, hyperlipemia, and an elevated basal metabolic rate without hyperthyroidism. The first case report appeared in 1928[28] but not until 1946, when Lawrence[10] reported a patient with adult onset of the disease, were the clinical findings described clearly. Lawrence suggested the designation, "lipoatrophic diabetes."

Seip[20] emphasized that when the syndrome is congenital, as it often is, additional abnormalities may be noted in the affected infants and children. Rapid growth, advanced bone age, hypertrichosis, hyperpigmentation, and enlarged genitalia are some of these variations. Hyperglycemia, however, is not usually present in the early years of the congenital disease, but develops later.

Lipoatrophic diabetes or generalized lipodystrophy, characterized by the absence of adipose tissue throughout the body, should not be confused with partial or progressive lipodystrophy, a far more common entity. The latter is often seen in women and usually involves only the upper half of the body.

Of the 35 reported cases of lipoatrophic diabetes or generalized lipodystrophy, 22 were congenital or probably congenital and 3, initially recognized before 2 years of age, were possibly congenital. These last mentioned 25 patients consisted of 16 females and 9 males. In 6 of the families reported, 2 siblings were known to have had the disorder.[2,5,8,15,20,26] Parental consanguinity was reported in 5 instances, producing a total of 7 affected children.[1,2,12,20] Of the 10 patients with acquired lipoatrophic diabetes, the disease became evident after the age of 20 years in only 4.[10,11,27,28]

The most striking clinical change reported in both congenital and acquired forms was an almost complete absence of adipose tissue. The effect on the subcutaneous tissue was a firmness of the skin which made it impossible to "tent" when pinched. Other tissues normally containing fat, such as the perirenal, properitoneal, mesenteric, and epicardial, were similarly affected. Hirsuties, especially in the region of the scalp, marked by a low-set anterior hairline, has been consistently observed in the congenital form. Generalized pigmentation was another frequent characteristic and localized acanthosis nigricans was present in 7 patients.[2,15,18,26] Phlebomegaly of the superficial veins has been described in 8 patients in whom the disease was congenital.[2,3,15,17,18,26]

A true hypertrophy of the striated muscles has been reported in almost every patient with congenital disease and, occasionally, in those with acquired disease. No muscle weakness was associated with the

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disease; to the contrary, muscle strength was sometimes described as greater than normal. An increase in glycogen content was demonstrated in at least 3 patients.[20] In most of the patients with the acquired disease, muscularity was less prominent and in 1 patient, a 15 year old girl, the muscles were atrophic.[7]

Senior and Gellis[23] noted that affected children were tall, tending to the ninetieth percentile. Concomitant increase of bone maturation, however, suggested curtailment of the final height. Nearly all of the patients in the congenital group attained more than 90 per cent of their growth in the first 10 years of life. After this age, growth decelerated rapidly, so that the final stature was average or even below average.[15] In adults, an increase in height has not been observed.

The abdomens of all of the children were markedly protuberant with hepatomegaly present in every instance. Histologic examination of the liver usually revealed a combination of fatty infiltration, glycogen deposition, and periportal fibrosis. The spleen was sometimes correspondingly enlarged, arising secondary to portal hypertension. Rupture of esophageal varices led to the death of several patients.[1,4,6,8]

Enlargement of the penis or clitoris, without signs of virilization, was the rule in congenital disease. In neither sex has an increased secretion of gonadotropins or adrenal androgens been demonstrated.

Hyperglycemia, although not present in infancy and early childhood, appeared to increase in incidence with age, so that eventually it could be expected in all patients. It was present in all with the acquired disease. Despite its poor response to insulin administration, hyperglycemia was never accompanied with ketosis, and only rarely with the usual clinical complications of diabetes.[7,11]

Hyperlipemia, especially of the triglyceride fraction, was a universal finding. It usually preceded the onset of hyperglycemia.

An elevated basal metabolic rate, with otherwise normal thyroid function, and without accompanying signs or symptoms of hyperthyroidism, was present in all patients tested.

In two-thirds of the children, some abnormality of the central nervous system was manifested, pneumoencephalography demonstrating ventricular dilatation in 6,[18,20,21] and mentality being retarded in another 6.[2,15,22,26]

Renal abnormalities were found in 12 of the 35 patients, 8 of them being from the congenital group. In these, an unexplained enlargement of the kidneys was the usual finding. The remaining 4 patients were from the acquired group, in 2 of whom histologic examination revealed typical Kimmelstiel-Wilson disease.[7,11]

Cardiac enlargement was present in 14 patients, occasionally associated with a soft systolic murmur. In 2 patients, cardiac catheterization demonstrated a moderately increased resistance in the pulmonary artery and, in 1, an atrial septal defect.[21] In most patients, the cause of the enlargement was obscure.

A study of the bone marrow aspirated from a 16 year old girl with probable congenital disease [18] revealed a normal cellular component, but absence of all marrow fat.

The pathophysiology of lipoatrophic diabetes is not well understood, but several hypotheses have been offered. (a) A primary absence of adipose tissue is believed by most to be untenable. A small amount of fat may be found in the subcutaneous tissue of even the congenital cases, such as in our own. (b) A pituitary somatotropic hyperfunction has been theorized,[20] but has never been demonstrated. (c) Hamwi *et al.*[7] recovered a potent lipid-mobilizing substance from the urine of a patient whose onset of the disease was in adulthood. These authors proposed that this substance promotes rapid hydrolysis of triglycerides to fatty acids and glycerol, thus inhibiting the accumulation of triglycerides in adipose tissue. The excessive production of fatty acids results in their increased conversion in the liver to triglycerides and other lipids.

Randle *et al.*[14] postulated that conditions resulting in excessive fatty acid mobilization lead to insulin resistance and interference with glucose utilization by muscle. This postulation may be conveniently combined with that of Hamwi *et al.*[7]

Seip[20] suggested that a simple recessive inheritance was responsible for the congenital form of the disease. A homozygous recessive autosomal transmission would explain the absence of the syndrome in the parents, and its presence in siblings of both sexes.

Roentgenograms were reproduced in only 9 of the 35 previously reported cases. Of these, 6 were studies of a single extremity,[1,3,12,21] and 3 were pneumoencephalographic studies.[20] Only 17 of the reports contained sketchy descriptions of the roentgenographic results. Skeletal maturation was studied in 17 patients from the congenital group and in only 1 was bone age not increased.[23] Aside from the absence of subcutaneous fat, the next most common roentgenographic finding was a generalized increase in bone density in 8 patients. Seven of these had had lipoatrophic diabetes since birth. Seip and Trygstad[21] found the bones to be sclerotic in all 5 of their patients, of 1 to 6 years of age. Ruvalcaba *et al.*[17] described increased cortical thickness in the long bones of a 15 month old girl. Choremis *et al.*[3] noted sclerosis of the entire skeleton in a 10 month old girl. In a 34 year old man who had had lipoatrophic diabetes for 12 years, Marcus[11] observed markedly thickened osseous cortices. Berardinelli,[1] in a legend of the roentgenogram of the knee of a 6 year old boy, commented on the advanced bone age (9 years) and the enlarged epiphyses. None of the other reports referred to enlarged or abnormally shaped epiphyses. A congenital anomaly of a radius was present in an 11 year old boy.[6]

Descriptions of roentgenograms of the skull were available in 8 reports. All were considered to be within normal limits[1,2,7,17,22,25] except 1, in which bone density was increased.[3] No unusual calcifications were described.

Vertebral abnormalities were described in only 2 patients. One was a spina bifida of the first 3 sacral segments in a 6 year old boy.[1] The other was a sclerotic vertebral column in a 10 month old girl with a generalized increase in skeletal density.[3]

Chest roentgenograms in 5 patients were reported as showing diffuse cardiomegaly.[2,3,18,21] Enlarged kidneys were noted in the roentgenographic reports of 2 patients.[2,18]

Two brothers whose cutaneous abnormality had been reported previously[14] were studied roentgenographically. All of the clinical features described in the other congenital cases were demonstrated in these two patients.

## REPORT OF CASES

CASE 1. This 19 year old mentally retarded Negro was admitted to the San Francisco Medical Center of the University of California for evaluation of fat metabolism. The patient was the product of a normal gestation and weighed 1.8 kg. at birth. At that time examination revealed a marked diminution of subcutaneous tissue. The boy was the third of 7 children. A younger brother (Case 11) also had lipoatrophic diabetes. No information was available regarding the father or the possible role of consanguinity. The patient gained weight slowly and motor development was retarded. Dentition did not appear until 8 months of age. At the age of 3 years, a surgical exploration was performed for a "possible adrenal tumor" but the results are not known. The patient was thoroughly evaluated for the first time at the age of 6 years. Height at that time (125 cm.) exceeded the average for his age and the external genitalia were described as remarkably enlarged. Patches of acanthosis nigricans were present. The head was increased in the biparietal diameter, there was frontal bossing, and the lower jaw protruded. The joints of the upper and lower extremities were enlarged and deformed. A large liver was palpated across the entire upper abdomen; a biopsy revealed Laennec's cirrhosis and fatty metamorphosis. Roentgenograms at the age of 6 years showed increased density of bones with bone age equal to that of a 12 year old boy. An intravenous urogram showed that both kidneys contained blunted and dilated calyces and were

uniformly enlarged. A glucose tolerance test yielded the following values: a fasting blood glucose level of 101 mg. per 100 ml. with a 2+ glycosuria; a 2 hour blood glucose level of 188 mg. per 100 ml. with 3+glycosuria, and a 6 hour glucose level of 76 mg. per 100 ml. with a 2+glycosuria.

At the age of 12 years, a single psychomotor seizure was experienced. The boy continued to grow in stature, his general physical appearance remaining unchanged. Initially his diabetes was controlled with insulin and later by diet alone, supplemented at times with Orinase.

*Physical examination.* Height was 163 cm., weight 54 kg., blood pressure 160/80 mm. Hg, pulse 90 with sinus rhythm. The patient was a thin, muscular Negro with broad shoulders. The peculiar shape of the head, described previously, sunken cheeks, furrowed forehead and scalp, and large ears, resulted in a striking facial appearance. The anterior hairline was low. It was impossible to "tent" the firm skin. Acanthosis nigricans was noted in the interscapular region of the back and over both Achilles tendons. On examination of the mouth, a large furrowed tongue and protruding lower teeth were seen. The chest was normal. The abdomen was protuberant and the soft edge of an enlarged liver was palpated 3 fingerbreadths below the right costal margin. The penis was remarkably large. The neurologic examination revealed moderate mental retardation, but results otherwise were within normal limits. Musculoskeletal examination disclosed a thoracic lordosis. The fingers and toes were noticeably long.

*Pertinent laboratory data.* The hemogram was normal. Urinalysis showed 2+glycosuria and no ketonuria or proteinuria. The following studies were within normal limits: serum electrolytes (including calcium and phosphorus), alkaline phosphatase, creatinine, lactic dehydrogenase, serum glutamic pyruvic transaminase, serum glutamic oxaloacetic transaminase, uric acid, total protein, blood urea nitrogen, total bilirubin, cephalin flocculation, bromsulphalein retention, protein electrophoresis, butanol extractable iodine, prothrombin time, urinary creatinine clearance, and sickle-cell preparation. The fasting blood sugar level ranged from 105 to 130 mg. per 100 ml. An intravenous glucose tolerance test revealed marked impairment of carbohydrate tolerance. Total cholesterol was 310 mg. per 100 ml. and free cholesterol was 91 mg. per 100 ml. The serum triglyceride was elevated at 436 mg. per 100 ml. and phospholipids were elevated at 284 mg. per 100 ml. The 24 hour urinary excretion of amino acids and creatine was markedly elevated. The 24 hour urinary 17 ketosteroid excretion was reduced (2.0 mg.), but the 17 hydroxycorticoid and testosterone excretions were within normal limits. Special studies were made of the patient's fat metabolism; these will be reported by other investigators at a later date.

*Other pertinent studies.* An electrocardiogram showed nothing abnormal. A biopsy of a specimen of skin, stained with oil red and sudan black, demonstrated marked reduction of subcutaneous fat, although some could still be detected. Attempted needle aspiration of the iliac crest was unsuccessful, moderately increased resistance of the bone to the needle being encountered.

Case II. (Fig. 1). This 11 year old mentally retarded Negro, the brother of Case 1, was admitted to the San Francisco Medical Center of the University of California for evaluation of fat metabolism. He was the product of a normal pregnancy, weighing 3.6 kg. at birth, at which time no adipose tissue was clinically apparent. Motor development was slow. At the age of $2\frac{1}{2}$ years, the patient was muscular and tall (91.4 cm.). The liver was palpable 1 fingerbreadth below the right costal margin. Generalized acanthosis nigricans was noted. At the age of $5\frac{1}{2}$ years, the bone age was that of a 10 year old boy. The fasting blood sugar at the age of $6\frac{1}{2}$ years was 142 mg. per 100 ml. with a 3+glycosuria. At 7 years of age the height was that of an 11 year old boy. A glucose tolerance test at that time revealed a fasting level of 66 mg. per 100 ml; at 30 minutes, 1 hour, $1\frac{1}{2}$ hours, 2 hours, and 3 hours, it was 96, 148, 116, 115, and 105 mg. per 100 ml., respectively. Although the liver had increased in size, studies disclosed that liver function was within normal limits.

*Physical examination.* The patient was a tall, muscular Negro boy. The examination revealed a weight of 52 kg. and a height of 167 cm. The facial features were similar to those of the older brother: a prominent wrinkled forehead, abundant black hair, low-set ears, deep-set eyes, sunken cheeks, and thickened tongue. The lower jaw protruded. The skin was firm and coarse with patches of acanthosis nigricans on



FIG. 1. Case II. The 11 year old brother. The cheeks are sunken, the abdomen is protuberant, and the muscles are prominent. The firm skin is markedly creased in areas of flexion from absence of cushioning subcutaneous fat. Phlebomegaly is present in the antecubital fossae.

the back of the neck, axillae, groin, and antecubital fossae. The chest was normal. The abdomen was protuberant with the liver palpated 4 fingerbreadths below the right costal margin. The penis was enlarged.

*Pertinent laboratory data.* The hemogram and results of urinalysis were normal. Glycosuria was not present nor was ketonuria noted at any time. An oral glucose tolerance test was within normal limits, but an intravenous glucose tolerance test was moderately abnormal. The following studies were within normal limits: fasting blood glucose level, triglycerides, phospholipids, total cholesterol, free cholesterol, total protein, protein electrophoresis, bromsulphalein retention, serum creatinine, and urinary creatinine clearance. The serum glutamic oxaloacetic transaminase was elevated (56 units), but the serum glutamic pyruvic transaminase was normal. Alkaline phosphatase was elevated at 15 Shinowara-Jones-Reinhart units. A 24 hour urinary 17 hydroxycorticoid excretion was normal, but the 17 ketosteroid excretion was reduced at 3.9 mg. per 24 hours. The 24 hour urinary creatine excretion was elevated at 512 mg. Bone marrow contained normal cellular elements, but no marrow fat. Special studies related to fat metabolism were undertaken. They will be reported by other investigators at a later date.

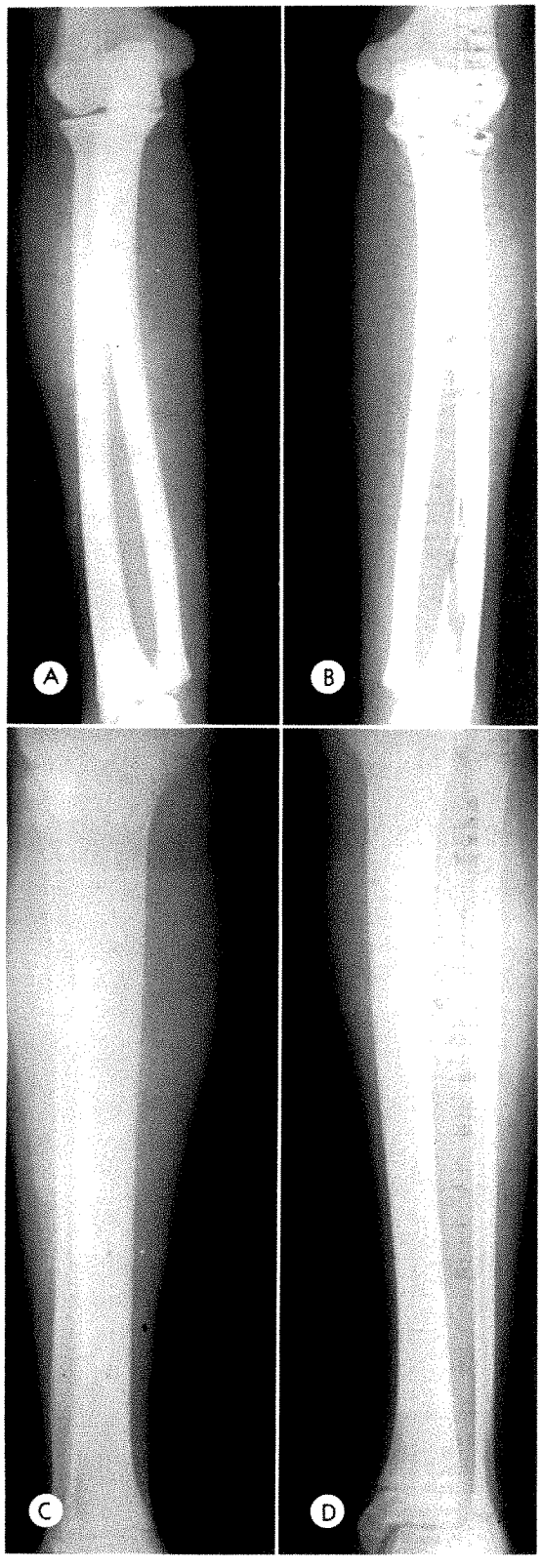
## ROENTGEN STUDIES

Roentgenographic studies of both patients demonstrated the following abnormalities.

*Soft tissue of the extremities* (Fig. 2, *A–D*). No fat was visible in the subcutaneous tissue. The muscles were well developed. The absence of fat between muscle bundles produced a homogeneous soft tissue density, except in the thighs of the older brother where faint traces of fat could be seen between the muscle bundles.

*Skeletal maturation.* The bone age in the younger, 11 year old brother, was comparable to that of 13 years. Skeletal maturation in the 19 year old brother was normal at that age, although at 6 years of age it had been reported as equal to that of 12 years. This pattern is consistent with that reported by others.

*Tubular bones.* Of the entire skeleton, the long bones appeared to have the greatest increase in density, and yet were quite slender. Thickened cortex extended into the shaft to a greater extent than normal, replacing part of the marrow that would have been present ordinarily (Fig. 3, *A–D*). In the older brother, a homogeneous density was seen in each of the femoral necks (Fig. 4*A*). These densities were without sclerotic borders and did not, therefore, present the usual appearance of infarcts (the sickle-cell preparation had been normal). The densities more resembled regions




of overabundant and too closely packed trabeculae, perhaps representing local myelofibrosis related to atrophy of bone-marrow fat. Such densities were not seen in the long bones of the younger brother. In this boy, the epiphyses were unusual. The length of the unfused epiphyses of the femoral head was increased, especially in relation to breadth (Fig. 4*B*). A similar change was seen in the humeral head and metacarpal and metatarsal epiphyses, but to a lesser degree. In the younger brother, the epiphysis of the distal phalanx of the great toe was fused only in its most lateral portion (Fig. 5). This might be expected to cause deformity later, although no deformity was present in the great toe of the older brother. The epiphyses in the older brother were closed; their earlier appearance is not known. Mild coxa valga deformities were present in both brothers.

*Pelvic bones.* Generalized increased bony density was present in both boys.

*Spine.* In the vertebral bodies of the younger brother, just within the superior and inferior borders, were transverse bands of increased density (Fig. 6, *A* and *B*). Especially prominent in the lower thoracic and lumbar regions, these bands were unlike anything we had seen before and did not resemble the findings in any other abnormalities producing dense bones. The vertebrae in the older brother were diffusely increased in density.

*Skull* (Fig. 7, *A–E*). Many similar abnormalities were seen in both brothers. Brachycephaly, present in both, was particularly marked in the younger. Calcification was present in the falx cerebri. The calvarium was thickened and dense

FIG. 2. (*A* and *B*) Case I. Forearms of the 19 year old brother. (*C* and *D*) Case II. Legs of the 11 year old brother. Fat is absent in the subcutaneous tissue and between muscle bundles. The muscles are prominent.

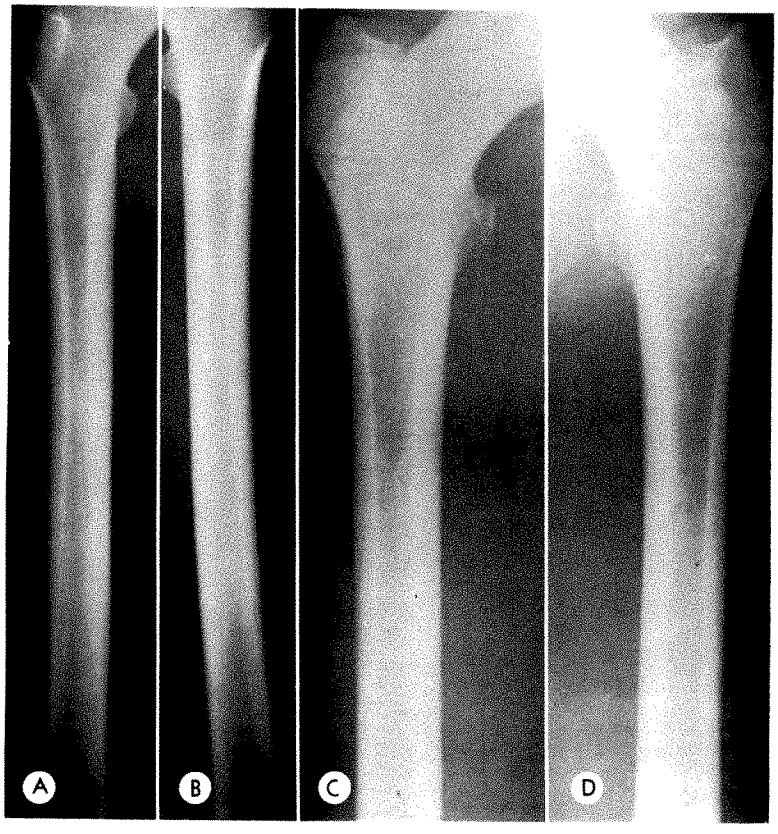



FIG. 3. (*A* and *B*) Case I. Femora of the 19 year old brother. (*C* and *D*) Case II. Femora of the 11 year old brother. Despite the cortical endosteal thickening, the bones remain quite slender.

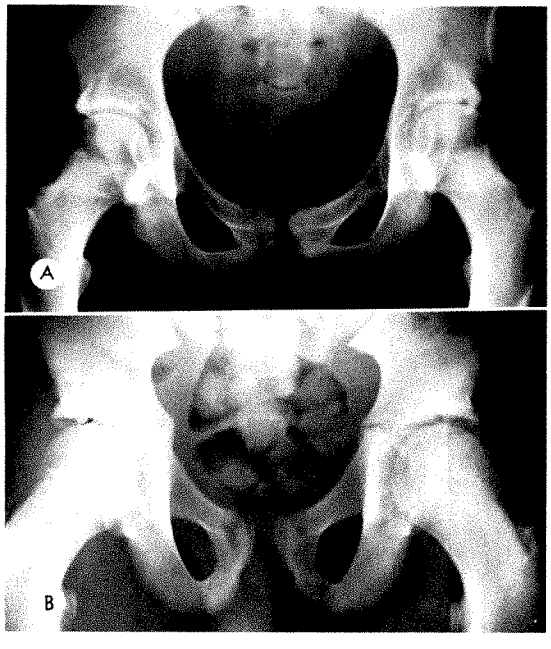



FIG. 4. (*A*) Case I. Hips of the 19 year old brother. A homogeneous density is seen in each femoral neck.

with diminished diploë. Numerous radiolucencies in the frontal bones represented meningeal vessels and pacchionian granulations. The intracranial volume was diminished. An exaggerated sloping of the frontal bones contributed to a decrease in the size of the anterior fossa. The sella was excessively inclined and deep, its anterior portion being almost vertical. The anterior clinoid processes and tuberculum sellae were unusually high in position relative to that

(*B*) Case II. Hips of the 11 year old brother. The length of the capital femoral epiphyses is increased in relation to breadth. Mild coxa valga is present in each patient.

of the posterior clinoid processes. The sella was not enlarged.

In the older brother the ethmoid sinuses were markedly enlarged and pneumatization extended into the supraorbital plates. The anterior clinoid processes were elongated, the left being pneumatized with sphenoid air-cells and the right being very dense. Two middle clinoid processes were present. The excessive pneumatization may have been related to cerebral atrophy. In neither patient was pneumoencephalography performed.



FIG. 5. Case II. Foot of the 11 year old brother. The epiphyses of the second through fifth metatarsals are elongated. The epiphysis of the distal phalanx of the great toe is fused laterally.

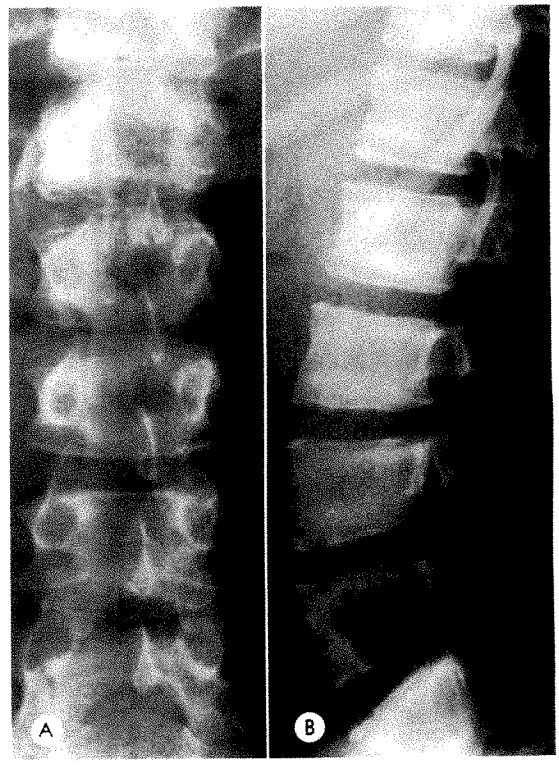



FIG. 6 (*A* and *B*) Case II. Lumbar spine of the 11 year old brother. Transverse bands of increased density are present within the superior and inferior borders of the vertebral bodies.

*Mandible.* A dense and long mandible with large neural canals was present in both brothers. The older brother had lost many teeth; those that remained contained numerous fillings.

*Scapula and clavicle.* Nothing remarkable was seen in these bones.

*Carpal and tarsal bones.* These bones were diffusely increased in density with closely packed trabeculae.

*Chest* (Fig. 8, *A* and *B*). Mild cardiomegaly of no particular diagnostic configuration was present in the 2 brothers. Splenomegaly in each caused a medial displacement of the stomach bubble. The ribs were slightly increased in density.

*Abdomen.* Intravenous urography was performed on the older brother. Spleno-

megaly was apparent but hepatomegaly less so. The properitoneal fat and that normally ensheathing and resulting in recognition of the psoas muscle bundles could not be demonstrated (Fig. 9). Renal calyces and infundibula were splayed bilaterally, suggesting cystic disease, although diffuse enlargement could produce a similar appearance (Fig. 10). Mild calyceal blunting was present on the right. Because perirenal fat was absent, kidney size could not be determined, although the calyceal pattern suggested bilateral enlargement. The ureters were of normal caliber.

SUMMARY OF ROENTGEN FINDINGS

The absence of fat in the soft-tissues of the extremities led to a striking homogeneous soft tissue density. Skeletal maturation was advanced. All the bones were increased in density. The greatest increase appeared to be in those in which hematopoietic marrow is replaced by fatty marrow in adult life. The best example of this increased density was in the long tubular

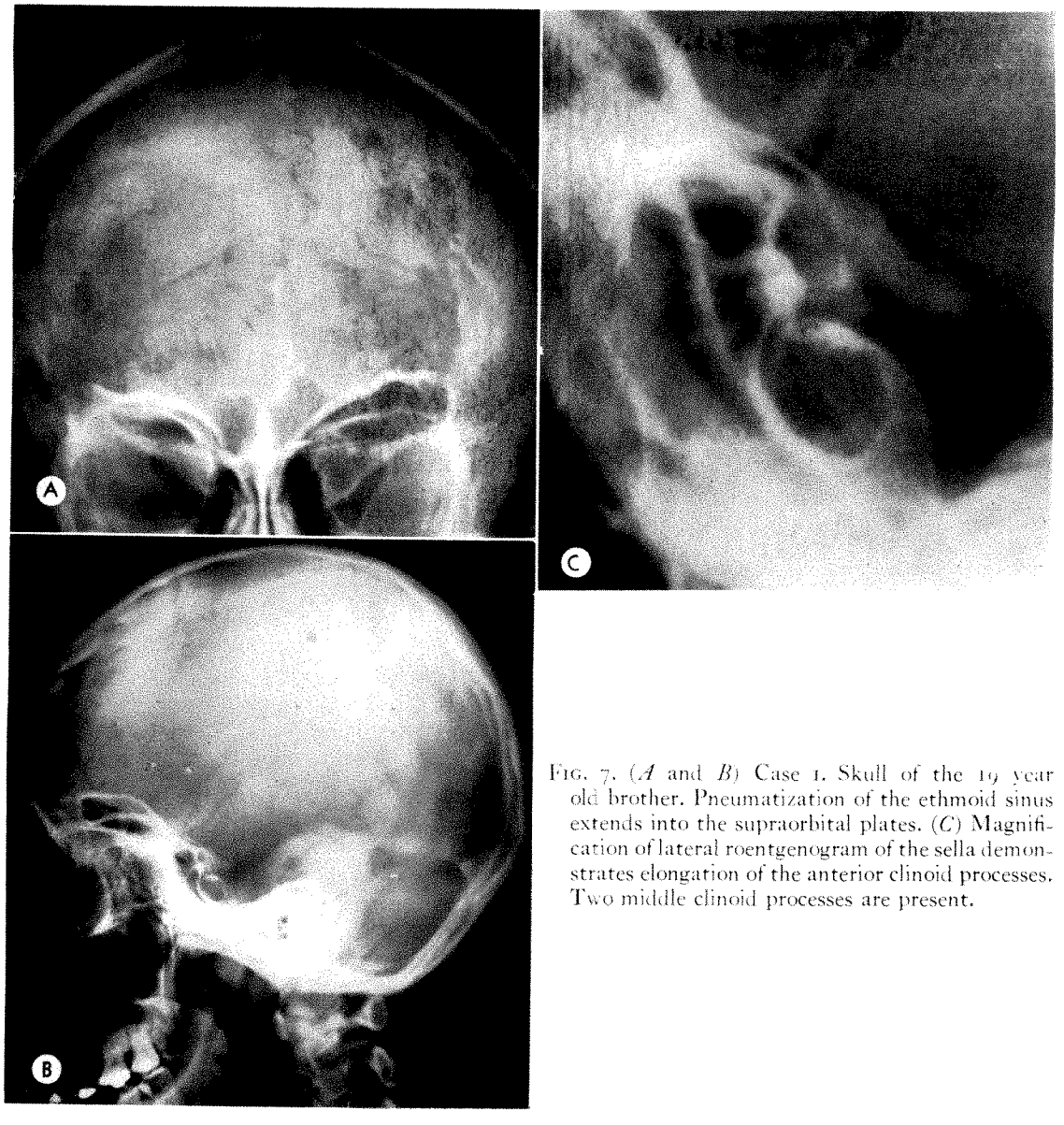



FIG. 7. (*A* and *B*) Case 1. Skull of the 19 year old brother. Pneumatization of the ethmoid sinus extends into the supraorbital plates. (*C*) Magnification of lateral roentgenogram of the sella demonstrates elongation of the anterior clinoid processes. Two middle clinoid processes are present.




Fig. 7. (*D* and *E*) Case II. Skull of the 11 year old brother. In both patients brachycephaly, calcification in the falx cerebri, and excessive inclination and depth of the sella are present. The diploic space is markedly diminished.

bones, in which excessive cortical endosteal thickening compromised the marrow cavity. The peculiar densities in the femoral necks of the older brother may have represented localized regions of myelofibrosis secondary to fat atrophy. The dense transverse bands in the vertebrae of the younger brother were more difficult to explain; we have not seen similar bands before.

Bone marrow aspiration of the iliac crest was unsuccessful in the older brother, the bone being described as more resistant to the needle than usual. A marrow biopsy was successfully performed in the younger brother and showed normal cellular elements, but a complete absence of marrow fat. Jackson[9] has described the transformation of marrow fat into skeletogenic cells by "lipo-skeletogenic modulation." Such a

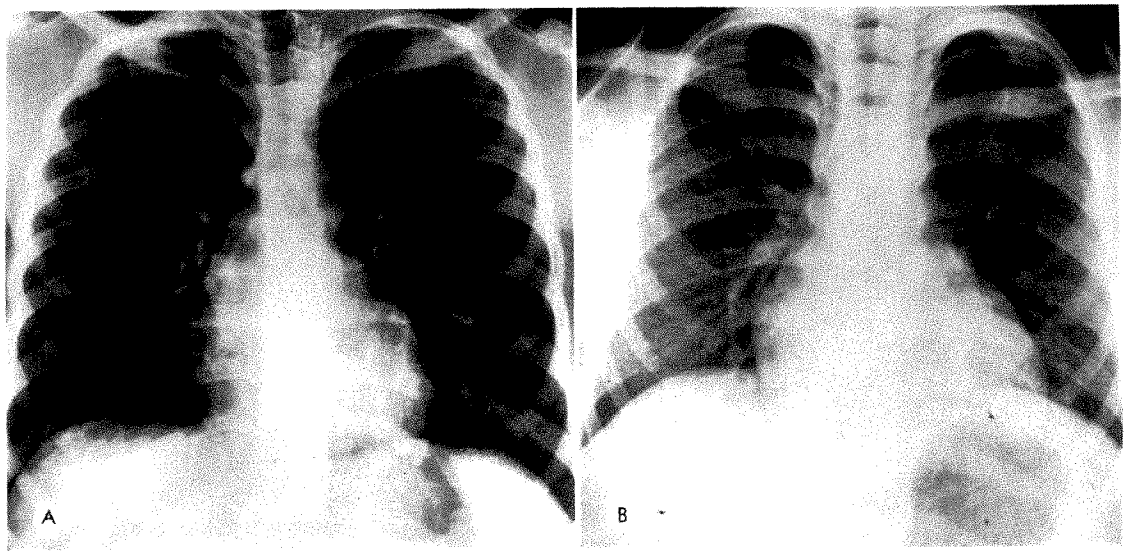



Fig. 8. (*A*) Case I. Chest of the 19 year old brother. (*B*) Case II. Chest of the 11 year old brother. Mild cardiomegaly and splenomegaly are present in both.



FIG. 9. Case 1. Right lower quadrant of the abdomen of 19 year old brother. The properitoneal fat stripe is absent.

process could explain the relationship of increased bone density to absent marrow fat.

In the younger brother the proximal femoral, proximal humeral, metacarpal, and metatarsal epiphyses were unusually elongated. Hepatosplenomegaly was present in both brothers and enlarged kidneys, possibly containing cysts, were demonstrated in the older brother. Cardiomegaly was present in both; it may reflect hypertrophy of cardiac muscle similar in origin to the puzzling hypertrophy of skeletal muscle. The skull findings were distinctive. The falx cerebri in both brothers was calcified.

## DIFFERENTIAL DIAGNOSES

*Diencephalic syndrome of infancy.*[13] First described in 1951, this syndrome consists of profound emaciation with accelerating early growth, increased local motor activity, and euphoria. It is caused by a tumor in the region of the anterior hypothalamus, most frequently an astrocytoma. The affected infants are normal at birth, but become symptomatic before 7 months. Death is usual before the end of the second year. Increased intracranial pressure is a late sign. Progressive emaciation leads to a loss of subcutaneous fat, producing a roentgenographic appearance of the soft tissue similar to that of lipoatrophic diabetes, except for less prominence of the muscles. Dense bones, hepatomegaly, genital enlargement, and hyperlipemia, features of lipoatrophic diabetes, have not been described in the diencephalic syndrome. Pneumoencephalography will demonstrate a tumor in the region of the anterior portion of the third ventricle.

*Progeria (the Hutchinson-Gilford syndrome).*[19] An incomplete loss of adipose tissue occurs in progeria, beginning in the second year of life in an infant normal at birth. A beak shaped nose, small orbits, high arched palate, and total alopecia combine with premature senility to create a characteristic facial appearance. The anterior fontanelle remains open. The long bones are thin and osteoporotic with marked coxa valga and characteristic elbow deformities. The distal phalanges of the hands and feet are atrophic. These

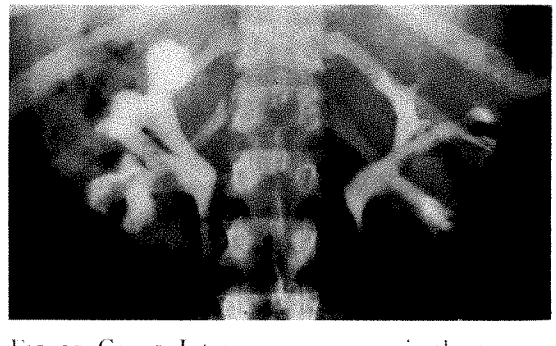


FIG. 10. Case 1. Intravenous urogram in the 19 year old brother shows that the renal calyces and infundibula are splayed bilaterally with mild calyceal blunting on the right. The renal outlines are lost because of absence of perirenal fat.

abnormalities are quite unlike those of lipoatrophic diabetes. Moreover, in spite of the severe arteriosclerosis in these patients, the values of the circulating lipids have been reported to be normal.

*Other conditions characterized by partial loss of subcutaneous fat.* (a) Renal hyperphosphatemic dwarfism (chronic glomerular failure) in children. In this disease the bones are usually decreased but may be increased in density with typical rachitiform changes and frequently changes of secondary hyperparathyroidism. (b) Hypophosphatemic vitamin D refractory rickets with amino aciduria (Debré-de-Toni-Fanconi syndrome), which is associated with decreased skeletal density and rachitiform changes. In mild cases, however, amino-aciduria may not be present and the bones may be actually increased in density.[24] (c) Chronic starvation.

SUMMARY

The syndrome of lipoatrophic diabetes is reviewed. The unique roentgenographic findings in 2 brothers with the congenital form of this disease are presented. Absent body fat, combined with well developed muscles, produced a characteristic appearance of the soft tissues. Other striking features were a generalized increase in skeletal density, localized areas of bone sclerosis, unusually elongated epiphyses, abnormalities of skull, and organomegaly. Advanced skeletal maturation was demonstrated in the younger brother. The possible pathophysiologic significance of some of these findings is discussed.

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# KNEE ARTHROGRAPHY

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THE first paper to be published regarding knee arthrography was written by Werndorff and Robinsohn[15] in 1905. The procedure did not acquire practical importance until the 1930s and 1940s when several detailed presentations on this subject were published.[1,10,13] These authors used oxygen or room air as their contrast medium and injected it directly into the joint. Others attempted pneumoarthrography by outlining the menisci with air produced within the joint as a manifestation of the vacuum phenomenon.[5,9] This method had little practical importance because it was not readily reproduced and difficult to attain if there was any fluid within the joint. It was, therefore, of little use in most of the patients suffering internal derangements of the knee.

Pneumoarthrography utilizing injected room air or oxygen achieved only a limited popularity as its accuracy left something to be desired when attempted by those not fully conversant with the method. While it was of value in examining the menisci, joint bursa and recesses, its use in evaluating the cruciate and medial collateral ligaments was negligible.

Initial attempts at positive contrast arthrography with opaque contrast media proved its usefulness in delineating the internal structures of the knee, but the media were irritating to the synovium and resulted in a postarthrography synovitis[11] in a large percentage of the cases. When opaque media were developed that proved less irritating, the use of positive contrast arthrography spread. Not only could one study the menisci, joint bursa and recesses, but one could outline the cruciate ligaments and identify ruptures of the medial collateral ligament. The accuracy of this method was better than with air, its range of usefulness greater, and it was more readily reproduced. Lindblom's monograph in *Acta radiologica* in 1948[8] probably did more to advance the use of this procedure and to detail the diagnostic criteria than any other publication. Lindblom used perabrodil and uroselectan B as his contrast material and had about a 5 per cent incidence of synovitis. With the newer media available to us today, no clinically detectable irritation of the synovium is produced.

Recently double contrast arthrography utilizing room air in conjunction with an opaque contrast medium has been advocated.[4] This method has also proved safe and accurate.

It is the purpose of this paper to show that positive contrast arthrography is safe, useful and a highly accurate procedure which can provide the physician treating the patient with information which he can utilize in making his decisions regarding therapy. A knowledge of the anatomy, pathologic processes, and the clinical features pertaining to internal derangements of the knee is an invaluable aid in the proper evaluation of these studies.

## CLINICAL PATHOLOGIC FEATURES

Not uncommonly, the minor as well as the more severe injuries of the knee will produce the same clinical pattern. Invariably the patient presents with pain, joint effusion, and the knee held in a position of semiflexion due to muscle spasm. The minor injuries recover rapidly following conservative treatment with little or no residual disability. The more serious injuries recover slowly or not at all, and the recovery depends on proper diagnosis and treatment. With fractures, the diagnosis is obvious and treatment prompt, but with ligamentous and cartilaginous injuries diagnosis often is not obvious and correct treatment is delayed, or omitted. This can result in near and long term disability which is undesirable and for some people,

such as professional athletes, disastrous.

The minor injuries include simple contusions of the soft tissues about the knee joint and traumatic synovitis—a diagnosis of exclusion. To make this diagnosis without excluding all types of internal derangements can result in much irreparable harm. The presence of an effusion indicates injury of the synovium. Trauma of fibrocartilage which does not involve synovial attachments produces no effusion. Most of the degenerative transverse tears of the menisci are in this category. An effusion may or may not be bloody depending upon the injury. If the synovium is irritated but not torn there is a serous effusion. Since the menisci are relatively bloodless except at their peripheral attachments, tears of the menisci may be accompanied only by a serous effusion. Because of their relatively rich blood supply, tears of the cruciate ligaments and the medial collateral ligament often are accompanied by a bloody effusion. Smillie[12] points out that bloody effusions usually develop within an hour of the time of injury, while the serous effusions develop over about 6 hours.

It is apparent that the presence or absence of fluid, and its nature has no direct relationship to the severity of the trauma. A transverse tear of the meniscus can occur without effusion, while trauma to the synovium alone can produce a bloody effusion. A knowledge of the mechanism by which an effusion occurs can lead to a correct impression of the underlying pathology if this information is correlated with other signs and symptoms.

Injury of the medial meniscus is a common occurrence and one of the most frequent causes of internal derangement of the knee. The medial meniscus is injured much more frequently than the lateral meniscus. This has been explained by the fact that the medial meniscus is much less mobile as a consequence of its firm peripheral attachments to the joint capsule and the medial collateral ligament. Injuries of the medial meniscus usually result from an inward twisting or rotation of the femur on the tibia when bearing weight with the knee in the semiflexed or nearly extended position. A common cause is the clipping violation in football. This same mechanism can produce medial plateau fractures of the tibia, and these fractures are not uncommonly associated with injuries of the medial meniscus.

Injuries of the medial meniscus can produce various signs and symptoms. With vertical tears one can get locking. This locking has to be distinguished from the knee which is held in the semiflexed position because of muscle spasm and/or joint effusion. Locking resulting from loose bodies is usually momentary and not associated with an athletic activity.[12] Some patients with posterior horn tears complain of a feeling that the knee is giving away, especially when turning around or walking on uneven ground. They have the sensation of something slipping in the joint.[12] Local swelling may occur over the joint line especially with tears in the posterior horn. Local tenderness along the joint line over the site of injury is common. Effusions may or may not be present and they may or may not be bloody. Quadriceps wasting occurs invariably in untreated cases. Medial meniscus injuries are often associated with injuries of the anterior cruciate and/or the medial collateral ligament. The association of all three is called O'Donoghue's triad.

Lateral meniscus injuries are uncommon compared to injuries of the medial meniscus. The lateral menisci are less likely to be injured, as the reverse type of rotational strain is much less likely to occur. Tears of the lateral meniscus are most common in the posterior horn, and longitudinal tears of the posterior horn often remain confined just to this segment; hence, the incidence of locking is low. In fact, the signs and symptoms which accompany tears of the lateral meniscus are invariably less severe than those accompanying a similar injury of the medial meniscus. Symptoms of the

lateral meniscus injury can refer to the medial side of the joint although the signs will be confined to the lateral aspect. The reverse is rarely true. This apparently results from an interference in the normal function of the anterior cruciate ligament and the medial meniscus as a consequence of an injury to the lateral meniscus.[12]

Medial collateral ligament injuries are either incomplete ruptures, a so-called sprain, or complete ruptures—an injury of great importance. These injuries usually result from a valgus strain on the knee associated with twisting of the femur inward or tibia outward with the knee in a slightly flexed position. The medial collateral ligament has both a superficial and a deep layer. Sprain or partial avulsion of the long fibers from their femoral attachment is a very frequent occurrence. It may or may not be associated with the detachment of a small piece of cortical bone. If this lesion is overlooked, calcification of the ligament may occur, the so-called Pellegrini-Stieda's disease. This can result in severe and prolonged disability. Although avulsion from its femoral attachment is most common, the medial collateral ligament can tear at any level. A not infrequent site of rupture is at the tibial attachment. Complete rupture of the medial collateral ligament is a serious injury and is often associated with other injuries such as a tear of the anterior cruciate ligament or, rarely, a tear of the posterior cruciate ligament. This injury can also be associated with fracture of the lateral tibial plateau or peripheral tears of the medial meniscus. When a complete rupture has occurred, widening of the medial joint space is an associated finding and can be demonstrated with forced abduction. A hemarthrosis will occur if the capsule is intact, but if not, the blood may drain into the soft tissues medially producing a brawny periarticular thickening. Later ecchymotic areas appear. The patient is tender at the site of disruption and occasionally a gap in the ligament may be palpated.

Injuries of the lateral collateral ligament are infrequent and occur when the joint is exposed to powerful adduction of the leg or the thigh. Complete rupture of this ligament rarely occurs as an isolated injury but is commonly associated with injuries to the lateral popliteal nerve, iliotibial band, popliteus tendon, joint capsule, and cruciate ligaments. Detachment usually occurs at its fibular attachment.

The anterior cruciate ligament can be injured as a result of an abduction force, then it is associated with an injury of the medial collateral ligament and possibly also a lateral plateau fracture. If injured as a result of a rotational force, it is often associated with tears of the medial meniscus. If injured as a result of a direct blow on the posterior aspect of the upper tibia, or the anterior aspect of the lower femur, it may be an isolated lesion. It can detach at any point but most commonly this occurs at its femoral attachment. When it detaches at its tibial attachment, it often is an isolated lesion and commonly a small fragment of the tibial spine is avulsed with the ligament. These injuries produce a hemarthrosis, restricted motion, and the anterior drawer sign. Examination of the patient under anesthesia after aspiration of the joint may be required to demonstrate this sign.

Injuries of the posterior cruciate ligament are not common. They usually result from a direct blow on the anterior aspect of the proximal tibia with the knee in a semiflexed position. These occur most frequently as a consequence of a dash-board injury in car accidents. The posterior crucial ligament usually detaches from its tibial attachment and may cause avulsion of a small fragment of bone. It also results in a hemarthrosis and restricted joint motion. The diagnostic sign is the posterior drawer sign, the tibia moving more posterior on the femur than normally. This sign may appear spontaneously with the patient supine and the knee in a semiflexed position.

PLAIN ROENTGENOGRAPHIC STUDIES

In almost all cases, the routine roentgenograms will be normal or show evidence of joint distention as a consequence of the traumatic synovitis or hemarthrosis which accompanies most, but not all, internal derangements. More specific findings indicating the nature of the injury are almost always absent.

With old neglected meniscal injuries, joint space narrowing and marginal spurring may be seen. Occasionally, with the production of a vacuum phenomenon, a torn meniscus may be visualized. Rarely old torn menisci may be calcified. The calcium usually forms in the torn or free fragment. This is a rare late sign and should not be confused with primary meniscal calcification as described by Weaver[14] due to various causes. Occasionally tibial plateau fractures are associated with meniscal tears, and thus evidence suggesting an old fracture of this type might alert one to the possibility of a meniscal injury.

With complete tears of the collateral ligaments, joint instability can be demonstrated medially with forced valgus strain on the extended knee and laterally with forced varus strain. Complete rupture of the lateral collateral ligament is a very rare injury. The medial collateral ligament is injured not infrequently. Sometimes small flecks of bone can be avulsed when these ligaments are injured. With the medial collateral ligament injury, the avulsed bone usually arises from its femoral attachment, while with the lateral ligament injury the avulsion usually occurs from the fibula.

With injuries of the anterior cruciate ligament, fragments of bone can be avulsed, usually from its tibial attachment. The same can occur with injuries of the posterior cruciate ligament but injury of this ligament is fairly uncommon.

Because of the paucity of findings with internal derangements, especially in fresh cases, positive roentgen diagnosis depends on contrast arthrography. With this method much more accurate information is available.

TECHNIQUE OF KNEE ARTHROGRAPHY

The technique of knee arthrography will be described in great detail so that the technical errors which are most commonly the cause of diagnostic error can be eliminated to as great an extent as possible.

Routine roentgenograms of the knee should be available prior to initiating this procedure. Important findings may be overlooked unless this is done in every case. These findings have already been discussed.

In addition to a routine knee examination, preliminary roentgenograms of the knee are made in the anteroposterior projection. These roentgenograms are made utilizing an extension cone for improved detail and with 0, 5 and 9 degrees of caudal angulation. The purpose of these studies is to establish correct exposure factors and the correct angulation required to direct the central beam through the joint space to produce the greatest width between the femoral condyles and the tibial plateaus. In general, this will require some caudal angulation, for with the knee extended in the anteroposterior position, the tibial plateaus slope caudally. In those patients unable to fully extend their knee, less caudal angulation will be required to direct the central beam through the joint space properly. In this situation, the preliminary roentgenograms should be made with 5 degrees of cranial angulation, zero angulation and finally, with 5 degrees of caudal angulation.

The two angles best suited for an optimum projection of the joint space are chosen. Two roentgenograms are made in each projection as this allows for verification of tears and exclusion of overlapping bony and bursal shadows. In most patients, the two optimal angles are the 5 and 9 degrees of caudal angulation. Another method of establishing the correct angulation is to examine the knee fluoroscopically in the

lateral projection with the leg as extended as possible and mark on the skin the slope of the tibial plateaus. However one wishes to accomplish this, it is necessary that the roentgenograms be made with the proper inclination so that the contrast outlined menisci are demonstrated with the least amount of overlapping by adjacent structures.

After obtaining the preliminary roentgenograms one can proceed with the examination. The skin at the site of injection should be properly cleansed. The injection is made on the lateral aspect of the knee at the level of the mid patella. Cleanse the area for 10 minutes with septisol; following this, the skin is prepared with merthiolate. This approach is laborious but necessary to insure prevention of a septic joint.

One per cent Xylocaine is used for local anesthesia with a 1½ or 2 inch 21 gauge needle. Local anesthesia is initiated at the site of puncture and carried into the joint from this approach. Displacement of the patella laterally with the free hand facilitates entry into the joint. One will know when the joint is entered as the resistance to the injection of Xylocaine suddenly lessens. After the joint has been entered, the fluid within the joint is removed as completely as possible. A normal joint may contain 1 or 2 cc. of clear or straw colored fluid. Complete removal of fluid can be facilitated by using a pad against the posterior recesses so that they can be emptied and by squeezing the knee both above and below the patella. This should cause the excess fluid to drain through the needle inserted into the joint space. If removal of fluid is incomplete, the contrast material will be diluted and poor coating of the structures to be examined can occur and thus be a source of diagnostic error. The amount of fluid removed and its character are recorded for the referring physician's information.

After the joint has been aspirated, 10–15 cc. of 50 per cent hypaque or conray is injected under fluoroscopic control. It is preferable to use 15 cc. when dealing with a large patient or when there is a suspicion of unremoved fluid in the joint in order to overcome the dilution effect. I always inject under fluoroscopic control to prevent extracapsular injection. One should take care not to introduce air bubbles as these shadows can be confusing.

Following the injection of the contrast material, the needle is removed and the joint exercised as vigorously as possible to ensure adequate distribution of the contrast medium. Flex and extend the knee several times, then abduct, adduct and rotate the lower leg. In patients who are unable to flex or extend their knee, encourage them to stand and bear weight. Adequate exercise of the joint after injection of the contrast medium is necessary if small meniscal tears are to be demonstrated. Once this has been accomplished, a 3 inch ace bandage is wrapped from the top of the suprapatellar bursa inferiorally to the joint line as tightly as possible, so as to compress the joint bursa and recesses and force contrast material into the joint space. By wrapping to the joint line, the lower edge of the bandage can be used as a centering mark by the technician for the exposures to follow.

Since the contrast medium is absorbed fairly rapidly, films have to be exposed as fast as possible. Heiser and his associates[6] have pointed out that undue delay in exposing films can result in nondemonstration of tears visualized on roentgenograms made earlier in the same position. The radiologist can increase the speed of this procedure by donning apron and gloves, positioning the patient himself, and immobilizing the part with his lead covered hands. This allows the technician to concentrate on the centering of the tube and film to the part. The technician can also exchange films more rapidly. A second technician is responsible for proper exposure of the film. By utilizing this procedure, completion of the entire series of arthrograms in 3 to 4 minutes is possible.

The required views depend on the angles chosen earlier. Most commonly one chooses 5 and 9 degrees of caudal angulation. Under these circumstances the following arthrograms are made in the order listed:

| *Angle* | *Position* |
|---|---|
| 9° caudal | anteroposterior |
| 9° caudal | 45° internal rotation |
| 9° caudal | 70° internal rotation |
| 5° caudal | 70° internal rotation |
| 5° caudal | 45° internal rotation |
| 5° caudal | anteroposterior |
| 5° caudal | 45° external rotation |
| no angle | 45° external rotation |
| no angle | 70° external rotation |
| 5° cranial | 70° external rotation |
| variable | true notch view—posteroanterior |
| no angle | lateral with 70° flexion |
| no angle | lateral with 70° flexion with pressure exerted so as to stretch anterior cruciate ligament. |

Some comment about this method would seem in order. The amount of tube angulation change is kept to a minimum. It is quicker to change the patient's position than to change the tube angle. I have chosen to use the true notch position as recommended by Lindblom,[8] rather than posteroanterior views with cranial angulation as recommended by Heiser *et al.*[6] In the true notch position, the tube and tibia should be parallel with the central beam centered through the joint. Excellent visualization of the menisci will result, especially of the lateral meniscus with the popliteus bursa projected lateral to the meniscus. The ace bandage is now removed and lateral arthrograms obtained. The lateral arthrograms should be sufficiently penetrated to visualize the cruciate ligaments. If this is done, tears of these ligaments can be readily seen. At least one lateral arthrogram is made with the knee flexed 70° and with pressure on the tibia. Pushing it forward in relation to the femur causes the anterior cruciate ligament to be taut. Visualization of a taut ligament helps exclude a tear of this structure. The angles listed are those used if the preliminary roentgenograms with caudal angles of 5° and 9° are chosen. If other angles are chosen, these have to be adjusted. For instance, if 0° and 5° of caudal angulations are chosen all anteroposterior and internal rotation arthrograms are made with these angles. The 45° external rotation arthrograms are made with 0° and 5° of cranial angulation. The 70° external rotation arthrograms are made with 5° and 10° of cranial angulation. Various other substitutions may be made if one follows the same formula. One should recognize that the greater the degree of external rotation, the greater the shift of the angle from caudal to cranial required to keep the joint space in profile.

Following the procedure, the arthrograms are viewed and if satisfactory the patient is released. The contrast material does not have to be aspirated as it absorbs rapidly and is not irritating.

## COMPLICATIONS AND CONTRAINDICATIONS

In this series, no complications were encountered. Allergic reactions to the local anesthetic or contrast material are possible and one should be alert to this. A known allergy to the contrast material would be a contraindication to this procedure. The procedure could easily be done without a local anesthetic if necessary. A serious bleeding tendency would also be a relative contraindication.

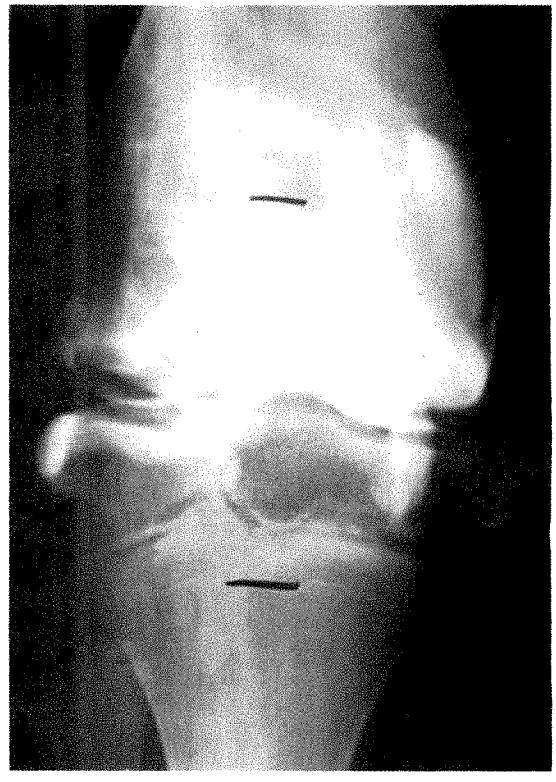
Joint sepsis is a dreaded complication but is preventable with meticulous technique.

This procedure can be done almost anytime in relation to the onset of injury. During a period of active bleeding with rapid accumulation of fluid in the joint, the procedure would be more difficult as the joint would prove difficult to aspirate sufficiently to prevent dilution of contrast material.

## THE NORMAL ARTHROGRAM

The appearance of the normal arthrogram is best appreciated after a careful study of normal examinations. In this way,

Fig. 2. Normal arthrogram, notch projection. Note the contrast filled popliteal bursa nearly overlapping the edge of the medial meniscus. Frequently, this overlapping is more pronounced.



the variations which can cause confusion can be recognized and errors of interpretation eliminated.

The menisci are visualized as sharply delineated triangular radiolucent shadows. With the patient in the anteroposterior or tunnel positions (Fig. 1 and 2), the midportion of the medial and lateral menisci is demonstrated. In internal rotation (Fig. 3) one sees the anterior portion of the medial meniscus and the posterior portion of the lateral meniscus. Just the reverse

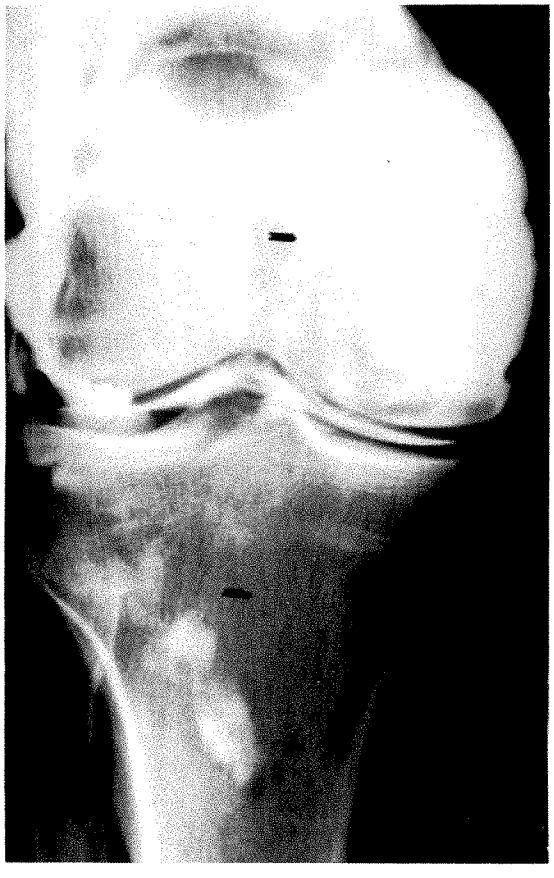


Fig. 1. Normal arthrogram, anteroposterior projection. Note prominent recess at superior lateral aspect of the lateral meniscus.



Fig. 3. Normal arthrogram, 45° internal rotation. Note the contrast medium filled bursa of the popliteus tendon overlapping the lateral meniscus.



FIG. 4. Normal arthrogram, 45° external rotation.

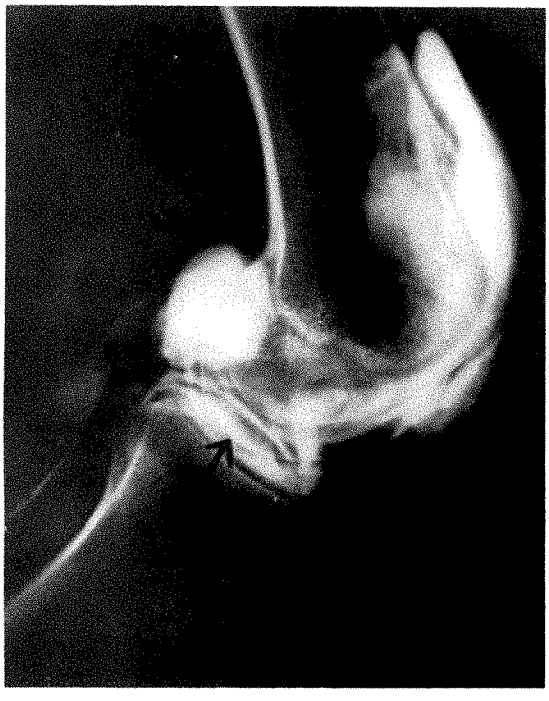


FIG. 5. Normal arthrogram, lateral projection. Note the anterior and posterior cruciate ligaments at the arrow and the prominent popliteal bursa posteriorly.

occurs with external rotation (Fig. 4). According to Lindblom,[8] the medial meniscus averages 6 mm. in width in its anterior and medial portions but posteriorly enlarges to average 14 mm. in width. The lateral meniscus is more nearly the same width throughout averaging about 10 mm. The attachments of the menisci to the capsule are complete except for the posteromedial portion of the lateral meniscus, where it is crossed by the popliteus tendon and its accompanying bursal sheath (Fig. 3). On rare occasions, it will not communicate with the joint space and thus is not outlined by the contrast medium.

The cruciate ligaments are visualized on properly exposed lateral arthrograms (Fig. 5). They appear as radiolucent bands extending from their femoral to their tibial points of attachment. The anterior cru-

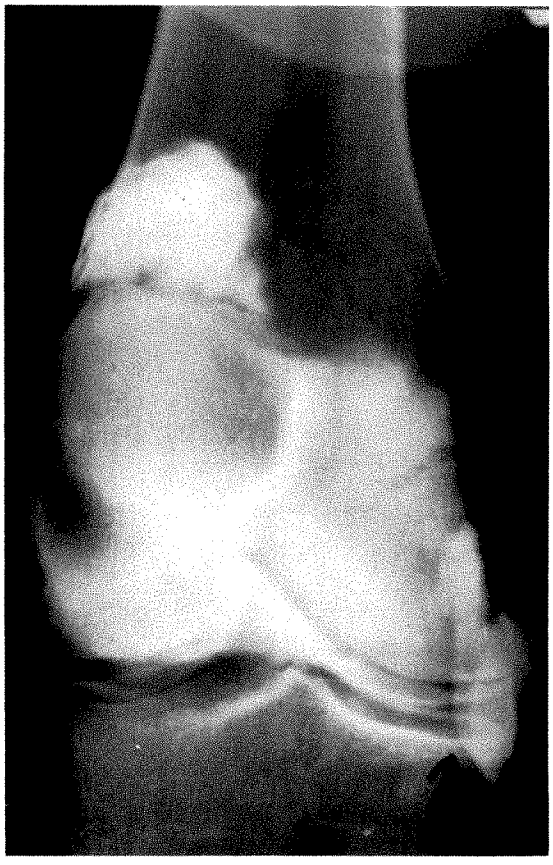


FIG. 6. Notch view with the popliteal bursa obscuring the medial meniscus (arrow). In this instance a tear was overlooked.

ciate ligament runs anteriorly to attach to the tibia at about a 45° angle. It measures about 5 mm. in width. The posterior cruciate ligament runs posteriorly to attach to the tibia and is about 7 mm. in width.

The medial collateral ligament is not directly visualized by this procedure. It is firmly attached to the medial meniscus. Brantigan and Voshell[2] found an inconstant bursa between these structures but Lindblom[8] points out that this is pathologic and indicates a peripheral detachment of the meniscus.

The lateral collateral ligament lies outside the capsule and tears of this structure cannot be visualized by this procedure.

Other normal structures which can be visualized include the bursal and joint recesses. The suprapatellar bursa is always well outlined and, except on rare occasions, communicates with the joint space proper. The bursa accompanying the popliteus tendon is almost uniformly visualized and in about one-third of the cases communicates with the tibiofibular joint. The bursa found with the tendons of the semimembranous-gastrocnemius is present in about 20 per cent of the cases. An expansion of this bursa is called a popliteal or Baker's cyst (Fig. 5). This bursa can overlap the medial and posterior portions of the medial meniscus when filled with contrast medium (Fig. 6) and unless the arthrograms are properly penetrated tears can be missed. The same problem occurs on the lateral side with the bursa of the popliteus tendon overlapping the medial and posterior aspects of the lateral meniscus when filled with contrast medium (Fig. 3). Proper penetration and routine inspection of the tunnel arthrogram will help avoid errors. In the tunnel position (Fig. 2), the popliteus tendon and bursa are displaced lateral to the lateral meniscus.

In addition to these bursae, many joint recesses are present which readily fill with contrast medium. These are often prominent in the para-articular portions of the tibia and femur. When filled, they can overlap the menisci and simulate a tear. A second arthrogram in the same position made with a different angle helps to avoid

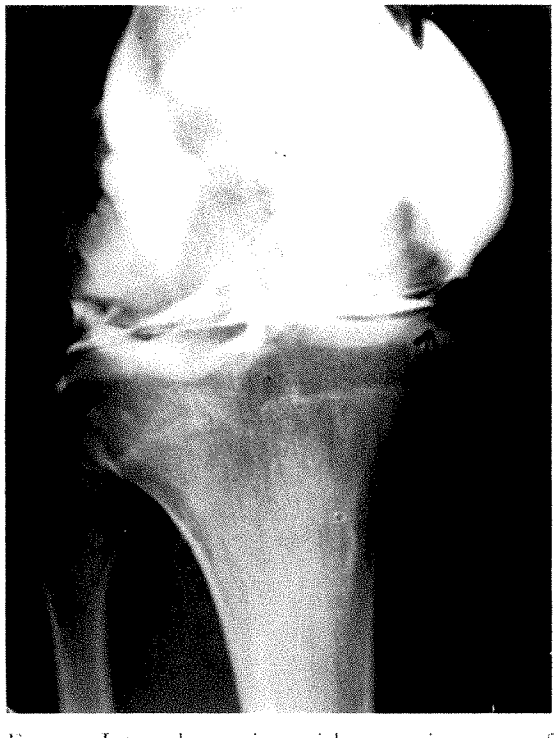


Fig. 7. Internal rotation with extensive tear of anterior horn of the medial meniscus (arrow). The tear is a bucket handle type with a transverse component.

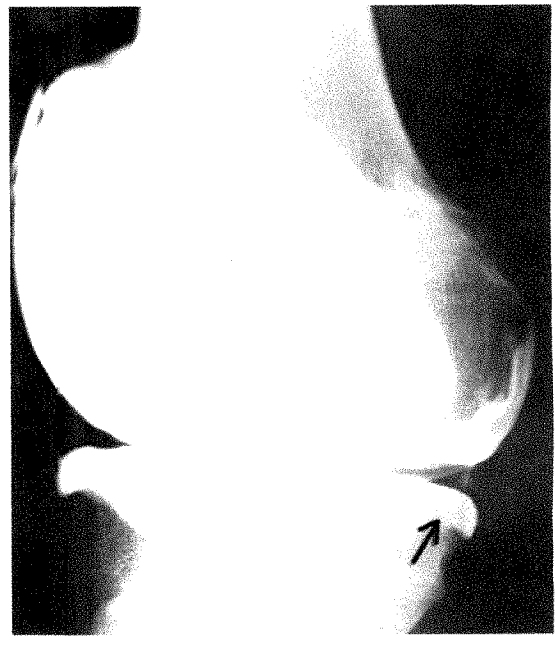


Fig. 8. Peripheral tear of the posterior horn of the medial meniscus (arrow).

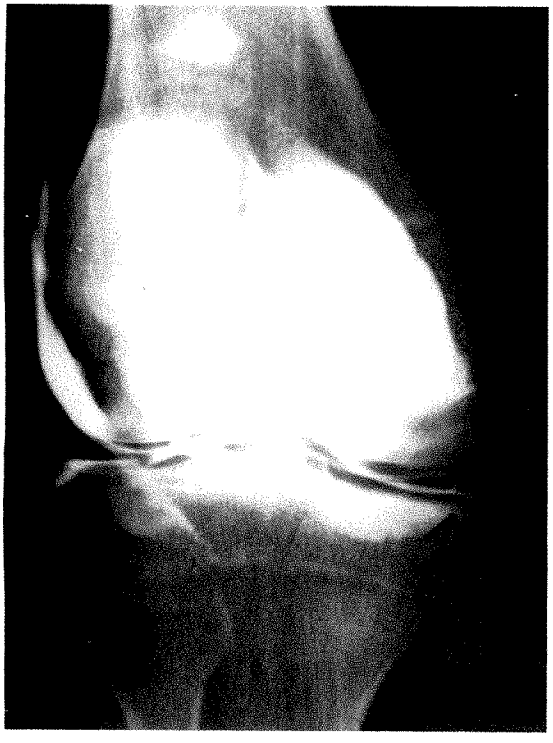


FIG. 9. Extensive tear of the posterior horn of the medial meniscus with vertical and transverse components (arrow).

this type of diagnostic error. Recesses at the capsular attachments of the menisci are present (Fig. 1 and 2) except at the anterior attachment of the medial meniscus. The recesses medially are usually shallow, according to Lindblom,[8] measuring 1 mm. or less. A rather large recess is often found in association with the anterior portion of the lateral meniscus at its tibial edge. Along the medial and posterior portions of the lateral meniscus the irregular recesses associated with the bursa of the popliteus tendon may be confusing if one is not alerted to their presence.

Finally, the joint space itself is outlined and indirectly the infrapatellar fat pad (Fig. 5). If this enlarges, encroachment on the joint space occurs and can be detected.

## THE ABNORMAL ARTHROGRAM

Abnormalities are readily detected if one is fully conversant with the types of normal variations one may encounter. Arthrography of the knee is performed primarily to detect meniscal lesions. The most common lesion of meniscus is a tear. Tears can involve a limited or extensive region and are of various types. Arthrographically they can be classified as:

A. Peripheral detachment from capsular attachment
B. Tear of the meniscus proper
   1. Vertical
      (a) without displacement
      (b) with displacement of central fragment (bucket handle)
   2. Transverse
   3. Combinations—this is common in extensive injuries.

Peripheral detachments are identified by the presence of the positive contrast medium between the base of the meniscus and the attachment of the meniscus to the capsule (Fig. 8). This is true both on the medial and lateral sides except posterolaterally where the bursa of the popliteus tendon is

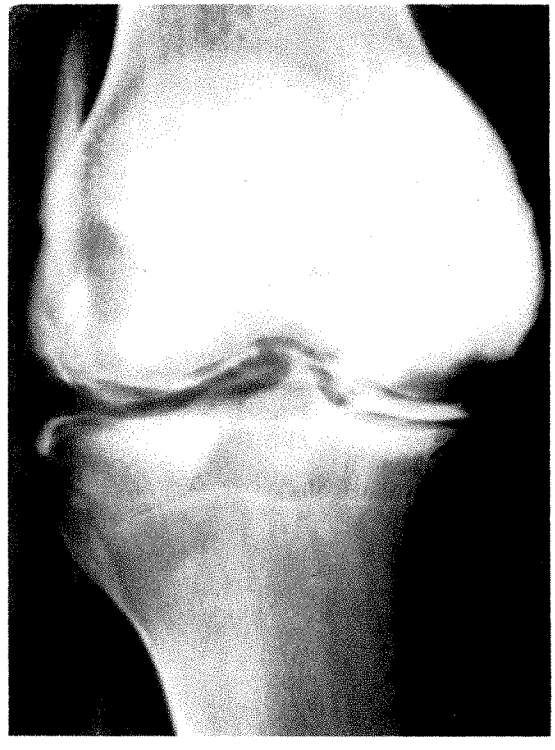


FIG. 10. Bucket handle tear of the medial meniscus (arrow).

situated between the base of the meniscus and the joint capsule.

Vertical tears can be quite extensive involving almost the entire meniscus or they can be localized to a segment such as the anterior or posterior horn. They are identified by the presence of a vertically oriented streak of opaque medium projecting into the meniscus proper (Fig. 9). One should be careful to differentiate a vertical tear from contrast medium in the adjacent bursa or joint recesses overlapping the body of the meniscus. Overlapping recesses can usually be projected away from the meniscus by varying the angle and, in addition, they usually project over the meniscus as a rounded collection of contrast medium, rather than as a sharply demarcated line. If the central meniscal fragment displaces into the joint, the edge of the meniscus will present a convex or flat border (Fig. 7 and 10) often somewhat irregular rather than the usual sharp tapered margins seen with normal menisci.

Transverse tears have been described as being the end result of minimal trauma incurred by a meniscus which is getting old. In other words, these occur in an older age group. They are recognized if a streak of opaque material is seen projecting into the meniscus oriented transversely or nearly so (Fig. 11). These can be difficult to demonstrate when compared to vertical tears as the contrast medium more readily enters the vertical rents.

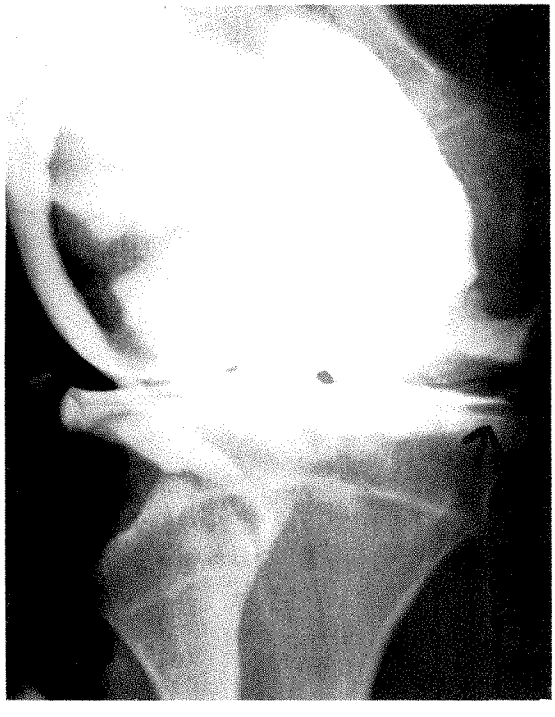


Fig. 11. Transverse tear of the posterior horn of the medial meniscus (arrow).



Fig. 12. Extensive tear of the posterior horn of the medial meniscus (arrow) with vertical and transverse components and with separation from the capsule peripherally.

Extensive tears of the meniscus can occur with tears being oriented vertically, transversely and obliquely (Fig. 12 and 13) in any combination.

If a meniscus has been removed surgically, one finds an absence of the lucent wedge formed by a normal meniscus when surrounded by opaque medium (Fig. 14). The degree to which this absence is complete reflects the extent of removal modi-

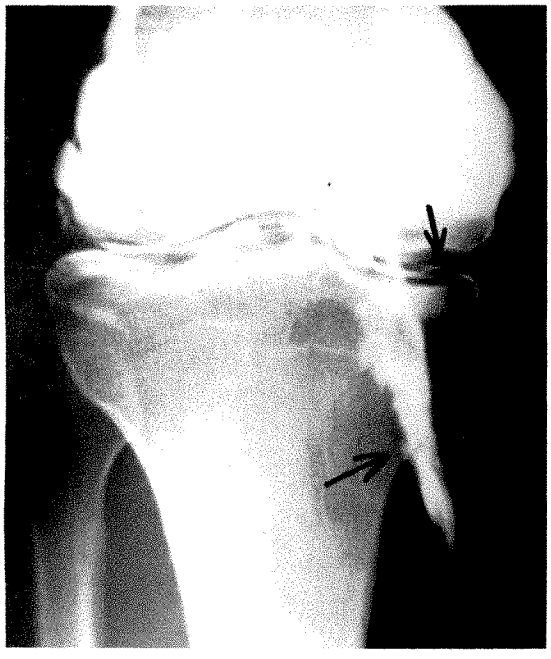


FIG. 13. Extensive tear of the medial meniscus with rupture of the popliteal bursa (upper arrow) and extravasation of contrast material into the posterior muscles of the leg (lower arrow).

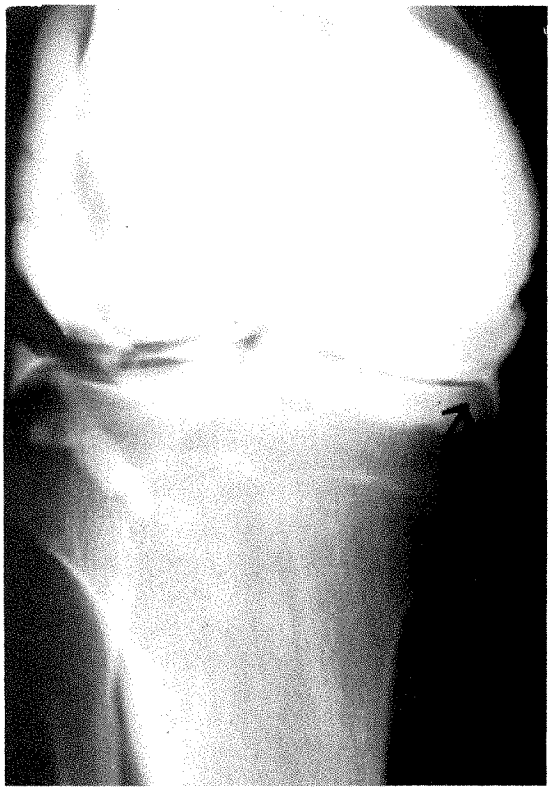


FIG. 14. Postoperative medial meniscectomy (arrow).

fied by the extent of the regrowth of the fibrocartilage. It is not common but one occasionally finds a retained posterior horn. On rare occasions, this retained posterior horn may harbor a tear.

Cysts of a meniscus are not too common. They are more common laterally than medially. Breck[3] states that they are 10 times more frequent laterally. They usually lie anterior to the lateral collateral ligament and they usually present outside the joint capsule. This is especially true of the largest ones. In these cases, they may cause some extrinsic pressure on the joint capsule and this can be detected on the arthrogram. In one case seen recently, a large cyst caused the bursa of the popliteus tendon to fill poorly (Fig. 15). The small cyst forming within the joint distorts the outline of the meniscus. These cysts often are associated



FIG. 15. Extrinsic compression of the joint capsule due to cyst of lateral meniscus (arrow).

with meniscal tears according to Lindblom[8] and Smillie.[12] Breck[3] does not agree.

Diskoid menisci are much more common laterally than medially and are more common in females than males. A diskoid meniscus is a congenital variation in which the meniscus forms a solid plate that extends completely between the articular surfaces. These menisci are wider and thicker than normal and extend almost to the midline (Fig. 16). They are quite susceptible to injury.

Ruptures of the anterior and posterior cruciate ligaments can be diagnosed arthrographically. Ruptures of the anterior cruciate ligament are much more common than ruptures of the posterior ligament. One will note on properly exposed films a lack of continuity of the ligament which will be manifested by the pooling of the opaque material in the gap caused by the rupture (Fig. 17). By applying forward pressure on the tibia the anterior cruciate ligament will hang lax if torn (Fig. 17), while with posterior pressure on the tibia, the posterior cruciate ligament will hang lax if torn. Cysts of these ligaments will be manifested as localized swellings.

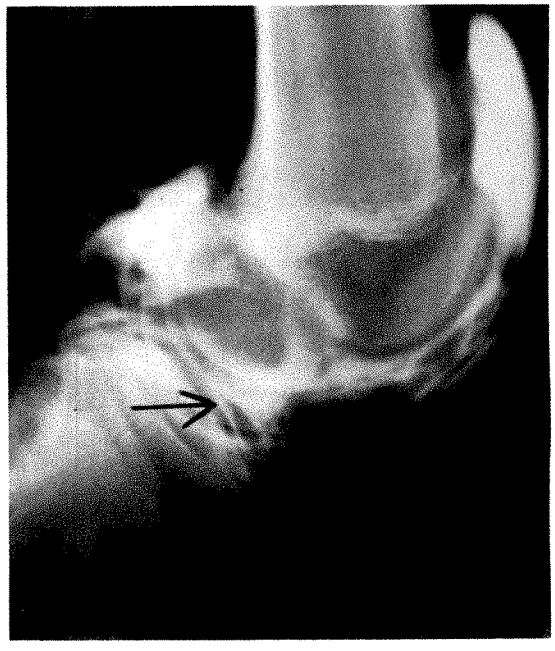


FIG. 17. Tear of the anterior cruciate ligament (arrow). Note lack of the continuity of the ligament. Compare with normal arthrogram (Fig. 5).

Tears of the medial collateral ligament can be identified if the synovial lining of the joint adjacent to this portion of the capsule is ruptured sufficiently to allow the positive

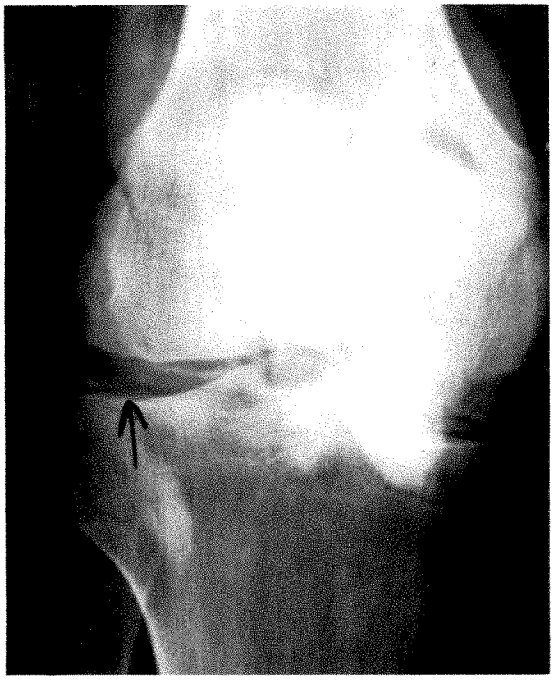


FIG. 16. Diskoid lateral meniscus with tear (arrow).

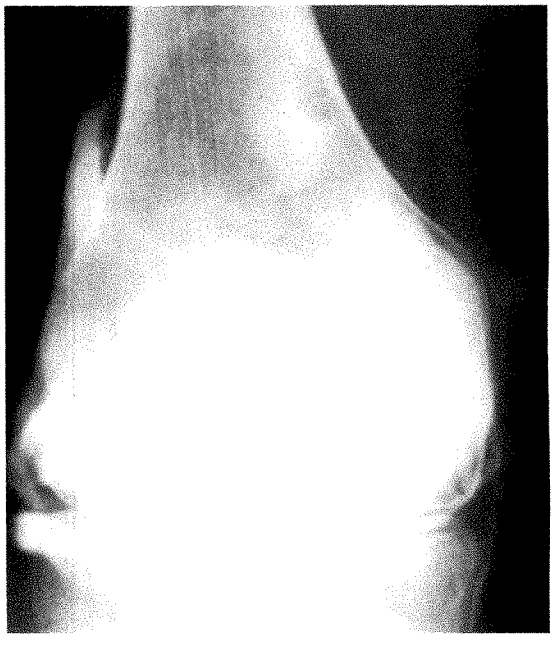


FIG. 18. Tear of the medial collateral ligament. Note contrast material in soft tissues of the medial side of the joint.



FIG. 19. Rupture of the popliteal bursa (arrow).

contrast material to escape from the joint into the soft tissues on the medial side of the joint (Fig. 18). The point at which the contrast medium escapes from the joint usually is at or near the femoral attachment. Injuries of the medial collateral ligament not involving complete rupture often are normal arthrographically, although in some incomplete ruptures, contrast medium may be noted in the soft tissue on the medial side of the joint usually confined to an area near the base of the meniscus.

Major capsular ruptures can be demonstrated. These most commonly occur posteriorly either from the popliteus bursa, or from the bursa of the semimembranous-gastrocnemius. This allows the opaque material to leak into the adjacent soft tissues forming an intramuscular deposit of irregular outline (Fig. 13 and 19). Ruptures of this type often occur when the joints are distended with fluid. They can occur as a result of an internal derangement or as a consequence of various types of knee pathology which produce a synovitis. As has been pointed out by Hench and associates,[7] posterior capsular ruptures with dissection of the fluid into the calf muscles can produce all the signs and symptoms of thrombophlebitis. In patients with these signs and symptoms and with known knee pathology, this possibility should be considered and is best diagnosed by arthrography.

Other less common entities can be recognized by this method. If a synovial tumor projects into the joint space, it will present as a filling defect. In a similar fashion, large nonopaque loose bodies can be detected. If the infrapatellar fat pad hypertrophies sufficiently to fill the space between the patella and anterior cruciate ligament, one can recognize the entity known as Hoffa's disease. Hypertrophy of this fat pad is associated with chronic synovitis and the fibrous lobules may overlap the menisci and cause pain and locking. Pigmented villonodular synovitis can also be recognized arthrographically as a result of the multiple lucent filling defects which almost obliterate the joint space proper.

## REVIEW OF MATERIAL AND EVALUATION OF RESULTS

It has been my good fortune to work with interested orthopedic surgeons who have permitted me to examine a total of 258 knees in 255 patients. Their interest is paramount in order to be able to examine a sufficient number of patients to gain the experience necessary to properly apply this method. It may seem redundant, but as other authors have indicated, the accuracy of the interpretations increases with experience. I can attest to that as 17 of the 25 errors occurred in the first half of this series.

Of the 258 knees examined, 122 had been operated on by the time this series was compiled. A careful review of this group will be undertaken. Of the remaining 136 knees, 89 were normal in every respect. Of these, 27 were lost to follow-up because they were evaluated on a one time basis for an industrial evaluation. Of the 62 that were followed, none have had a complaint serious enough to warrant surgery. Most were signed out as traumatic synovitis or as minor strains of the medial collateral ligament. All in this group were treated conservatively with apparent good results.

There remains a group of 47 patients who had abnormalities on the arthrograms who

TABLE I

MEDIAL MENISCUS

| Roentgen Diagnosis | No. of Patients | Surgical Diagnosis | No. of Patients |
|---|---|---|---|
| Normal medial meniscus | 32 | Normal medial meniscus | 26 |
| | | Torn medial meniscus | 6 |
| Torn medial meniscus | 82 | | |
| | | Torn medial meniscus | 81 |
| | | Normal medial meniscus | 1 |
| Retained posterior horn medial meniscus | 2 | | |
| | | Retained posterior horn medial meniscus | 2 |
| Previous complete removal of medial meniscus | 6 | Previous complete removal of medial meniscus | 6 |
| Total | 122 | Total | 122 |

have not undergone surgery. Of these, 5 had small peripheral tears of the medial meniscus treated conservatively with apparent good results. Another 21 had various abnormalities diagnosed but have been lost to follow-up, usually because they were evaluated on a one time basis for industrial purposes. Of the remainder, 16 had lesions diagnosed and surgery has been recommended but for various reasons has not yet been performed and, finally, 5 patients had persistent remnants of the posterior horn of the medial meniscus. In 2 of these no surgery was recommended, in 2 it has been recommended but not yet performed, and on the final patient, it was recommended and done elsewhere.

In the 122 patients who were subjected to surgery, good correlation between the roentgen interpretation and the surgical findings was obtained (Tables I, II, and III).

In addition to the findings tabulated on the Tables, 16 ruptures of the medial collateral ligament were diagnosed and confirmed. On no occasion was a complete rupture missed. On several occasions in both the treated and nontreated group, incomplete rupture of the medial collateral ligament was diagnosed involving only the short fibers. In those seen at surgery, this finding was confirmed in about half of the cases. In the remainder, no mention in the operative summary about the condition of the medial collateral ligament was available. The orthopedists feel that in many of the patients reported as normal, an incomplete tear of the medial collateral ligament had occurred which was treated conservatively with apparent good results. Evidently, this procedure is not too accurate in detecting incomplete tears of the medial collateral ligament, although when identified it is a useful positive finding.

No examples of torn posterior cruciate ligaments were included in this series. Since the lateral collateral ligament is not attached to the joint capsule, tears of this structure are not expected to be demonstrated with this method.

On one occasion, a rupture of a popliteal cyst with extravasion of opaque material into the calf was noted in conjunction with a torn medial meniscus.

Villonodular synovitis was identified in one patient and adhesive capsulitis in another. On several occasions, loose bodies could be identified within the joint but this procedure is not accurate in demonstrating nonopaque loose bodies because the opaque medium obscures most of these loose bodies sufficiently to preclude their roentgenographic demonstration.

The exploration of 122 knees after knee arthrography revealed 25 errors in interpretation. The evaluation of 122 medial

TABLE II

LATERAL MENISCUS

| Roentgen Diagnosis | No. of Patients | Surgical Diagnosis | No. of Patients |
|---|---|---|---|
| Normal lateral meniscus | 100 | Normal lateral meniscus | 93 |
| | | Torn lateral meniscus | 7 |
| Torn lateral meniscus | 21 | Torn lateral meniscus | 20* |
| | | Normal lateral meniscus | 1 |
| Previous complete removal of lateral meniscus | 1 | Previous complete removal of lateral meniscus | 1 |
| Total | 122 | Total | 122 |

* Of the torn lateral menisci, two were diskoid in type.

menisci produced 7 errors (Table 1). Of these, 6 were false negative studies. In 2 instances, transverse tears were found at surgery which apparently had not filled with contrast material. On 3 occasions, contrast filled popliteal cysts overlapped the medial posterior portion of the medial meniscus obscuring tears in this region. On one occasion a small peripheral tear did not visualize. The false positive study was the result of diagnosing a small peripheral tear in the posterior horn. This particular patient had an obvious tear of the lateral meniscus for which he was operated. The surgeon opened the knee laterally and stated on inspection that the medial meniscus appeared normal. The study of 122 lateral menisci produced 8 errors (Table 11). There were 7 false negative errors. Five of these were tears of the posterior horn which were obscured by the overlapping popliteus bursa. Two errors occurred when peripheral tears were overlooked. The false positive error resulted when a tear was described in the posterior horn of the lateral meniscus in a patient who had gross tearing of the medial meniscus. Surgery was performed with a medial approach and the lateral meniscus was described as normal. Analysis of the anterior cruciate ligament produced 10 observer errors (Table III). Six were false negative studies and in 5 of these the lateral arthrograms were insufficiently penetrated to see the cruciate ligaments satisfactorily. One case appeared normal in retrospect even though it was torn at operation. The 4 false positive studies were also due to insufficient exposure for proper evaluation of the cruciate ligaments and, in addition, in 3 of these patients, the lateral arthrogram with anterior pressure on the tibia was not made;

TABLE III

ANTERIOR CRUCIATE LIGAMENT

| Roentgen Diagnosis | No. of Patients | Surgical Diagnosis | No. of Patients |
|---|---|---|---|
| Normal anterior cruciate | 106 | Normal anterior cruciate | 100 |
| | | Torn anterior cruciate | 6 |
| | | Torn anterior cruciate | 12 |
| Torn anterior cruciate | 16 | Normal anterior cruciate | 4 |
| Total | 122 | Total | 22 |

hence, a lax appearing anterior cruciate ligament was not put to the test designed to check its integrity. These cases were done early in the series.

As can be surmised from the discussion concerning errors, most are due to technical factors, either insufficient exposure, which can be corrected, or overlapping of contrast medium filled bursae. Nonfilling of transverse meniscal tears can occur and peripheral tears can be missed, especially where the lateral meniscus is not firmly attached to the capsule posteriorly.

A statistical analysis of the results would depend on how one wishes to use the figures. Twenty-five errors in 122 operated knees gives an accuracy figure of 79 per cent. Seven errors in the evaluation of 122 medial menisci gives an accuracy of 94 per cent. Eight errors in the interpretation of 122 lateral menisci gives a 93 per cent figure, while 10 errors in 122 evaluations of the anterior cruciate ligaments yields a 91 per cent accuracy rate.

## DISCUSSION

Arthrography of the knee is not a new procedure. The early attempts utilizing air as a contrast medium had some success. The method, however, did not prove widely acceptable as it had certain limitations. It was not applicable when fluid was present in the joint; therefore, the procedure was not available in the acute stage of most knee injuries. During the late 1940s, positive contrast arthrography became popular with improvement in the opaque materials. It was popularized by Lindblom[8] and is now a well accepted procedure in many centers. Recently, double contrast arthrography has been introduced[4] and is a procedure which is preferred by those who use it regularly. These latter methods offer all the advantages of pneumoarthrography, but without the disadvantages. The presence of a small amount of fluid in the joint at the time of the procedure is no great problem as the opaque media are miscible with the synovial fluid and are able to adequately delineate the internal structures of the knee. Positive contrast



Fig. 20. Double contrast arthrogram with vertical tear of the lateral meniscus (arrow).

arthrography is more accurate than pneumoarthrography in delineating meniscal lesions and has an additional advantage of being able to demonstrate cruciate ligament injuries with accuracy as well as demonstrating ruptures of the medial collateral ligament. The positive contrast arthrography is preferable to the double contrast procedure as it is just as accurate and much easier to perform and much more reproducible. Double contrast arthrography is especially valuable when tears of the lateral meniscus are suspected (Fig. 20).

Arthrography is most useful in the determination of whether or not a serious internal derangement has occurred shortly after an acute injury to the knee. If one is able to accurately determine whether or not a significant internal injury has occurred, proper treatment could be instituted much more promptly. This approach is especially valuable in the athlete in whom a trial of conservative therapy can result in significant atrophy of the quadriceps muscles. Arthrography is also extremely useful in the evaluation of industrial or workman's compensation cases. Often these patients will present with some of the signs and symptoms of internal derangement but one

wonders whether or not such a derangement is actually present. If arthrography shows a lesion, it helps to expedite the case toward proper treatment. Furthermore, this procedure may be used postoperatively in those patients who continue to have pain. Not infrequently, retained posterior horns, especially on the medial side, can be identified. The procedure has likewise proved valuable in the study of masses about the knee joint and, as recently advised by Hench *et al.*,[7] the examination should be considered in cases where the diagnosis of thrombophlebitis is suspected but in which known knee pathology is present. It is only by this method that a dissecting popliteal cyst can be differentiated from a deep vein thrombophlebitis.

In addition to demonstrating the nature of the pathology for the surgeon prior to knee exploration, the procedure can reduce the number of negative explorations. Lindblom[8] states that prior to institution of this procedure 16 per cent of knee explorations at their institution proved negative, while after the institution of this procedure only 1 per cent were negative.

## CONCLUSION

Positive contrast arthrography of the knee is a useful procedure and is quite accurate in determining the presence of significant internal derangement. Errors, when they do occur, are usually false negatives rather than false positives which is a distinct advantage. It would be very unusual for a surgeon to operate on a lesion detected by arthrography which was not present in fact at the time of surgery.

The examination is entirely safe and can be done even in young children without a great deal of difficulty. The youngest patient in this series was 6 years of age.

If this method is utilized on a routine basis by the referring physicians, not only they but also the Radiologist can gain confidence in it. Employed in this way, it is a valuable adjunct to the diagnosis of internal derangements of the knee; in addition, it can demonstrate pathology which is extremely difficult to diagnose otherwise such as dissecting popliteal cysts, hypertrophy of the infra-patellar fat pad or the presence of a diskoid meniscus.

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# ROENTGENOGRAPHIC MANIFESTATIONS OF CHRONIC RENAL DISEASE TREATED BY PERIODIC HEMODIALYSIS*

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PERIODIC hemodialysis has considerably increased the life expectancy of many patients which chronic renal failure. These cases demonstrate particularly gross osseous and soft tissue manifestations of the associated metabolic bone disease previously described by others.[2,22,24] The present study comprises a review of the roentgenographic changes in patients undergoing periodic hemodialysis at the University of Washington Hospital and the Seattle Artificial Kidney Center.

## MATERIAL AND METHODS

Thirty-three cases (Table 1) were reviewed. The duration of hemodialysis ranged up to 75 months (through June 1966). Twenty-one patients were male, 12 female. The ages ranged from 11 to 57 years. Primary diagnoses included chronic glomerulonephritis (22 cases), chronic pyelonephritis (7), polycystic kidney disease (3), and systemic lupus erythematosus (1). The approximate year of disease onset was recorded.

All patients were treated with hemodialysis at least once and generally twice a week for a total weekly hemodialysis time of from 15 to 36 hours. The dialysate calcium concentration varied from 4.6 to 6.6 mg. per cent during the course of this study, most commonly being 5.6 mg. per cent. No phosphate was added to the dialysate. Oral intake of calcium ranged from 300 mg. to 800 mg. per day. Oral vitamin D in amounts varying from 50,000 to 200,000 units was given to those patients who developed symptomatic bone disease or unexplained elevations of serum alkaline phosphatase. Antacids based on aluminum gels (aludrox, basogel) were given orally to combat gross metastatic soft tissue calcification.

In the extensive follow-up of these patients, roentgenograms were frequently obtained. Hands and wrists were routinely examined, occasionally supplemented by views of other body areas. The roentgenograms were studied for phalangeal subperiosteal and ungual tuft resorption, periarticular erosions, hypomineralization, and soft tissue calcification. Despite dual and repeat interpretations there may have been a tendency for the observers to overread questionable and minor alterations, especially regarding periarticular "cysts" and decrease of bone density.

Subjective evaluation of osseous mineralization was supplemented by application of a photodensitometric method. This technique, modified after Doyle,[10] utilizes a narrow aluminum wedge superimposed on soft tissues adjacent to the ulna. Between 1 and 3 "bone density" studies were performed on 30 of the 33 cases.

An attempt was made to quantitate the varying amounts of soft tissue calcification observed in the hands and wrists. Aggregates of calcium were designated as being in relation to digits 1 through 5 (digits being extrapolated proximally into their respective metacarpals and through the wrist). Each calcific complex was measured in 2 right-angle dimensions and its area expressed roughly in cm.$^2$ Aggregates along any 1 "digit line" were combined. The

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TABLE I

ROENTGENOGRAPHIC FINDINGS IN 33 PATIENTS UNDERGOING HEMODIALYSIS

| Patient | Age/Sex | Renal | Onset of Disease (Approximate Year) | Hemo-dialysis Duration (months) | Sub-periosteal Resorption | Ungual Tuft Resorption | Peri-articular Erosion | Hypomineralization | | Soft Tissue Calci-fication |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Subjective | Photoden-sitometric | |
| H.G. | 28 M | CGN | 1956 | 75 | No | No | Yes | No | Not done | Yes |
| C.S. | 46 M | CGN | 1938 | 75 | No | No | Yes | Yes | No | Yes |
| R.H. | 35 M | CGN | 1959 | 74 | Yes | No | Yes | Yes | Yes | Yes |
| F.S. | 44 M | CGN | 1930 | 54 | No | No | No | Yes | Yes | No |
| J.A. | 32 M | PC | 1959 | 53 | Yes | Yes | Yes | Yes | Yes | Yes |
| J.My. | 41 M | CGN | 1946 | 53 | No | No | No | Yes | Yes | No |
| P.F. | 38 M | CGN | 1952 | 52 | No | No | No | Yes | Yes | Yes |
| D.S. | 17 F | CGN | 1958 | 44 | Yes | Yes | No | Yes | Yes | No |
| K.H. | 33 M | CGN | 1953 | 44 | No | No | No | Yes | Yes | Yes |
| J.D. | 30 F | CPN | 1956 | 43 | Yes | Yes | Yes | Yes | Yes | No |
| D.B. | 45 F | CGN | 1942 | 42 | No | No | No | No | No | No |
| J.Mi. | 45 M | CGN | 1953 | 40 | No | No | No | Yes | Yes | Yes |
| C.L. | 42 M | CPN | 1944 | 30 | No | No | No | Yes | Yes | No |
| C.H. | 18 F | SLE | 1960 | 27 | No | No | Yes | Yes | Yes | Yes |
| L.O. | 46 F | CPN | 1927 | 26 | No | No | No | Yes | Yes | No |
| M.O. | 33 F | CPN | 1963 | 26 | No | No | No | Yes | Not done | No |
| C.Y. | 56 M | CGN | 1959 | 25 | No | No | No | Yes | Not done | No |
| H.S. | 10 F | CGN | 1960 | 25 | Yes | No | No | Yes | Yes | No |
| W.M. | 34 M | CGN | 1943 | 25 | No | No | No | Yes | Yes | Yes |
| W.D. | 21 M | CGN | 1950 | 23 | No | No | No | Yes | No | Yes |
| M.L. | 30 F | CGN | 1943 | 19 | No | No | Yes | Yes | No | Yes |
| F.W. | 43 F | CPN | 1935 | 17 | No | No | Yes | Yes | Yes | Yes |
| S.A. | 57 F | PC | 1938 | 16 | Yes | No | No | Yes | Yes | Yes |
| D.D. | 44 M | CGN | 1942 | 16 | No | No | No | No | No | Yes |
| C.C. | 26 M | CGN | 1962 | 13 | No | No | No | Yes | No | No |
| M.M. | 50 F | CGN | 1952 | 12 | Yes | No | Yes | Yes | Yes | No |
| F.T. | 50 M | CPN | 1955 | 12 | No | No | No | Yes | No | No |
| B.F. | 33 M | CPN | 1948 | 10 | Yes | Yes | Yes | Yes | Yes | Yes |
| I.B. | 35 M | CGN | 1954 | 8 | No | No | No | No | No | No |
| G.G. | 28 M | CGN | 1958 | 8 | No | No | No | No | No | Yes |
| C.B. | 52 M | CGN | 1953 | 6 | No | No | No | Yes | No | No |
| E.M. | 52 M | CGN | 1961 | 5 | No | No | No | Yes | No | No |
| B.M. | 35 F | PC | 1954 | 3 | Yes | Yes | Yes | Yes | Yes | Yes |

CGN = chronic glomerulonephritis; CPN = chronic pyelonephritis; SLE = systemic lupus erythematosus; PC = polycystic disease.

gross quantitative estimates were recorded in graph form (example, Fig. 6), indicating the changing patterns and amounts of calcification over intervals.

Finally, the roentgenographic appearances were correlated with certain laboratory and clinical information.

## RESULTS

The roentgenographic findings are summarized in Table 1. Presence or absence of a specific abnormality is indicated by "yes" or "no." It should be emphasized that the former includes both transient and permanent changes of varying degrees. Transient changes were rare and could be related to alterations in management, of which several case examples are presented below.

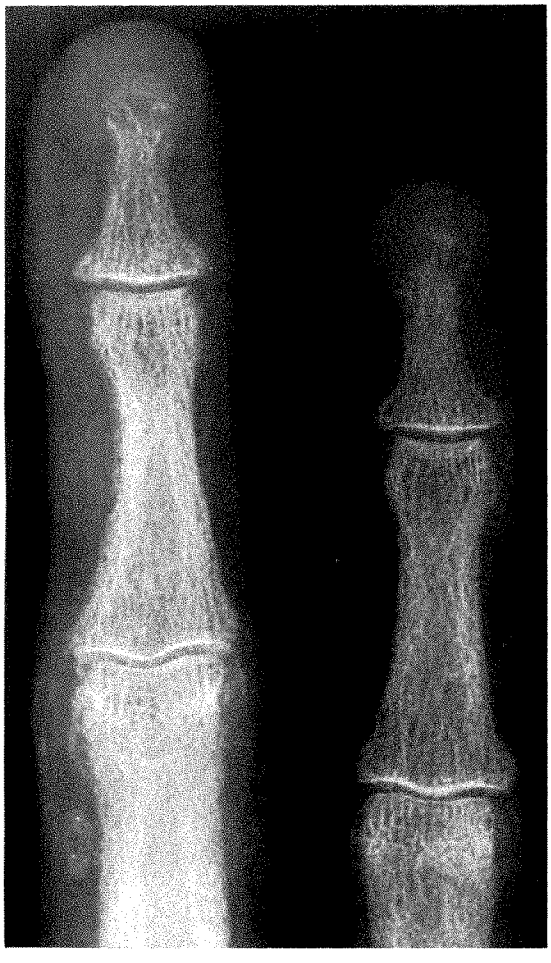


FIG. 1. Patient B.M. Renal failure due to polycystic disease. Right middle and ring fingers. Gross subperiosteal and ungual tuft resorption. Soft tissue calcification.



FIG. 2. Patient C.S. Renal failure secondary to chronic glomerulonephritis. Proximal interphalangeal joint of right ring finger. Periarticular erosion. Soft tissue calcification with radiolucent "septa."

Subperiosteal resorption (Fig. 1) was unequivocally present in 9 patients (27 per cent). This sign could have possibly been recognized in a few others if it had not been for their severe generalized decrease in bone density. Five of these 9 patients also had resorption of the terminal phalangeal tufts, resulting in shortened phalanges. Ungual tuft resorption was not observed in the absence of subperiosteal resorption. A few cases with extreme hypomineralization had shortened terminal phalanges secondary to shaft resorption and apposition of the remaining bone.

Periarticular erosions of "cysts" (Fig. 2) were noted in 11 patients (33 per cent). Whereas 6 had concomitant phalangeal resorption, the remaining 5 did not. Three of the latter appeared to be normally mineralized. These probably represented

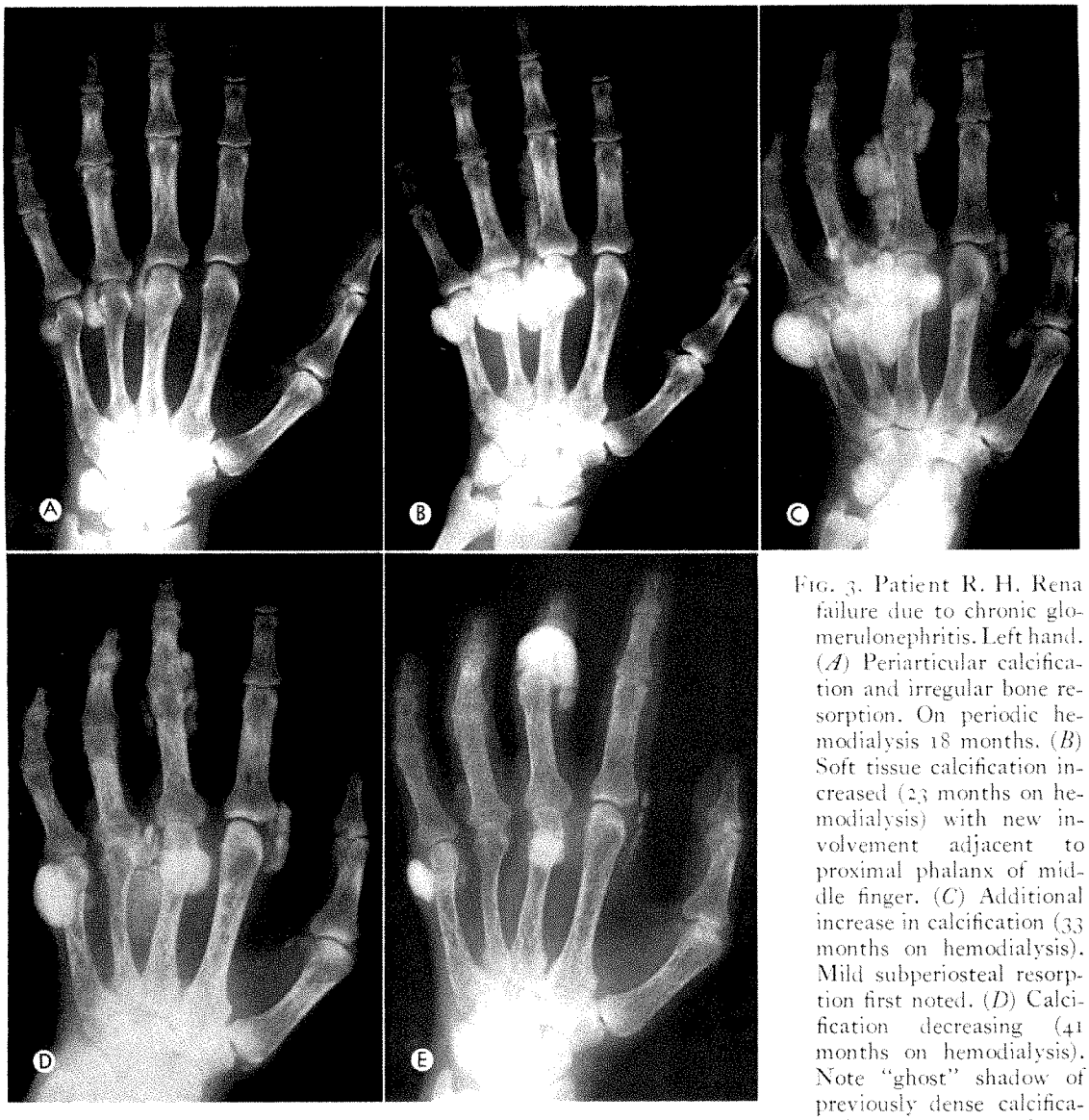



FIG. 3. Patient R. H. Renal failure due to chronic glomerulonephritis. Left hand. (*A*) Periarticular calcification and irregular bone resorption. On periodic hemodialysis 18 months. (*B*) Soft tissue calcification increased (23 months on hemodialysis) with new involvement adjacent to proximal phalanx of middle finger. (*C*) Additional increase in calcification (33 months on hemodialysis). Mild subperiosteal resorption first noted. (*D*) Calcification decreasing (41 months on hemodialysis). Note "ghost" shadow of previously dense calcification between third and fourth metacarpals. (*E*) Further over-all decrease of calcification (68 months on hemodialysis), although increased in the middle finger distally.

anatomic variations or minor osteoarthritic changes. Erosions were sometimes located adjacent to aggregates of soft tissue calcification, but this association was too infrequent to be regarded as significant.

The most frequently observed abnormality was generalized hypomineralization, which presented in a spectrum ranging from slight uniform decrease of density to gross irregular resorption with erosion of the endosteum. This diagnosis was made subjectively in 28 patients (85 per cent) and in 19 of the 30 cases (63 per cent) studied by the photodensitometric method. Increased bone density was not noted.

There was evidence that the observers tended to overread hypomineralization. Seven of the 28 cases with subjectively decreased density had one or more normal determinations by the more objective technique. Furthermore, 3 of these had no other sign of bone resorption and in 4 it was

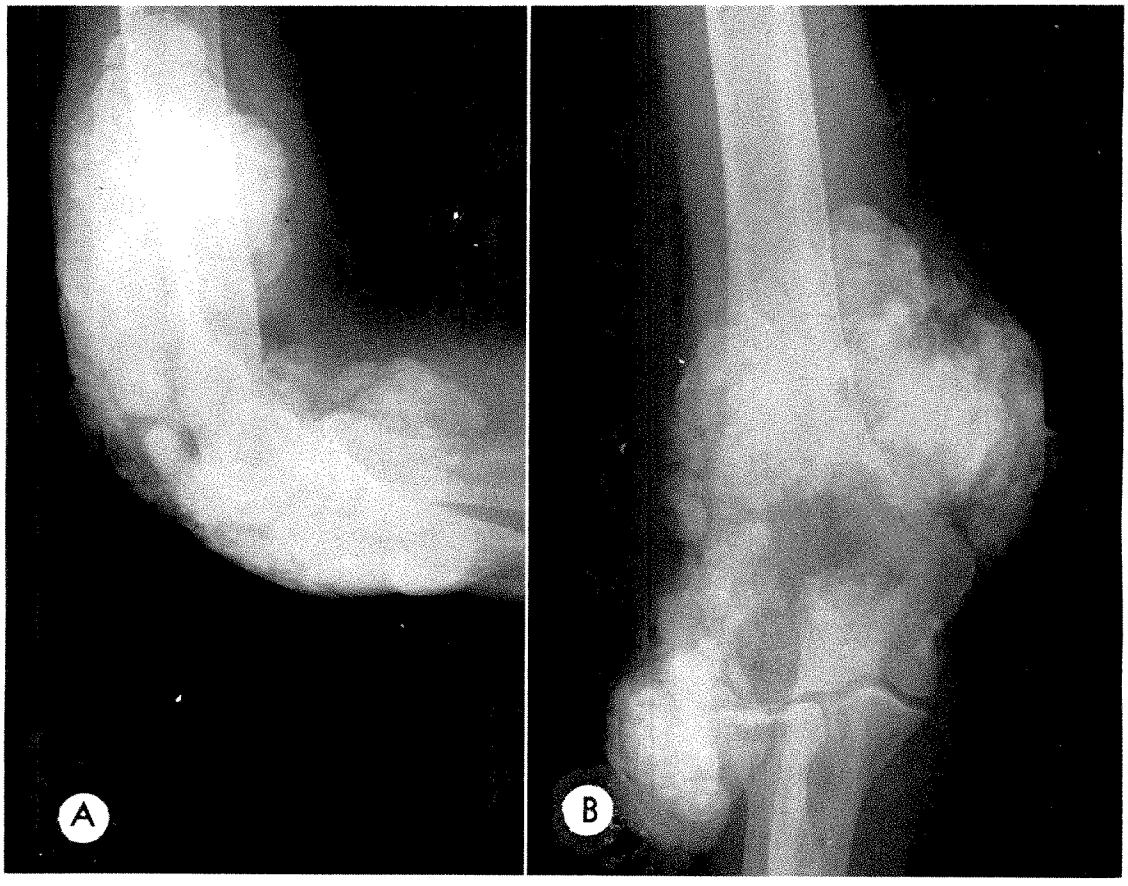



FIG. 4. Patient B. M. (*A* and *B*) Right elbow. Very extensive soft tissue calcification.

the only roentgenographic abnormality. Conversely, there was a good correlation between the photodensitometric bone density determination and other roentgenographic observations.

The true incidence of hypomineralization in this series was, therefore, closer to 6 out of 10. It was more frequent in those patients who had been on periodic hemodialysis for longer intervals. Seventeen patients had multiple determinations and, whereas 8 were noted to drop below previously normal levels, none showed improvement.

Soft tissue calcification (Fig. 3, *A–E*) was noted in the hands and wrists of 17 patients (52 per cent). The aggregates were typically asymmetric, rather random in location, and greatly variable in extent and texture over intervals. There may have been a slight tendency for concentration adjacent to the bone ends more so than opposite the shafts; however, there was no unusual accumulation across or within joints. Growing aggregates were usually quite nodular and contained lucent "septa." When resolving, they developed a more uniform and less dense texture. Aggregates were frequently seen to be waxing and waning concurrently on follow-up examination, even on opposite sides of the same bone.

There was no clear correlation between soft tissue calcification and the other roentgenographic findings. For instance, 5 cases with calcifications had normal bone density determinations by the photodensitometric method. Calcification was the only roentgenographic abnormality in 2 patients.

Roentgenograms of other body areas

yielded some additional signs not included in Table 1 but which are associated with metabolic bone disease. Seven cases had examinations of the spine and 5 of these showed gross hypomineralization and typical biconcave deformaties. Four patients had skull examinations of which 3 demonstrated a thickened and granular calvarium. Vascular calcification was seen in the feet in 4 of 9 cases examined, and in 1 of these it was extensive in the hands. The latter was a particularly gross example of hyperparathyroidism. Seven patients had abdominal roentgenograms and aortic calcification was noted in 3 of these. "Nephrocalcinosis" was not seen, but calcification was noted in the large renal cysts of 1 case (B.M.). Calcification frequently occurred in large aggregates around the shoulder, elbow, hip, and knee joints (Fig. 4, *A* and *B*; and 5, *A* and *B*).

Changing roentgenographic manifestations are illustrated by the following examples. Patient R.H. demonstrated rather marked variations in the degree of soft tissue calcification during over 5 years of hemodialysis (Fig. 3, *A–E*; and 6). Calcification increased steadily during the first 3 years and subsequently showed an over-all decrease in extent, with a transient resurgence. The initial decrease coincided with a period during which the blood phosphate level was lower than had previously been observed. A further decrease followed another interval with lowered phosphate values.

Patient J.A. had comparatively little soft tissue calcification during periodic hemodialysis (Fig. 7), although he initially demonstrated marked changes of secondary hyperparathyroidism. His phosphate levels were noted to be consistently lower than those of R.H.

Particularly gross changes of secondary hyperparathyroidism and soft tissue calcification were demonstrated in patient B.M. (Fig. 1; 4, *A* and *B*; 8*A*; and 9*A*). Disability from bone pain and discomfort at the sites of calcific aggregates was so severe that subtotal parathyroidectomy was performed.

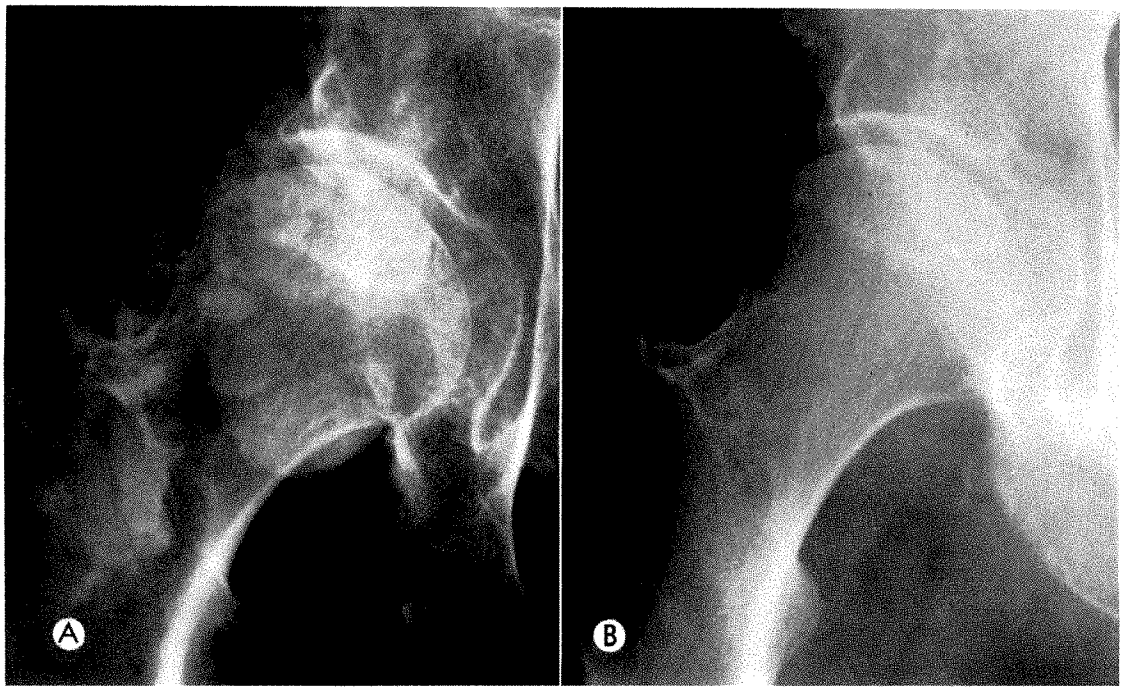



FIG. 5. Patient C. S. Right hip. (*A*) Extensive soft tissue calcification. On hemodialysis 30 months. (*B*) Calcification almost cleared (58 months on hemodialysis).

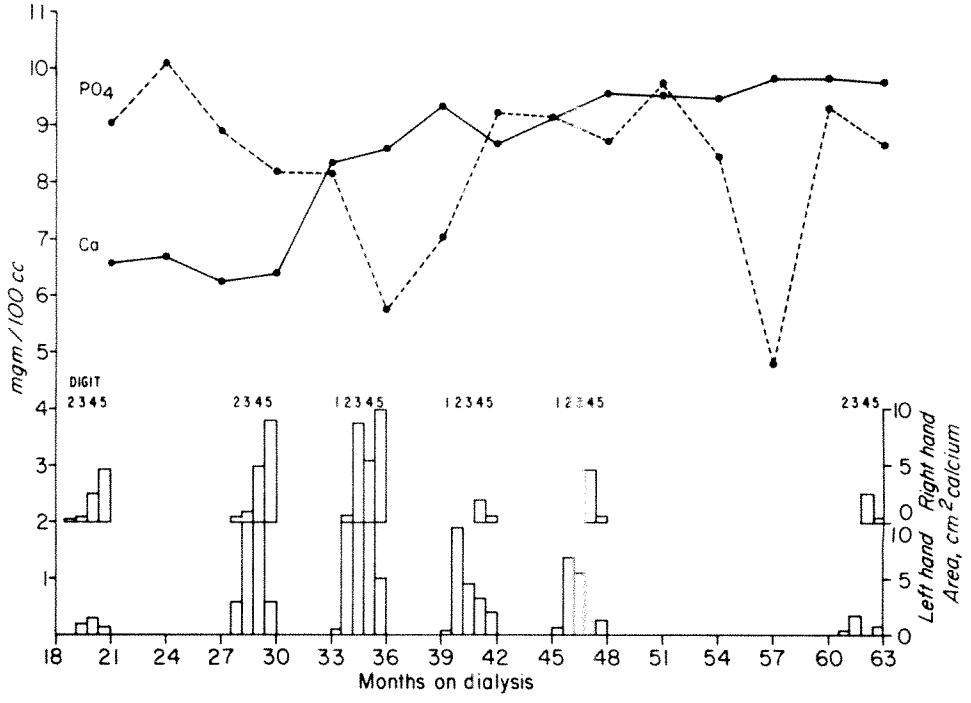



FIG. 6. Patient R. H. Average (three month intervals) serum calcium and phosphate levels during periodic hemodialysis. Block diagrams approximate the extent of soft tissue calcification in the hands (see text). Note the marked fluctuations of serum phosphate.

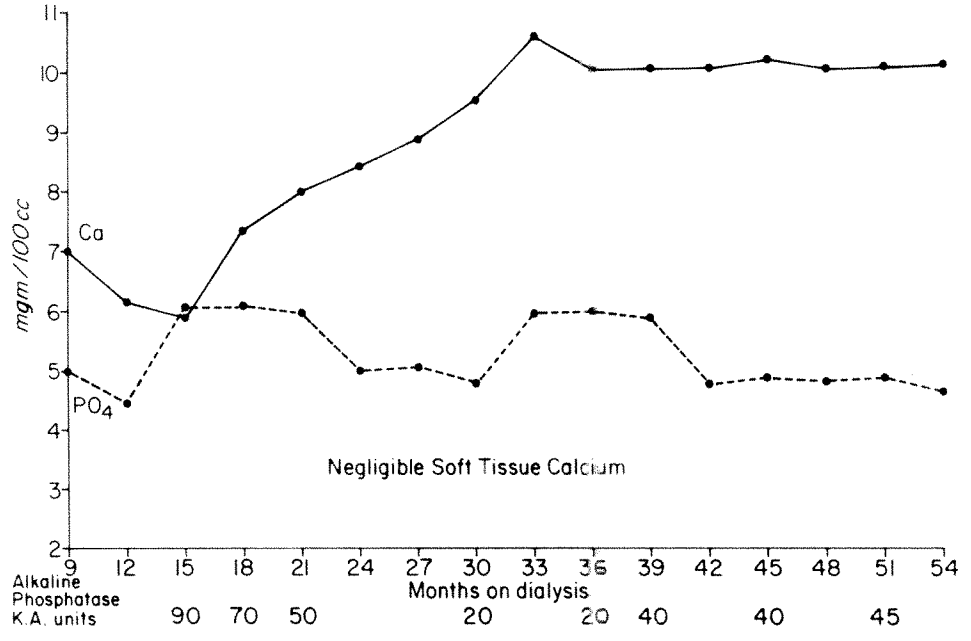



FIG. 7. Patient J. A. Renal failure due to polycystic disease. Serum calcium and phosphate levels during hemodialysis. Although he had advanced bone changes of secondary hyperparathyroidism, soft tissue calcification was minimal. Serum phosphate was kept consistently low through the use of phosphate binders.

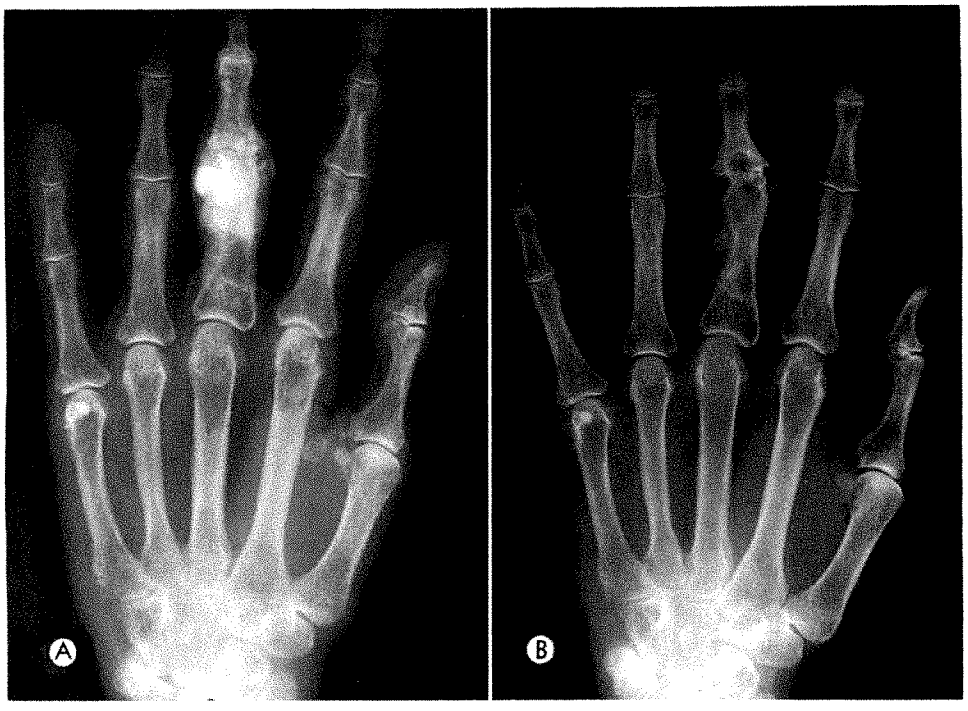



FIG. 8. Patient B.M. (*A*) Left hand. Subperiosteal and ungual tuft resorption. Particularly severe bone changes and soft tissue calcification of the middle finger. (*B*) Six months later and 3 months following subtotal parathyroidectomy. Re-ossification of previous areas of resorption. Residual deformity of middle finger. Calcification greatly reduced.

Subsequent changes in her blood calcium and phosphate levels are shown in Figure 10. Improvement of subperiosteal resorption and soft tissue calcification followed (Fig. 8*B*; and 9*B*).

## DISCUSSION

Periodic hemodialysis, although life-saving, was not necessarily associated with a favorable effect on the bone resorption, hypomineralization, and soft tissue calcification so frequently noted in these patients.

Subperiosteal resorption persisted throughout hemodialysis in 6 cases, ranging up to 44 months. Patient R.H. first developed this sign of secondary hyperparathyroidism after being hemodialyzed 30 months. Only 3 (R.H., J.A., and B.M.) have re-ossification of their subperiosteal resorption. In each case, a supplemental form of therapy was employed. R.H. responded to intermittent use of oral phosphate binders, and, although his patchy hypomineralization persisted, the resorption was no longer recognizable. J.A. was treated with phosphate binders and large doses of vitamin D. His serum alkaline phosphatase fell, and more satisfactory calcium and phosphate levels were maintained. The resorption gradually healed. In the case of B.M., disappearance of this sign followed subtotal parathyroidectomy.

Osseous hypomineralization, a well known manifestation of renal osteodystrophy, was one of the two most frequent roentgenographic abnormalities in this series. Over half of the cases with decreased bone density had no subperiosteal resorption, whereas the opposite association was always seen. Hyperparathyroidism may show lowered density without subperiosteal resorption.[27] Also, the irregular "patchy" hypomineralization often associated with parathyroid hyperactivity was observed in a few of these patients.

There was, however, no instance of improvement of bone mineralization, even following subtotal parathyroidectomy for obvious secondary hyperparathyroidism. Neither did it respond to large doses of vitamin D. And, it is of considerable interest that no Looser's zones, the roentgenographic hallmark of osteomalacia,[28] were noted. The exact nature of the hypomineralization remains a matter of some speculation.

Similarity of the joint lesions of hyperparathyroidism and rheumatoid arthritis has been previously described.[7,24,29] The main roentgenographic changes are decreased bone density, cyst formation, and loss of articular cartilage. Bywaters[7] stated that in some cases it was difficult to exclude

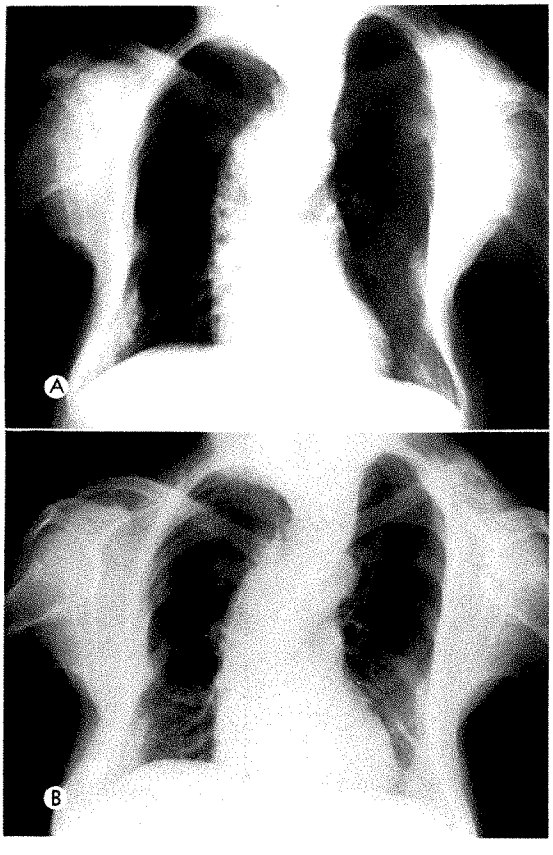



FIG. 9. Patient B.M. (*A*) Chest and shoulders immediately before subtotal parathyroidectomy. Gross soft tissue calcification. Partial resorption of clavicles. (*B*) Three months later. Disappearance of calcification and re-ossification of clavicles.

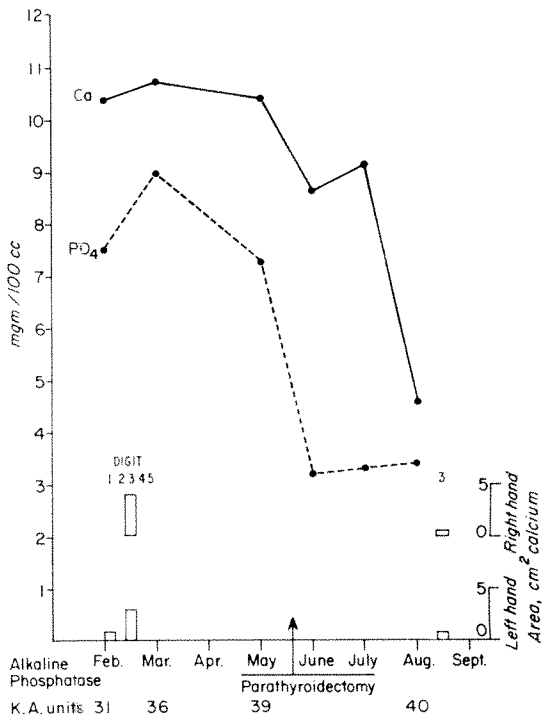



FIG. 10. Patient B.M. Serum calcium and phosphate levels and approximate extent of hand soft tissue calcification. Note decrease of all three following subtotal parathyroidectomy.

a dual diagnosis. None of his patients, however, had laboratory evidence of the latter. Zvaifler *et al.*[29] noted these lesions in 3 patients with proven parathyroid adenomata who suffered pain like that of rheumatoid arthritis. Pugh[24] considered the joint changes of hyperparathyroidism to be articular manifestations of the more commonly described subperiosteal resorption.

The most advanced articular erosions in the present study were seen in those cases exhibiting this sign of secondary hyperparathyroidism. In some of these, marked loss of cartilage was noted and clinically the patients suffered joint pain. Thus, periarticular erosions in hyperparathyroidism may, possibly as a result of altered joint mechanics, progress to a gross arthritis.

Caner and co-workers[8,9] described attacks of arthralgia in several patients on periodic hemodialysis. Three of their cases,

(C.S., R.H., and J.A.) are included in the present series. The attacks were clinically typical of gout in that they were of acute onset, showed rapidly increasing severity, and appeared to respond to treatment with colchicine. In 1 patient, attacks occured following artificial elevation of the blood uric acid level through addition of uric acid to the dialysate. These workers considered the soft tissue aggregates to be gouty urate deposits, but repeated aspirations failed to produce urates. An autopsy specimen revealed calcium phosphate.

Murray *et al.*[20] postulated that metastatic calcification within joints may initiate the arthritis of uremia. This suggestion has some support in the work of McCarthy *et al.*,[18] who found calcium phosphate crystals in the synovial fluid of arthritic patients.

Attacks of gouty-type arthritis in hyperuricemic states not due to a primary defect in purine metabolism have been documented.[1,13,21,26] Others have suggested that such attacks are rare in chronic renal disease.[22,25] Sorensen[26] recommended the term "secondary gout" for cases resulting from defective excretion of urates. He states, "The reason why overt gout does not complicate this condition (uremia) as often as one might expect may be that many patients do not outlive the initial phase of asymptomatic hyperuricemia." Roentgenographic joint changes are relatively late manifestations of gout and would not necessarily be expected in cases of comparatively short duration. With further longevity, these typical features may become manifest in patients on periodic hemodialysis.

Other than hypomineralization, the most frequent roentgenographic abnormality in this series was soft tissue calcification. It has been frequently described in association with chronic renal disease.[3,5,17,19,22] Caner and Decker[3] studied these nodules and found them to be principally hydroxyapatite. Connective tissue bands separated the aggregates into smaller amorphous portions, hence the "septa" seen on roentgenograms.

Soft tissue calcification appears to be related to serum calcium and phosphate levels. Herbert *et al.*[15] postulated that under normal conditions the blood levels of these ions are near their "saturation" point. The initial effect of chronic renal disease is to bring about phosphate retention with a reciprocal lowering of serum calcium. However, this causes parathyroid stimulation and mobilization of bone calcium, elevating serum calcium to its original level or even higher. As the original renal failure does not allow a reciprocal fall in phosphate, "oversaturation" is reached and metastatic calcification occurs.[6]

In primary hyperparathyroidism, conversely, soft tissue calcification is not usually a prominent early feature,[15,27] because elevation of serum calcium can be compensated for by dropping the phosphate level. However, primary hyperparathyroidism can itself result in chronic renal disease and gradually assume a clinical picture virtually indistinguishable from some of the cases in this study.

The relationship between serum phosphate level and soft tissue calcification is illustrated by patients R.H. and J.A. (Fig. 6; and 7). The former had progressive increase of calcification until oral phosphate binders reduced his phosphate intake and its blood level. Metastatic calcification immediately decreased. However, subsequently he took his medication only intermittently and there were rather marked fluctuations of the phosphate level. This was associated with a slight exacerbation and another remission of his soft tissue calcification.

Through the judicious use of these binders, J.A. maintained a consistently lower phosphate level than R.H. And, despite the fact that both had comparable serum calcium values and evidence of secondary hyperparathyroidism, the former had negligible metastatic calcification during over 4 years on hemodialysis.

SUMMARY

The roentgenographic abnormalities in 33 chronic renal disease patients undergoing periodic hemodialysis are presented. The principal findings are subperiosteal resorption, osseous hypomineralization, and metastatic soft tissue calcification. These are described and discussed in relation to pertinent clinical, laboratory, and reference information.

Minor joint changes are also observed, and it is speculated whether these are additional manifestations of metabolic bone disease or might represent early gout.

Study of illustrative case examples suggests that metastatic calcification is related to elevation of the serum phosphate level.

Hemodialysis, as practiced at this Medical Center, appears to be relatively ineffective in combating these associated changes on chronic renal disease. The results obtained with supplementary forms of therapy are described.

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# THE VARIOUS ROENTGEN APPEARANCES OF PULMONARY HYPERTROPHIC OSTEOARTHROPATHY*

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THE description of clubbed fingers occurring in a patient with emphysema was first reported by Hippocrates in the Fifth Century B.C. In 1889 Bamberger, and in 1890 Pierre Marie independently described the entity of hypertrophic osteoarthropathy. Lock in 1915 combined this entity with clubbing.[2] The disease picture, as now conceived, consists of a triad of periosteal new bone formation, clubbing of the fingers and synovitis of the joints, associated with a multitude of intrathoracic diseases.

## ETIOLOGY

There have been two theories proposed to account for this phenomenon, the humoral and the neurogenic.[1,2,5] Pierre Marie in 1890 suggested that a toxin originating from the disease process in the lungs affects the target tissues. Current investigation, however, favors the neurogenic concept, with the afferent pathway via the vagus nerve.

The anatomic changes in limbs of dogs with osteoarthropathy are an overgrowth of vascular connective tissue, followed by subperiosteal new bone formation. Blood flow is increased in the affected areas. A major portion of this blood flow is directed to newly formed tissue.

Reduction of blood flow to the affected areas is lessened by vagotomy. In man, osteoarthropathy regresses following removal of the primary lung lesion, exploratory thoracotomy or unilateral vagotomy.

Cross circulation experiments in dogs with pulmonary hypertrophic osteoarthropathy and normal dogs failed to cause development of changes in the normal dogs. This evidence tends to negate the humoral theory.

The afferent pathway is thought to be via the vagus. The efferent pathway is still unknown. An increase in estrogen excretion in pulmonary hypertrophic osteoarthropathy has been reported,[3] but the significance of this has not yet been determined.

## CLINICOPATHOLOGIC FINDINGS

Clubbing of the fingers can be associated with many different intrathoracic and extrathoracic conditions. Clubbing due to intrathoracic neoplasms is somewhat different than that due to chronic pulmonary insufficiency. The former is often painful, appears acute with a red rim surrounding the base of the nail, and develops rapidly. There is a definite increase in blood flow to the affected areas.[1] The full blown picture of pulmonary hypertrophic osteoarthropathy includes periosteal new bone formation, which is painful, and arthritis which shows some similarity to the rheumatoid type. This classic triad of clubbing, periosteitis and arthralgia may be entirely present or be manifest in various combinations and in various orders of appearance.[7]

On pathologic examination, the tubular bones show periosteal new bone apposition and cortical thickening most marked at the diaphysis. The epicondyles and tendon insertions are not involved.[7]

The frequency of involvement in decreasing order is: the radius and ulna; the

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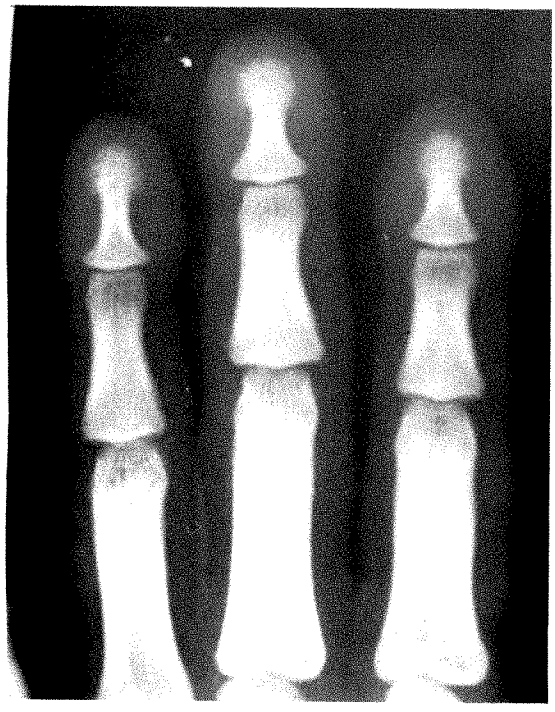


FIG. 1. A 35 year old Negro female with metastases from carcinoma of the parotid gland to lungs, of 3 years' duration. Soft tissue swelling of the distal phalanges is noted. There were no bone changes in the hands.

tibia and fibula; the humerus and femur; the metacarpal and metatarsals; and the proximal and middle phalanges.

The terminal phalanges and the bones of the trunk are very rarely involved.[8]

Microscopically, the osteoperiostitic deposit is at first sharply demarcated from the original cortex and is composed of meshing trabecular primitive bone. As the deposit becomes thicker, the deeper part undergoes lamellar reconstruction and can merge with the cortex. There is no endosteal deposition of bone.

In the joints there are inflammatory changes in the synovial membranes and sometimes intra-articular effusion. The articular cartilages may be modified by connective tissue and subcapsular vascularization.[7]

## ROENTGEN FINDINGS

Soft tissue signs are clubbing of the fingers with soft tissue prominence at the terminal tufts. (Fig. 1 and 2). There is no evidence of bone changes, particularly of the spade like configuration of the terminal digits as seen in acromegaly, nor is there periosteal elevation involving the distal terminal tufts. Periosteal changes involve the diaphyses of the long bones and tubular bones (Fig. 3). The new bone formation tapers off as the end of the shaft is approached and at the ends there is no evidence of enlargement.

Various types of periosteal elevation are seen: (1) Simple elevation of the periosteum wherein a radiolucent area is seen between the periosteum and the cortex; (2) laminated or "onion-skin" appearance of elevated periosteum with smooth layers of new bone formation (Fig. 4); (3) irregular areas of periosteal elevation seen sporadically (Fig. 5); (4) irregular solid areas of periosteal cloaking with a wavy contour

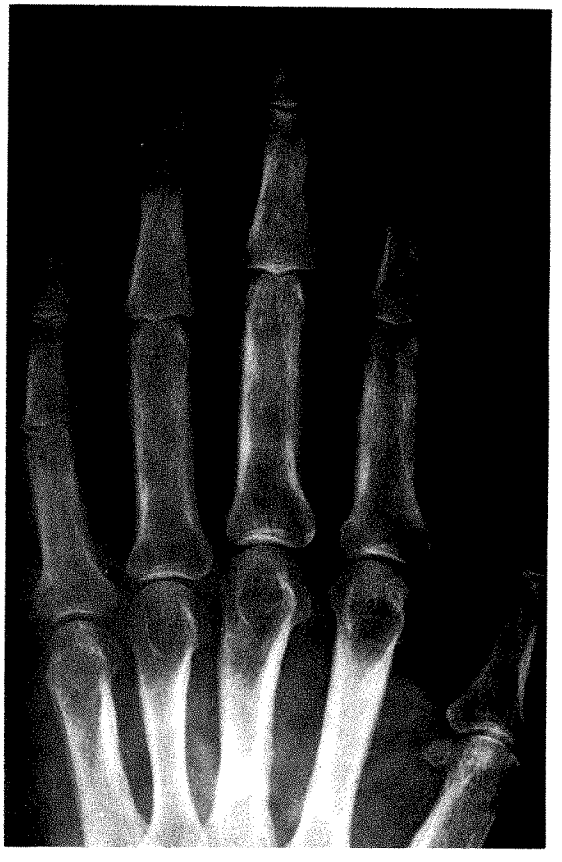


FIG. 2. A 50 year old Negro female with carcinoma of the lung with massive atelectasis. There is smooth periosteal elevation of the metacarpals and proximal and mid phalanges. The distal phalanges are spared.

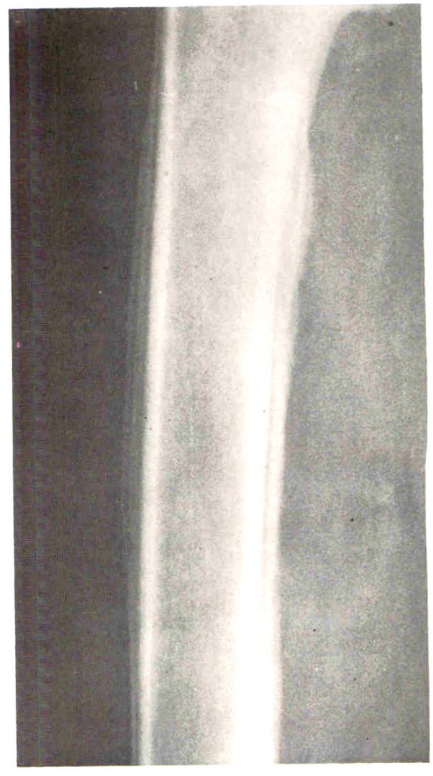


Fig. 4. A 14 year old Negro male with pulmonary metastases from osteogenic sarcoma of the right knee. There is a lamellated "onion skin" type of periosteal new bone formation.

(Fig. 7); and (5) cortical thickening where the periosteal elevation has merged with the cortex and no radiolucent line is evident (Fig. 8 and 9).

No recognizable roentgen manifestations of joint involvement were observed.

## DISCUSSION

The awareness of this entity is of great importance as often it can be the first manifestation of a pulmonary or pleuritic lesion.



Fig. 3. A 46 year old Negro male with bronchogenic carcinoma. There is smooth periosteal elevation of the shafts of the radius and ulna. The bone ends are spared.



Fig. 5. A 71 year old white male with adenocarcinoma of the lung. Irregular "bursts" of periosteal new bone formation are shown.

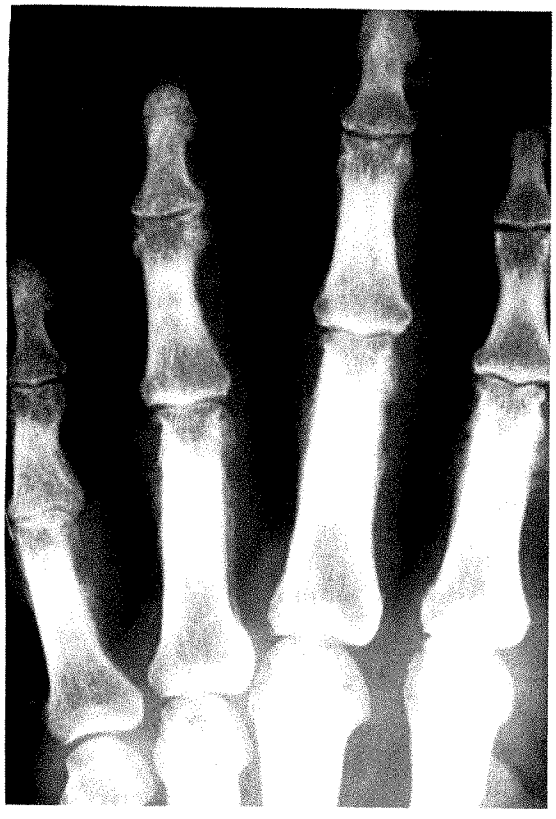
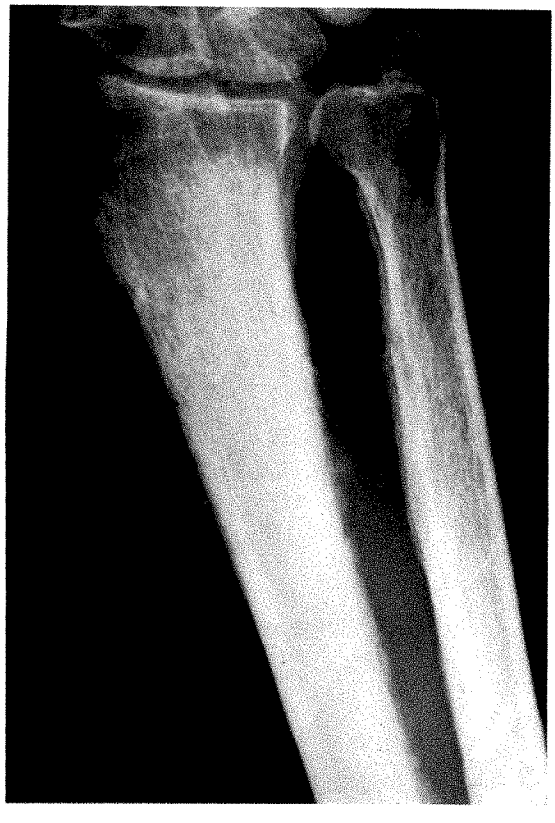


FIG. 6. A 66 year old Negro male with histoplasmosis. "Lacy" periosteal elevation involving the shafts of the proximal and mid phalanges is seen. The distal phalanges are spared.

There are many causes of clubbing of the fingers including acromegaly, chronic heart disease, long-standing chronic pyelonephritis, liver and biliary disease. This entity is different from pulmonary hypertrophic osteoarthropathy; periostitis is very rarely if ever associated.[6]

Other conditions can cause clubbing of the terminal digits and periosteal proliferation; *e.g.*, thyroid acropachy and pachy-



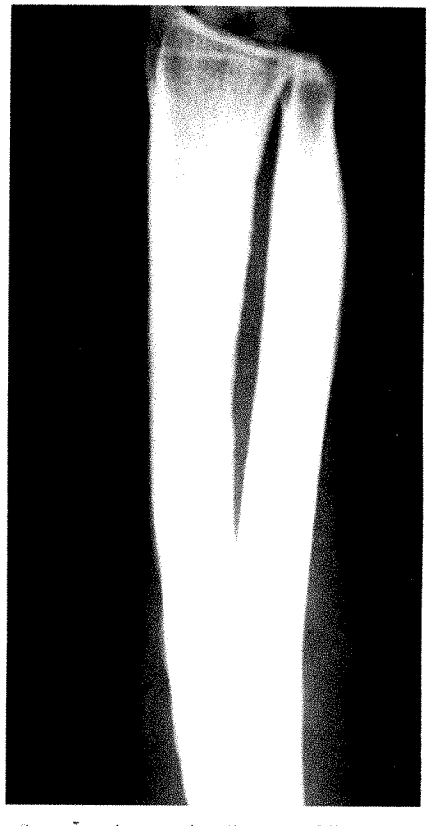


FIG. 8. Same patient as in Figure 1. There is cortical thickening of the shafts of the radius and ulna. The bone ends are not involved.

FIG. 7. Same patient as in Figure 5. There is periosteal cloaking with an irregular contour of the radius and ulna. The bone ends are not involved.

dermoperiostitis. There are many other conditions that can cause generalized periosteal elevation including leukemia, hypervitaminosis A, congenital lues, rubella, sickle cell dactylitis and osteitis, osteomyelitis, Gaucher's disease, scurvy, fibrous dysplasia and hyperphosphatasia.

The two conditions most likely to be confused on viewing roentgenograms of the hands and extremities are pachydermoperiostitis and thyroid acropachy. Pachydermoperiostitis is a familial condition which begins at puberty. It is self-limiting and progresses for several years. The hands, forearms and the tibia are chiefly affected. There is nontender cortical thickening[6] and a thickening of the skin, especially of the forearms and legs and sometimes of the forehead.[7] Thyroid acropachy usually results several years after ablation of the thyroid gland either by surgical or radioactive means. There is a triad of exophthalmos, pretibial myxedema and clubbing of the digits.[7] The periosteal proliferation is chiefly at the radial aspects of the involved bones. It can be irregular with small radiolucent areas or a spiculated appearance.[4] The clinical pictures are not likely to be confused with pulmonary hypertrophic osteoarthropathy.

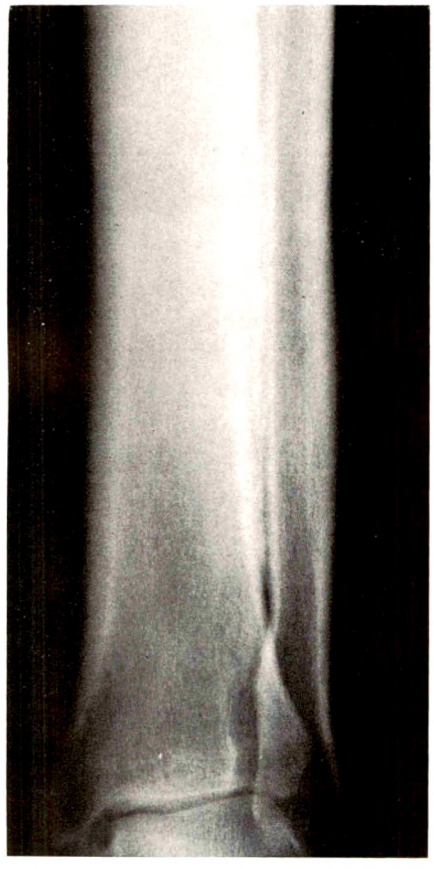


Fig. 9. Same patient as in Figure 1. There is cortical thickening of the shafts of the tibia and fibula. The bone ends are spared. There is no endosteal proliferation.


## SUMMARY

The clinical, pathologic, physiologic, and roentgenologic aspects of pulmonary hypertrophic osteoarthropathy are presented and discussed. The various roentgen appearances of the types of periosteal proliferation are illustrated. The differential diagnosis is considered.



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# THE ROLE OF ASPIRATION BIOPSY OF FOCAL LESIONS IN LUNG AND BONE BY SIMPLE NEEDLE AND FLUOROSCOPY*

*By* KEITH RABINOV, M.D., HARVEY GOLDMAN, M.D., HILDE ROSBASH, *and* MORRIS SIMON, M.D.

THE method of percutaneous aspiration biopsy of focal lesions in the lung and other organs has been known for many years.[3,4,5,11] Major improvements since the original description include use of smaller needles, the application of fluoroscopy and preparation of the specimens for cytologic staining. Although extensively practiced in a few institutions where their value and safety have been demonstrated,[10,11] these procedures have not received widespread usage. The reasons for this may be related to fear of possible complications and to doubt that specimens suitable for diagnosis will be obtained. Interest in these procedures has been predominantly surgical and relatively few reports were to be found in the radiologic literature[2,11] until recently when the method as applied to lung lesions has been refined and popularized by Nordenström.[5,12] However, because of recently acquired skills with other minor surgical techniques and familiarity with fluoroscopic technique, radiologists are now in a favorable position to perform these procedures.

Our experience with percutaneous aspiration biopsy of focal lesions in lung and bone over the past several years, stimulated in the first instance by the work of Nordenström, is described below.

## PERCUTANEOUS ASPIRATION BIOPSY OF LUNG LESIONS

### MATERIALS AND METHOD

Patients with focal lesions in the chest in whom sputum cytology was negative or unobtainable were accepted for biopsy. Many of the patients had also had negative bronchoscopies and scalene lymph node biopsies as well as normal gastrointestinal and urographic studies. Two patients also had nondiagnostic open biopsies.

Contraindications to biopsy included systemic bleeding tendency, suspected vascular lesion, previous pneumonectomy or very severe pulmonary emphysema.

For localizing the lesion during the biopsy procedure, television fluoroscopy was employed in the vertical plane supplemented with filming in the horizontal plane. The biopsy needles used were usually ordinary straight needles size No. 18 (O.D.–.049 inch) or 19 (O.D.–.042 inch) with long bevel. It is now felt that in the occasional patient in whom a particularly firm lesion is encountered by the needle and a satisfactory specimen cannot be aspirated, a cutting type needle should probably be used,[9] since a satisfactory biopsy may otherwise not be obtained. Other materials included a 30 cc. syringe moistened with heparin for aspirating the specimen, a local anesthetic, skin antiseptic, and sterile drapes and gauze. Premedication was rarely necessary.

The biopsy technique used has been patterned after that described by Dahlgren and Nordenström.[5] The procedure was performed with the patient recumbent. An approach to the lesion was selected so that there was no obstruction by bone structures, heart or great vessels, and a minimum of lung tissue was traversed. Neurovascular structures on the inferior margin of a rib were avoided. We have had the best results when the patient was positioned with the lesion uppermost and the needle was inserted vertically. A small metallic marker was placed directly over the lesion and vertical and horizontal roentgenograms were taken so that the precise depth

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to which the needle was to be placed could be measured.

After antiseptic preparation, the skin and pleura over the lesion were infiltrated with a local anesthetic agent. The biopsy needle was then advanced to the predetermined depth directly into the lesion with fluoroscopic surveillance, while the patient maintained quiet respiratory movements. The position of the tip of the needle in the lesion was confirmed by anteroposterior and lateral roentgenograms (Fig. 1, *A* and *B*). The mandrin was removed from the needle which was then gently rotated a few times

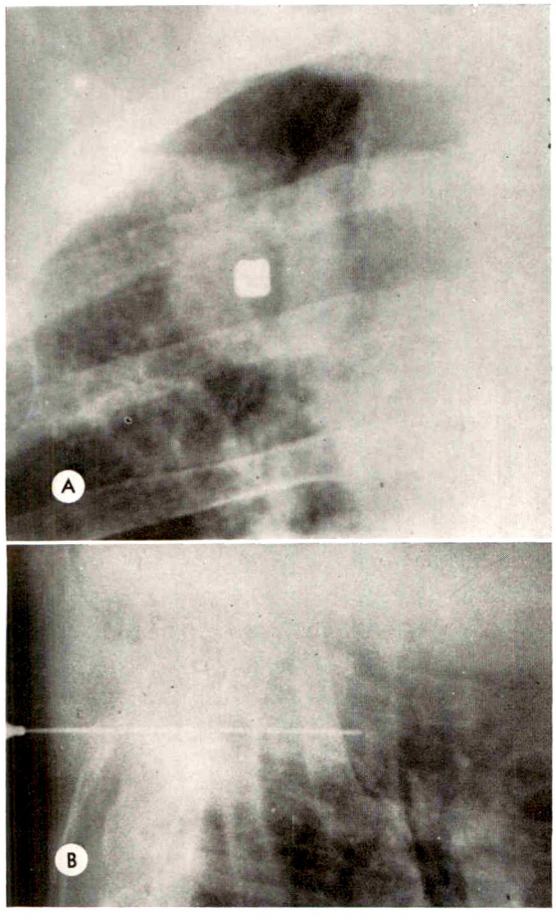



FIG. 1. (*A* and *B*) Anteroposterior and lateral roentgenograms of the chest during performance of an aspiration lung biopsy showing the needle in the consolidation. The patient was a 58 year old male with severe chronic lung disease, whose chest roentgenograms showed a consolidation in the right upper lung. Sputum cytology, bronchoscopy, and scalene lymph node biopsy were reported as negative.

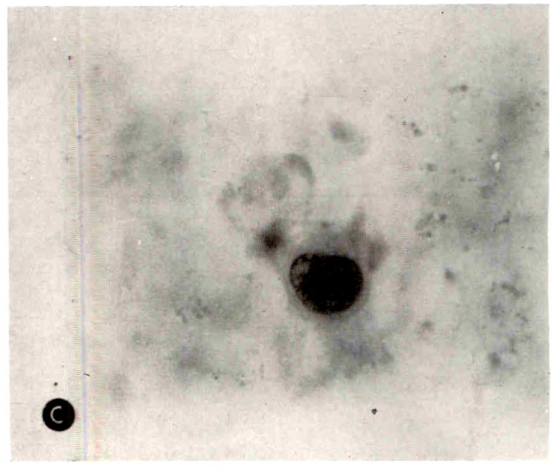



FIG. 1. (*C*) Cytologic smear shows adenocarcinoma. Papanicolaou's stain, ×960.

in both directions to loosen cells. The syringe was attached and strong suction was applied until a drop or two of serosanguineous fluid appeared in the syringe. Suction was maintained until the needle was withdrawn from the chest in order to minimize the chance of seeding along the needle track. If frank blood was obtained, the needle was repositioned. A chest roentgenogram was taken immediately after the procedure and repeated 2 hours later to evaluate any complications, and the patient was observed carefully in the hospital at least until the following morning.

### PREPARATION OF THE ASPIRATED SPECIMEN

The specimen was prepared for examination by several methods: (1) Smears on glass slides were made from the aspirate, fixed immediately in 95 per cent ethyl alcohol, and stained with the Papanicolaou technique; (2) air dried, unfixed smears were prepared for potential Wright-Giemsa stain in cases of lymphoma, Gram stain for bacteria, stains for acid-fast bacilli and other special procedures as required; (3) material was prepared for microbiologic cultures in suspected cases; (4) 10 ml. of saline were then drawn into the syringe through the biopsy needle and any tissue fragments were isolated and fixed in 10 per cent neutral formalin for 1–4 hours; (5) a cell block was prepared by centrifugation of the remaining aspirate and fixed in Bouin's

TABLE I

LUNG PATHOLOGY RESULTS OF ASPIRATION LUNG BIOPSY IN 39 PATIENTS

| Tumor Type | Examination | | |
|---|---|---|---|
| | Cytology | Tissue | Combined Results |
| *Malignant* | | | |
| Squamous cell carcinoma | 10 | 5 | 10 |
| Adenocarcinoma | 10 | 6 | 10 |
| Oat cell carcinoma | 2 | 2 | 2 |
| Transitional cell carcinoma | 0 | 1 | 1 |
| Carcinoma, type undetermined | 4 | 7 | 3 |
| Total | 26 | 21 | 26 |
| *Other** | | | |
| Suspicious A | 1 | 2 | 2 |
| Negative B | 9 | 6 | 8 |
| Unsatisfactory C | 3 | 5 | 3 |
| Total | 39 | 34 | 39 |

* Final diagnosis established on clinical or surgical grounds:
A. Includes
1 metastatic lesion,
1 lesion initially interpreted as ?lymphoma at autopsy proved to be thymoma.
B. Includes
3 lesions proved to be benign at surgery,
1 false negative—no malignant cells obtained at biopsy,
4 diagnosis believed benign on basis of repeated clinical and radiologic follow-up.
C. Includes
1 diffuse lung disease ?fibrosis,
2 carcinomas; in one patient a bloody biopsy was obtained and in the other insufficient material was aspirated.

solution for 24 hours. Sections of the paraffin-embedded tissues and cell blocks were stained with hematoxylin and eosin, and in selected cases with the periodic acid-Schiff procedure after diastase digestion for identification of epithelial mucin.

## RESULTS

Forty-three biopsies of intrathoracic lesions have been performed in 39 patients (Table 1). In 37 patients the lesions were focal consolidations located in the peripheral or hilar regions; 1 patient had a mass in the mediastinum and in 1 patient there was diffuse lung disease. The smallest focal lesion biopsied was 2 cm. in diameter.

The cytologic examinations proved to be the most accurate of the pathologic methods both for demonstrating the presence of malignancy (in 26 of 29 malignant tumors), and for determining the specific cell type (in 22 of these 26). There were 5 patients in whom the cytologic evaluation of the specimen was positive for carcinoma, while the tissue analysis was either not available (2 cases) or not satisfactory (3 cases). In only 1 patient with malignancy was the tissue fragment interpreted as suspicious for carcinoma, while the cytology study was negative. By combining the results of both methods, a diagnosis of carcinoma was made in 26 of the 29 proven malignant cases, and a specific cell type was identified in 23 of these patients. Examination of the cell block preparations proved to be the least accurate of the three pathologic methods. In no case was it diagnostic when the cytologic or tissue methods were unrevealing.

Of the 2 patients whose biopsies were interpreted as suspicious, 1 was proved by operation to have a metastatic lesion; in the other, the initial interpretation of probable lymphoma was later changed to thymoma.

Eight patients had biopsies which were interpreted as negative. In 3 of these patients, the lesions found at surgery were

TABLE II

BONE PATHOLOGY RESULTS OF ASPIRATION BONE BIOPSY IN 11 PATIENTS

| Patient | Area Biopsied | Cytolcgy | Tissue |
|---|---|---|---|
| 1 | Femur | Adenocarcinoma | Adenocarcinoma |
| 2 | Scapula | Negative | Negative |
| | First lumbar vertebra | Unsatisfactory | Adenocarcinoma |
| 3 | Pelvis | Carcinoma | Carcinoma |
| 4 | Eighth dorsal vertebra | Negative | Negative |
| 5 | Rib | Unsatisfactory | Unsatisfactory |
| 6 | Sacrum | Undifferentiated carcinoma | Undifferentiated transitional cell carcinoma |
| 7 | Sacrum | Unsatisfactory | Negative |
| 8 | First lumbar vertebra | Negative | Negative |
| 9 | Femur | Squamous carcinoma | Squamous carcinoma |
| 10 | Twelfth thoracic vertebra | Suspicious for carcinoma | Renal cell carcinoma |
| 11 | First lumbar vertebra | Unsatisfactory | Unsatisfactory |

respectively a neurofibroma, a benign cystic middle lobe and multiple granulomas. Four other cases in this group continue to be regarded as benign on the basis of repeated clinical and roentgenologic examinations. Probable diagnoses in this group include 2 patients with pulmonary infarcts, 1 with probable granuloma and 1 with a very slowly resolving pneumonia. In none of these benign cases was a definite histologic diagnosis made by aspiration biopsy. In 1 patient with a 2 cm. lesion in this group, no diagnosis could be made from the aspirated material; surgery, done before the recommended repeat biopsy could be made, revealed a small lung carcinoma.

Of the 3 patients in whom the biopsies were interpreted as unsatisfactory, 1 had diffuse lung disease, not ordinarily considered an indication for aspiration biopsy; in 1 patient a grossly bloody aspirate was obtained; and in 1 patient extremely dense tissue was encountered in the greatly thickened pleura and insufficient material was aspirated. Repeat biopsy was suggested in these last 2 cases, but not done, and carcinoma was found at operation in each case. These 2 patients and the 1 mentioned earlier are the only cases in the series with malignant lung lesions in whom the diagnosis was not established by aspiration biopsy. There were no known false positive diagnoses.

### COMPLICATIONS

The incidence of minimal pneumothorax was approximately 35 per cent. One patient who had several biopsies early in the series had a slowly progressive pneumothorax that required tube drainage. This was the only serious complication.

### PERCUTANEOUS ASPIRATION BIOPSY OF BONE LESIONS

The technique of performing the biopsy and the method of handling the pathologic material do not differ from those described under aspiration biopsy of lung except in details. Biopsies (Table II) were performed using long bevel No. 18 needles except for use of a No. 17 translumbar aortographic needle for vertebral biopsies and use of a bone biopsy needle in 1 case of a sacral lesion because of the presence of very dense bone. Any bone fragments obtained must, of course, be decalcified prior to sectioning. Twelve biopsies were done in 11 patients. In Patients 1, 3, 6, 9, and 10, focal metastatic lesions were found. Patient 2 was an elderly woman with the recent appearance of generalized sclerosis of skeleton. All other studies were unrevealing of specific diagnosis. Percutaneous aspiration biopsy of scapula was interpreted as negative, but aspiration biopsy of the destroyed anterior part of the body of the first lumbar vertebra (Fig. 2, *A* and *B*) was interpreted as metastatic carcinoma tissue. Patient 4

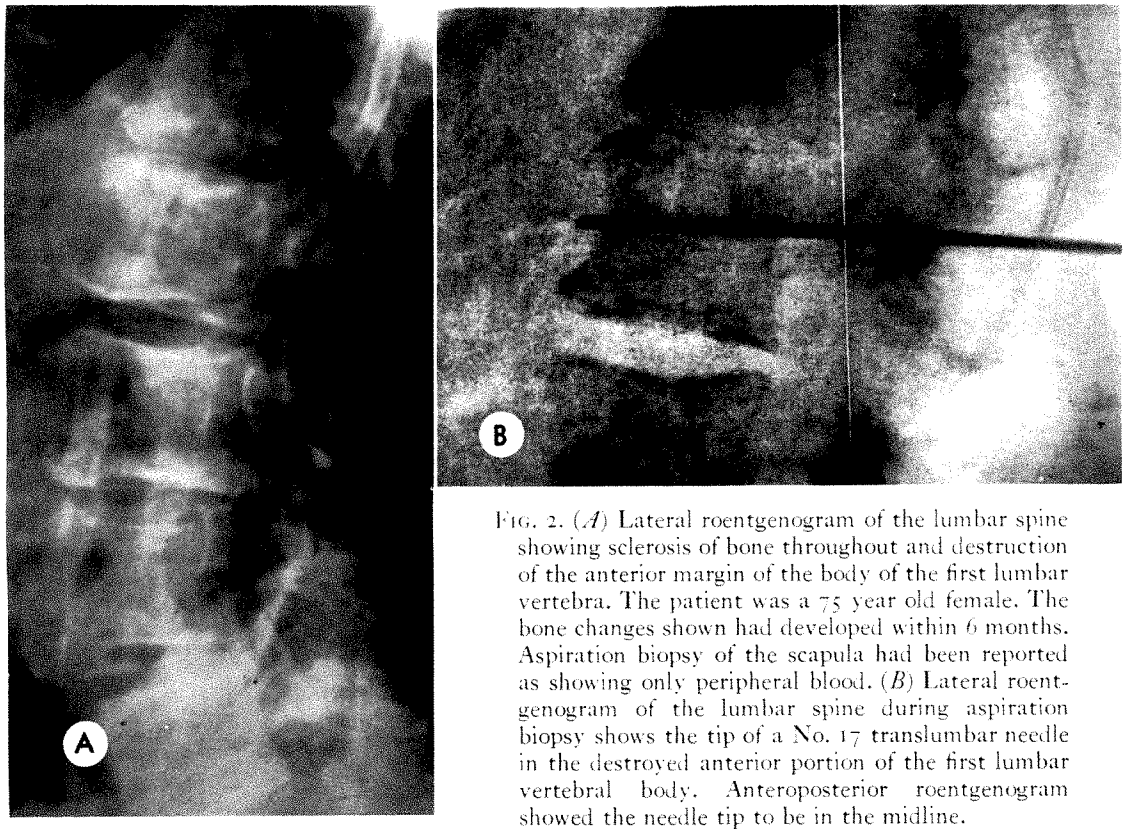


FIG. 2. (*A*) Lateral roentgenogram of the lumbar spine showing sclerosis of bone throughout and destruction of the anterior margin of the body of the first lumbar vertebra. The patient was a 75 year old female. The bone changes shown had developed within 6 months. Aspiration biopsy of the scapula had been reported as showing only peripheral blood. (*B*) Lateral roentgenogram of the lumbar spine during aspiration biopsy shows the tip of a No. 17 translumbar needle in the destroyed anterior portion of the first lumbar vertebral body. Anteroposterior roentgenogram showed the needle tip to be in the midline.

was a 56 year old man who had recent compression fractures of the fifth and eighth dorsal vertebrae, but no other symptoms or pathologic findings. He was believed to be suffering from metastatic carcinoma. However, a specimen of the eighth thoracic vertebra obtained by aspiration biopsy was interpreted as normal bone marrow. Neither clinical symptoms nor roentogenographic findings have progressed in approximately 1 year now, and it is thought probable that the changes may have been due to osteoporosis. Patient 5 had multiple destructive rib lesions. Several open biopsies and an aspiration biopsy had been nondiagnostic, showing only fibrosis and reactive tissue.[3] A final open biopsy in this case showed lymphosarcoma.

Patient 7 was an elderly man with a single sclerotic lesion in the sacrum, thought possibly to be metastatic from the pro state. In this case, biopsy was done with a No. 18 bone biopsy needle. Several specimens of bone removed from the lesion showed no tumor cells. In Patient 8 biopsy was done to exclude the presence of carcinoma in a patient believed to have sustained traumatic fracture of the first lumbar vertebra. Biopsy was negative. In Patient 11 an unsatisfactory specimen (bloody) was

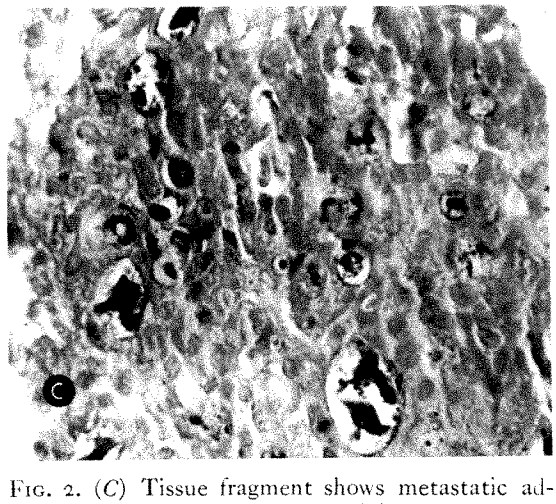


FIG. 2. (*C*) Tissue fragment shows metastatic adenocarcinoma. The dark material represents epithelial mucin. Periodic acid-Schiff reaction, × 600.

obtained; the patient was later found to have Hodgkin's disease.

## DISCUSSION

Percutaneous aspiration biopsy is especially well suited for lesions in deep seated tissues such as lung and skeleton where surgical exploration for biopsy is a major operation with appreciable risks.[1] Many structures which present a barrier to open biopsy are easily bypassed or traversed by the needle so that there is scarcely any area of the body that is inaccessible to biopsy by this method. It should be emphasized regarding the lung that this approach should be applied only to focal lesions. Diffuse lesions are better examined by open biopsy or by use of the Vim-Silverman type needle.[8,9] Some authors[10] have recommended that any patient with an undiagnosed focal lesion in the lung should have an aspiration biopsy performed prior to surgery.

The specific advantages of aspiration biopsy are many:

(a) In the event that the biopsy of a focal lung lesion establishes the diagnosis of a benign lesion or a malignant one, such as oat cell carcinoma, lymphoma or metastatic carcinoma,[6] an open surgical procedure may be avoided. Aspiration biopsy has also proved useful in excluding malignancy in lesions which are thought to be probably benign, but which are slow to resolve.

(b) The diagnosis may be readily obtained in patients with inoperable or metastatic tumors, in those considered to be prohibitive surgical risks, and in patients with lung lesions who also have evidence of metastatic disease in another organ, such as the brain.

The results of an aspiration biopsy of focal changes in a bone may indicate whether or not radiation therapy is indicated.

(c) With the diagnosis established in advance, surgical procedures, where indicated, can proceed on a pre-planned basis without the need for studies by frozen section during operation.

(d) The diagnostic work-up of a patient may be shortened if the diagnosis is made available by an aspiration biopsy relatively early in the course of the evaluation. If a positive diagnosis of a primary lung lesion such as squamous cell carcinoma or oat cell carcinoma, or of a specific metastatic lesion in lung or bone can be made by this method further search for a possible unknown primary tumor may be unnecessary. Our current practice in the evaluation of patients who have focal lung lesions, but who have no signs or symptoms referable to other organs, has tended toward examination of the sputum for cells and search for metastatic skeletal lesions followed by performance of an aspiration biopsy.

The cytologic study has frequently been able to differentiate between adenocarcinoma, squamous cell carcinoma and undifferentiated carcinoma. In addition in 75 per cent of the malignant lung lesions a fragment of tissue was also obtained by the aspiration biopsy method even though a small bore needle was used. Study of these tissue fragments has not only confirmed the cytologic diagnosis in many cases, but has also shown the actual architecture of the tumor. In the 2 patients who had had prior operations for malignancies elsewhere in the body positive identification of the lung tumor as a metastatic lesion was possible by comparison of the histologic appearances of the original tumor and the aspirated tissue fragments. Furthermore, even though the cytologic study may not be specific in benign lesions, study of the tissue fragments has sometimes allowed identification of a specific lesion.[5] Special stains, *e.g.*, for mucin, have been also applied to the tissue fragments in some cases.

For practical purposes, the pathologic interpretation may be grouped into three categories:

(1) The specimen may be interpreted as malignant, providing a definite diagnosis.

(2) On rare occasions, it may be interpreted as diagnostic of a benign lesion such as hamartoma.[5]

(3) The biopsy may be interpreted as "negative," "nondiagnostic," or "unsatis-

factory." In this event, the biopsy should be repeated if suspicion of a malignant lesion is high, since previous experience[4] has shown that the majority of "missed malignancies" will be correctly identified by a second biopsy. In the one lung cancer and one metastatic carcinoma to bone rebiopsied in this series, a diagnosis of carcinoma was obtained on each of the second biopsies.

Spread of tumor along the needle tract is rare.[7,10,11,13] In a series of 648 biopsies followed for a minimum of 5 years, not a single case of such "seeding" was reported.[10] Although there was approximately a 35 per cent incidence of minimal pneumothorax in our series of aspiration lung biopsy, these patients had no symptoms and required no therapy, except for 1 patient in whom closed drainage was necessary. Other complications reported in the literature include occasional hemothorax or hemoptysis.[10] The needling of tuberculous and pneumonic lesions has not been accompanied by untoward reactions or unusual incidence of empyema,[4] nor has puncture of a pulmonary vessel or bronchus usually been accompanied by harmful sequelae. Other complications have rarely been reported.[10]

SUMMARY

The method of percutaneous aspiration biopsy is described and discussed.

Results in 39 patients with thoracic lesions and 11 patients with skeletal lesions are shown. The clinical safety and value of the method are emphasized. The procedures are simple to perform and well accepted by patients. Correct diagnoses were made in 90 per cent of the patients with proved malignant lesions of the lung. Technical suggestions are made to further improve the diagnostic accuracy of the method.

The success of percutaneous aspiration biopsy procedures depends on a cooperative effort by the radiologist and pathologist and requires proper selection of patients, biopsy under fluoroscopic control, and provision for combined cytologic and histologic examination of the specimen.

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# IPSILATERAL HYPOPLASIA OF A PULMONARY ARTERY IN ADVANCED BRONCHIECTASIS*

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HYPOPLASIA of a pulmonary artery in association with ipsilateral pulmonary disease has recently received a great deal of attention in the literature.[2,3,5,12,14,21] When bronchiectasis is the complicating condition bronchial-pulmonary arterial shunts have sometimes also been demonstrated.[1,9] Terms, such as the hyperlucent lung[4,8,10,11] and unilateral pulmonary emphysema,[7,20] usually in association with attenuation of the pulmonary artery have also appeared. That these may be different stages of the same disease—long standing pulmonary infection, especially bronchiectasis, with resultant attenuation of the pulmonary arterial blood flow—has also been postulated by several authors.[4,12] It is not always easy to determine whether a pulmonary vascular abnormality is the original lesion or has resulted from long standing chronic pulmonary disease. It is for this reason that 3 adult patients with advanced bronchiectasis of many years duration with apparently acquired hypoplasia of the ipsilateral pulmonary artery are herein recorded.

## REPORT OF CASES

CASE I. *Left Fibrothorax and Cystic Bronchiectasis.* A 49 year old woman was admitted to Kings County Hospital on February 9, 1957, with chief complaints of frequent colds and chronic productive cough. She reported having been diagnosed having pulmonary tuberculosis 20 years previously, and subsequently she had spent long periods at tuberculosis sanatoria, although she had never had sputa or gastric smears positive for tubercle bacillus. Besides being sickly from birth, the patient had had asthma and occasional hemoptysis. Bronchoscopy and bronchography were first performed in 1930. Physical examination on admission in 1957, revealed a well developed and well nourished woman. Blood pressure was 120/70 mm. Hg; the heart was shifted into the left hemithorax, the rate was regular, and there were no murmurs. Fine inspiratory rales were heard in the left upper lung field posteriorly, and coarse inspiratory and expiratory rales were present at the left base.

Conventional roentgenograms and bronchograms of the chest on April 24, 1957, disclosed a left fibrothorax and widespread saccular bronchiectasis of the left lung (Fig. 1, *A* and *B*). Frontal and lateral angiocardiograms showed rotation of the heart into the left hemithorax and a hypoplastic left pulmonary artery (Fig. 1, *C* and *D*). The patient has been seen regularly in the clinic during the past 10 years. She has continued to have recurrent "colds" with expectoration of thick yellow sputum but improved following postural drainage, bronchodilators, and antibiotic drugs; pneumonectomy has not been advised.

CASE II. *Fibrothorax and Suppurative Bronchiectasis Cured by Left Pneumonectomy.* A 45 year old Negro male was admitted to Kings County Hospital on October 10, 1957, with complaints of fever, chills, severe cough productive of yellow, foul smelling sputum (about one-half cup per day), night sweats, and chest pain of 10 days' duration. He was well until 1953, when because of cough, a roentgenogram of the chest was made and it showed the heart and mediastinum pulled into the left hemithorax with scattered infiltrations in the upper two-thirds of the left lung field. He had then been admitted to another hospital on May 8, 1953, but was discharged on June 12, 1953, because of alcoholism. He had been followed by a Board of Health Chest Clinic. The tuberculin test was negative and several sputum specimens were also negative for acid fast bacilli. A diagnosis of pulmonary suppuration

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† Recipient of Career Scientist Award of The Health Research Council of the City of New York under contract I-549.

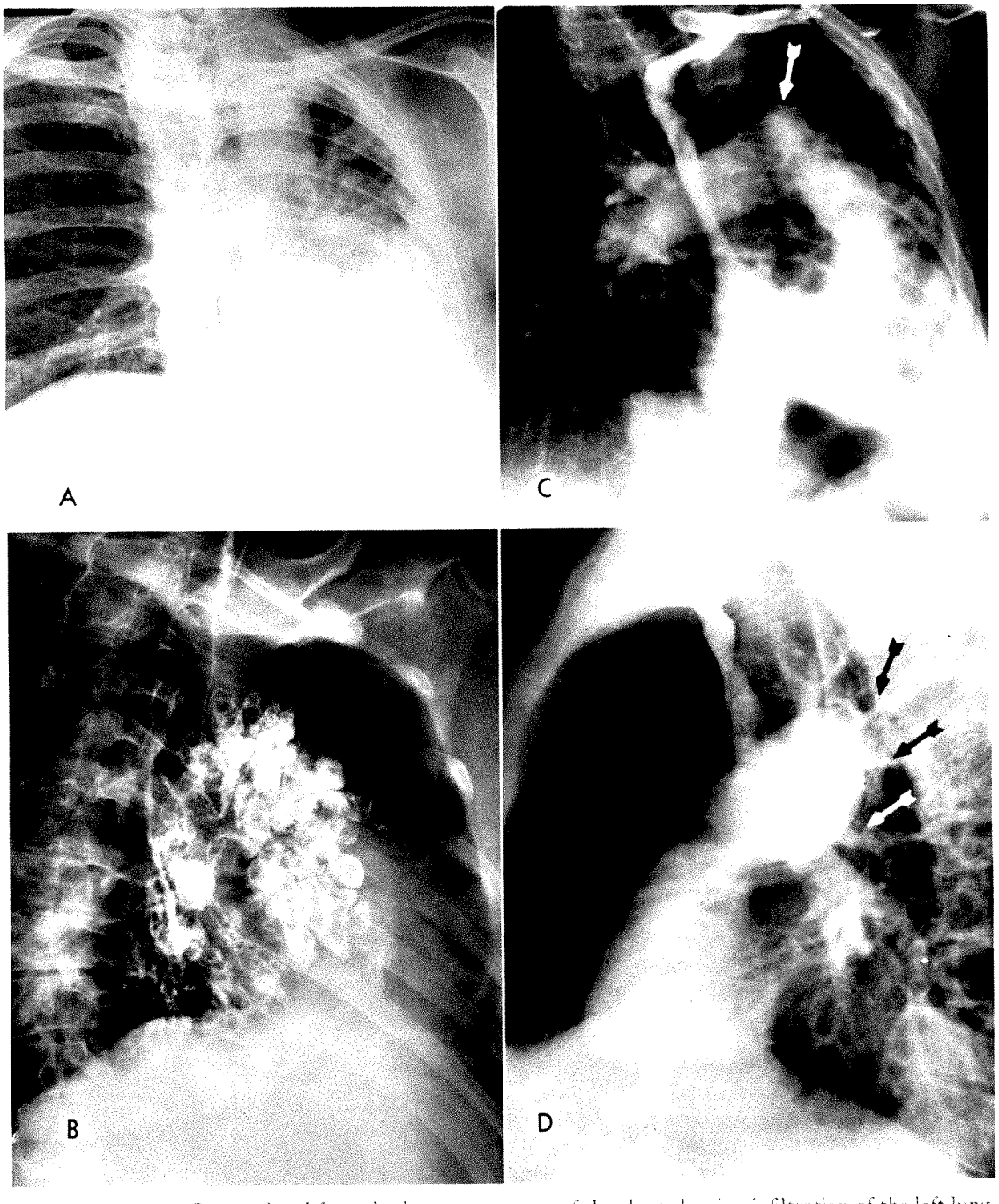



FIG. 1. Case 1. (*A*) Conventional frontal teleroentgenogram of the chest showing infiltration of the left lung, deviation of the trachea, mediastinum, and heart into the left hemithorax (fibrothorax). (*B*) Oblique bronchogram showing saccular bronchiectasis of the entire left lung. (*C*) Serial frontal angiocardiogram showing rotation of the superior vena cava, right atrium, ventricle, and pulmonary artery into the left hemithorax. Note the large right pulmonary artery, whereas the left branch is hypoplastic and "on-end" (arrow). (*D*) Serial oblique angiocardiogram showing the normal ascending and descending branches of the right pulmonary artery and meager scattered left branches of the pulmonary artery (arrows).

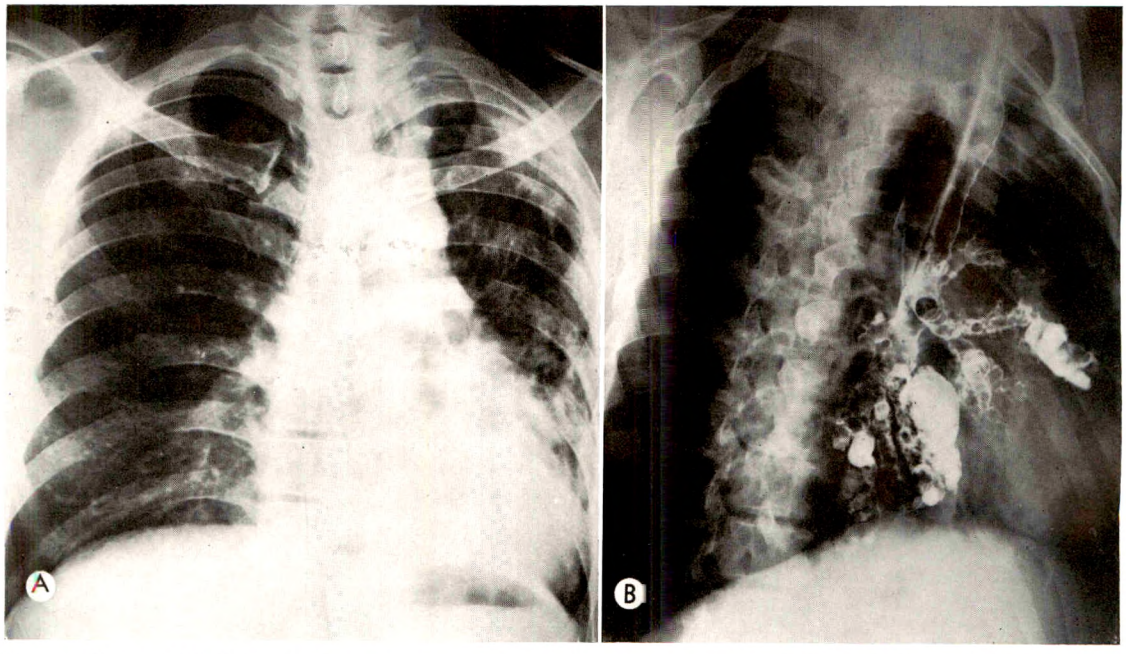



Fig. 2. Case 11. (*A*) Frontal teleroentgenogram of the chest showing the diffusely infiltrated left lung, the fibrothorax on the left side, and the cavitary lesions of the left mid lung. (*B*) Oblique bronchogram showing saccular bronchiectasis involving practically the entire left lung.

was made and the patient was referred to the hospital on October 17, 1953, but signed out in December. He was subsequently readmitted and treated with antituberculous drugs from March to October 1956. Positive cultures for tubercle bacilli were never obtained. During 1957, the patient continued to cough and expectorate one-half cup of yellowish, foul smelling sputum daily.

A week before the present admission after heavy drinking, the patient had generalized convulsions. Physical examination showed a well developed acutely ill man, coughing frequently, but without dyspnea. The blood pressure was 120/60 mm. Hg, the pulse 120, respirations 20, temperature 104.4°F., and the skin was hot and dry. The mouth was foul smelling and the teeth were in poor condition with multiple cavities. The trachea was deviated to the left and there was decreased respiratory excursion of the left lung. On percussion of the lung, there was dullness over the posterior lower one-third of the chest. Vocal and tactile fremitus were decreased over the left lower thorax. Tubular breathing and coarse moist rales were heard over the upper one-half of the left chest. No cardiac enlargement or murmurs were found. Marked clubbing of the fingers without cyanosis was present. Roentgenograms of the chest revealed dense and radiolucent areas and saccular bronchiectasis involving the entire left lung (Fig. 2, *A* and *B*). Sputum culture grew out *Staphylococcus aureus*, *Streptococcus viridans*, and *Pneumococci*. The sputum and urine cultures were negative for the tubercle bacillus. The serologic test for syphilis was negative. There was no leukocytosis.

On admission in October 1957, the patient was treated with large doses of penicillin and streptomycin. He became afebrile in November 1957, and had a left pneumonectomy on January 21, 1958. The roentgenograms of the chest showed a left fibrothorax with deviation of heart, trachea, and mediastinum into the left hemithorax (Fig. 2*A*). Bronchography on November 14, 1957, showed cystic bronchiectasis involving practically the entire left lung (Fig. 2*B*). Pulmonary function studies showed diminution of the vital and maximal breathing capacities, while cardiac catheterization showed normal hemodynamic findings and diminished blood flow in the left lung. Angiocardiography on November 7, 1957, disclosed rotation of the cardiovascular structures into the left hemithorax with marked attenuation of the left pulmonary artery (Fig. 2, *C–E*). A bronchovascular injection cast of the excised left lung,

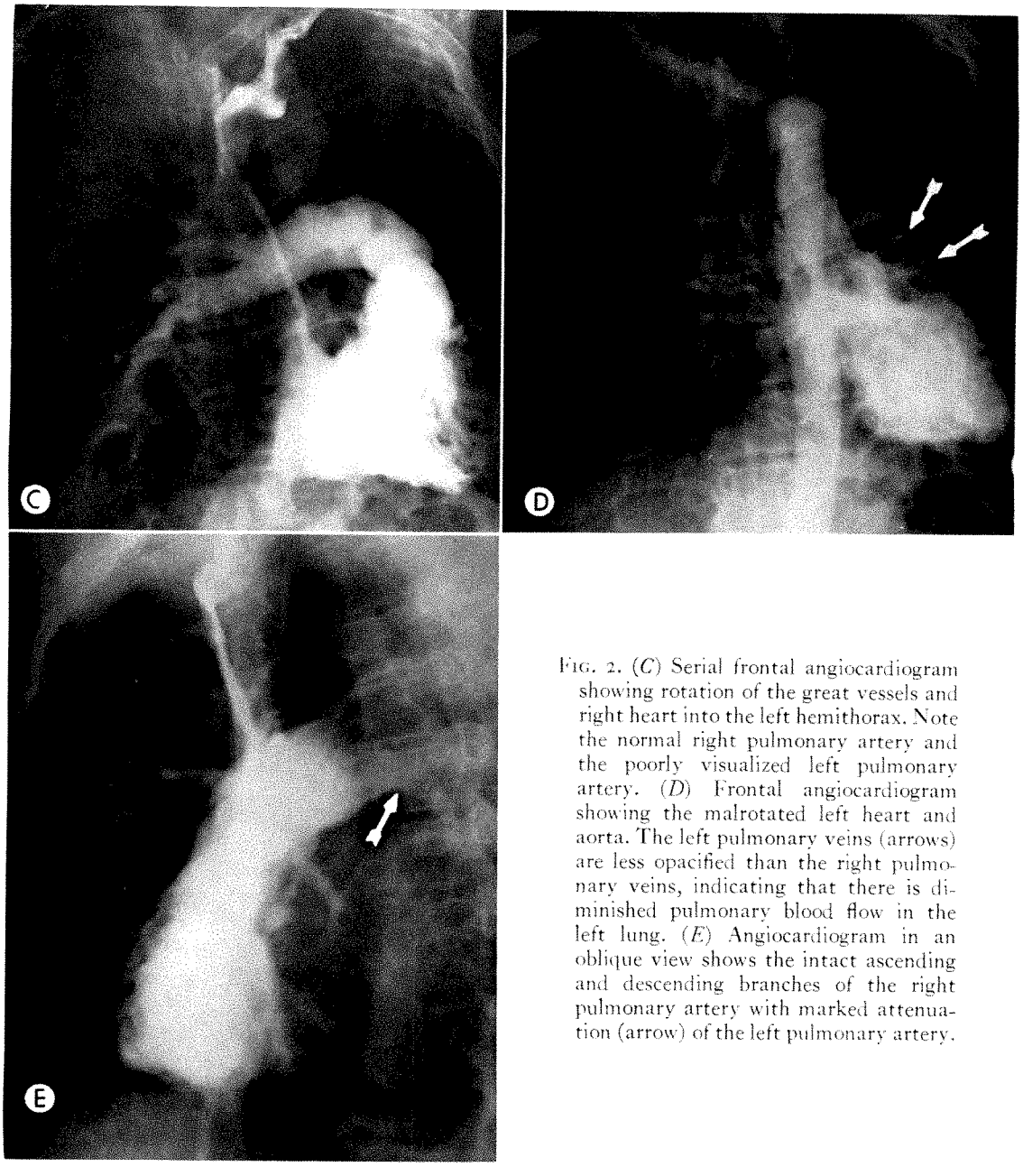



FIG. 2. (*C*) Serial frontal angiocardiogram showing rotation of the great vessels and right heart into the left hemithorax. Note the normal right pulmonary artery and the poorly visualized left pulmonary artery. (*D*) Frontal angiocardiogram showing the malrotated left heart and aorta. The left pulmonary veins (arrows) are less opacified than the right pulmonary veins, indicating that there is diminished pulmonary blood flow in the left lung. (*E*) Angiocardiogram in an oblique view shows the intact ascending and descending branches of the right pulmonary artery with marked attenuation (arrow) of the left pulmonary artery.

made by Dr. Robert Ferguson, showed a hypoplastic left pulmonary artery (Fig. 2, *F* and *G*). The left lung was almost completely airless, firm, and hard, and markedly decreased in size. The patient had an uneventful postoperative course and was discharged improved on February 17, 1958.

CASE III. *Hyperlucent Left Lung and Asymptomatic Bronchiectasis.* A 34 year old Negro female was seen because of an abnormal roentgenogram of the chest on the third day following delivery of a normal healthy child on September 26, 1966. She had had no respiratory symptoms except for an occasional cough. She was well developed and well nourished. There was increased resonance over the left lung and coarse moist rales were heard over the posterior three-quarters of the left chest. The heart was normal; there were no murmurs and the pul-

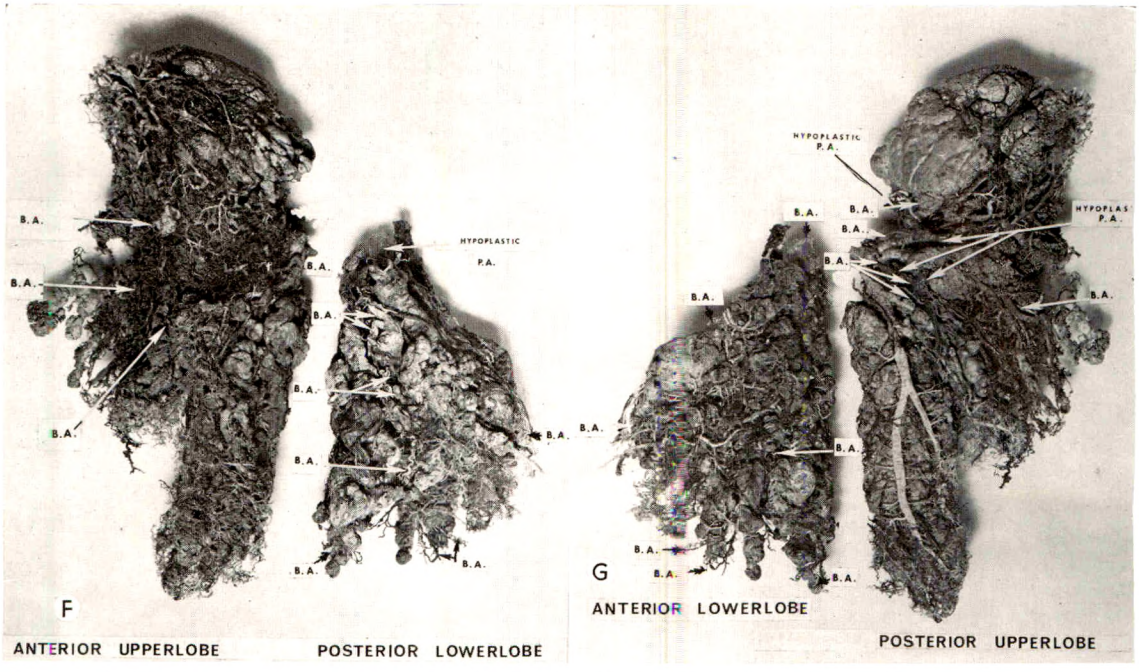



FIG. 2. (*F*) Photograph of the excised specimen showing the injected pulmonary bronchial circulations of the left anterior and posterior surfaces of the lower lobes. Arrows point to the hypoplastic pulmonary artery. The bronchial (B.A.) arterial blood supply was increased but anastomoses between the two systems could not be demonstrated. (*G*) Photograph of the anterior lower and posterior portions of the left lung also showing the hypoplastic pulmonary and plethoric bronchial arterial (B.A.) circulations. (*F* and *G* were made available by Dr. Robert Ferguson.)

monic second sound was greater than the aortic second sound. A roentgenogram of the chest showed increased aeration of the left lung, absence of the pulmonary vasculature, and a normally centrally-placed heart (Fig. 3*A*). Bronchograms made on September 29, 1966, showed cylindrical bronchiectasis of the left upper lobe and saccular and cystic bronchiectasis of the left lower lobe (Fig. 3, *B* and *C*). Angiocardiograms made on October 3, 1966, revealed only a rudimentary ascending branch of the left upper pulmonary artery (Fig. 3, *D* and E). Because she was asymptomatic, treatment did not seem indicated. She was well when last seen in the Pulmonary Clinic on December 19, 1966.

## DISCUSSION

Roentgenography, especially serial angiocardiography and bronchography, permits differentiation of a hypoplastic (attenuated) pulmonary artery from congenital absence of a main pulmonary artery,[16] traumatic rupture of a bronchus[17] and agenesis of a lung or branch.[18] Pulmonary function studies, especially bronchospirometry, cardiac catheterization, and bronchial arteriography provide additional and valuable diagnostic data.[1,5,8,9,12] Only Case II of our series had pulmonary functional studies and these showed only mild diminution of the vital and maximal breathing capacities. Cardiac catheterization studies in the same patient showed normal hemodynamic findings except for diminution of the oxygen content and blood flow in the left pulmonary artery. In the other 2 patients, cardiac catheterization and pulmonary function studies were not offered because surgical treatment was not anticipated. Bronchial arteriography would have been of interest in Case III but, here too, it seemed to be of academic rather than of practical importance.

In Cases I and II, the conventional frontal roentgenograms and serial angiocardiograms showed the classic features of fibrothorax with rotation of the cardio-

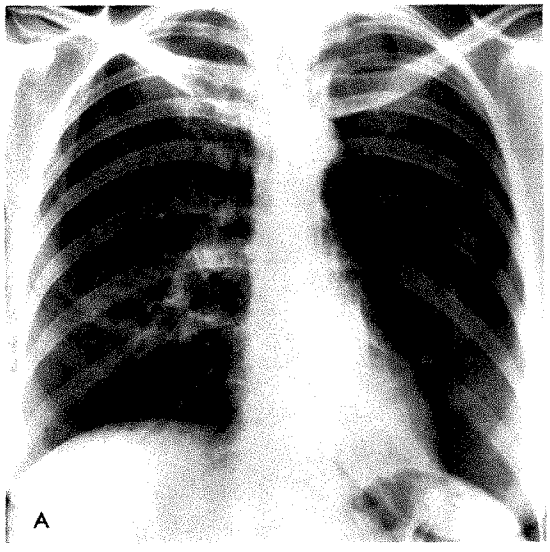



FIG. 3. Case III. (*A*) Frontal teleroentgenogram of the chest showing a hyperlucent left lung practically devoid of pulmonary vasculature. Note the normally centrally-placed heart and great vessels.

vascular structures, lung, and trachea into the left hemithorax (Fig. 1, *A* and *C*; 2, *A*, *C* and *D*).[15] In these patients advanced bronchiectasis was apparently responsible for the pulmonary changes (Fig. 1*B*; and 2*B*). These were in marked contrast to the roentgenographic findings in the third patient. Instead of opaque and infiltrated left lungs, the left hemithorax

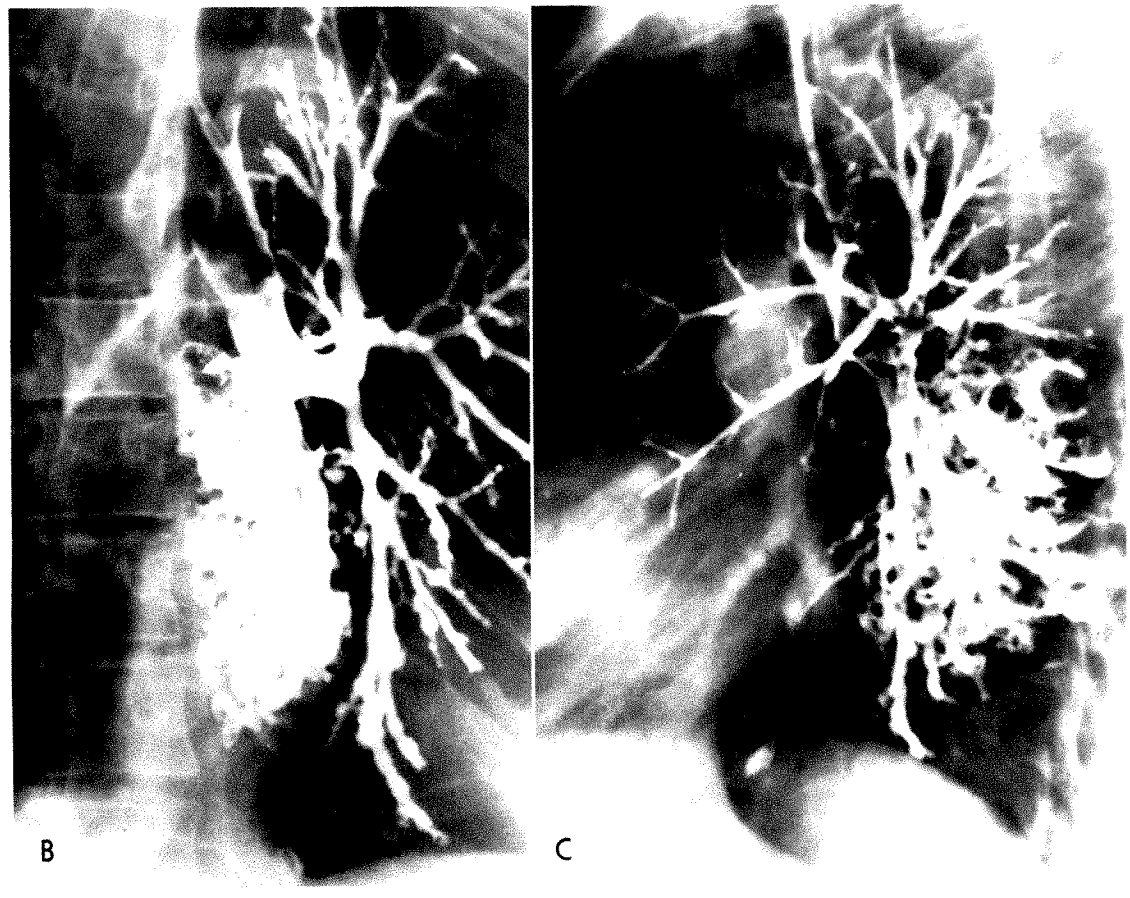



FIG. 3. (*B*) Frontal bronchogram showing cylindrical bronchiectasis of the left upper and saccular bronchiectasis atelectasis of the left lower lobes. (*C*) Oblique bronchogram showing the cystic bronchiectasis of the left lower lobe to advantage.

was translucent and the pulmonary vasculature unrecognizable (Fig. 3*A*). Also, instead of shifting of the mediastinal and tracheal structures, the heart was centrally-placed. The bronchographic studies, although also showing involvement of the left upper lobe, revealed advanced cystic bronchiectasis of only the lower lobe (Fig. 3, *B* and *C*). Serial angiocardiography in the 3 patients demonstrated attenuation of the left pulmonary artery (Fig. 1, *C* and *D*; 2, *C*, *D* and *E*; and 3, *D* and *E*).

The conventional roentgenogram and the angiocardiograms of Case III, fulfill many of the criteria that have been attributed to patients with a hyperlucent lung,[4,8,10,11] and with unilateral pulmonary emphysema.[7,20] Because of her asymptomatic state and absence of cough and expectoration, it is tempting to classify the bronchiectasis of the left lung to be of congenital origin. Yet, many patients with acquired bronchiectasis may be asymptomatic until a suppurative pulmonary complication occurs. Indeed, in childhood bronchiectasis may go unrecognized and be attributed to recurrent colds and bronchitis. Even the development of severe unilateral bullous emphysema may also be well tolerated and go unrecognized for many years.[19] It seems reasonable, therefore, to suppose that the bronchiectasis in Case III is of acquired rather than of congenital origin. The hyperlucency is probably related to complicating generalized emphysema, especially of the upper lobe, owing to the severely bronchiectatic and atelectatic lower lobe. It may be that when pulmonary suppuration complicates bronchiectasis, fibrothorax develops. Whether such a condition can ensue is conjectural because the apparently advanced emphysema and hyperlucency of the left lung may prevent the formation of fibrothorax. It is tempting though, to consider that the hyperlucent picture is a stage of bronchiectasis which has become complicated by emphysema rather than by pulmonary suppuration. In both conditions the hypoplastic pulmonary arterial circulation is apparently owing to decreased blood flow due to long standing pulmonary disease.

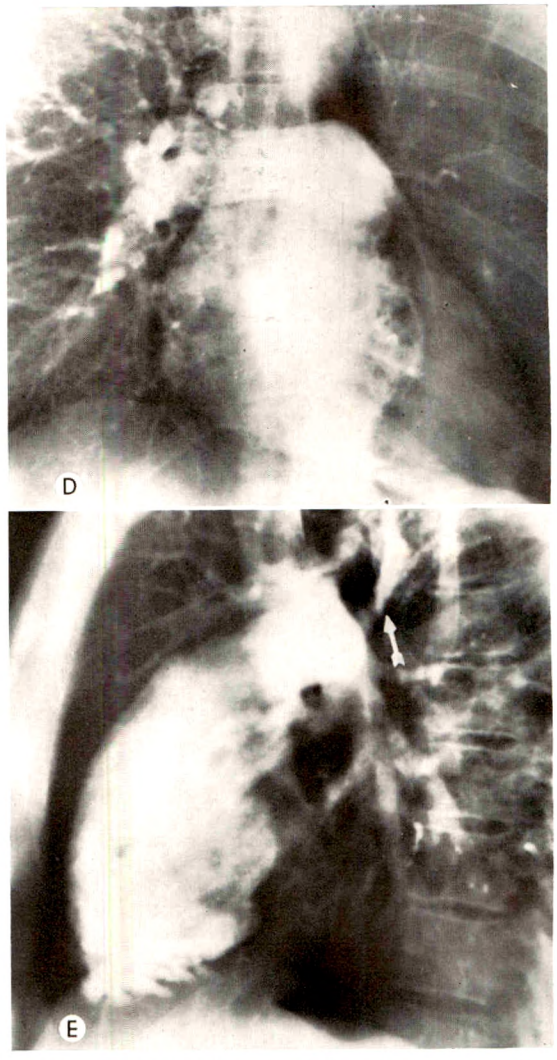



FIG. 3. (*D*) Serial frontal angiocardiogram showing an attenuated ascending branch of the pulmonary artery of the left upper lobe with almost total absence of the pulmonary circulation to the left lower lobe. (*E*) Lateral, biplane view of *D*, showing the intact right pulmonary arterial blood flow and the meager left upper ascending pulmonary arterial circulation (arrow).

## SUMMARY AND CONCLUSIONS

Three adult patients, 2 women and 1 man, varying in ages from 34 to 49 years, with advanced bronchiectasis of the left

lung and hypoplastic pulmonary arteries are reported. The 2 older patients also had suppurative bronchiectases and fibrothoraces. One patient had a pneumonectomy; injection casts of the excised lung demonstrated the hypoplastic pulmonary artery which confirmed the angiocardiographic findings. In contrast, the younger patient although asymptomatic had many moist rales at the left base and showed a hyperlucent left lung with a centrally-placed heart without deviation of the trachea and mediastinal structures. She too had advanced bronchiectasis, especially of the lower lobe, and only an attenuated left ascending pulmonary artery.

Rather than being a separate entity, a hyperlucent lung with a hypoplastic pulmonary artery and bronchiectasis may be an advanced stage of bronchiectasis with diminished blood flow complicated by emphysema owing to chronic bronchiectasis.

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VOL. 101, NO. 4

# PULMONARY VARICES MISTAKEN FOR PULMONARY AND HILAR DISEASE*

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BECAUSE a pulmonary varix seen end-on may simulate a "coin lesion" or mediastinal tumor, diagnosis is important if exploratory thoracotomies are to be avoided. The condition is rare, only 20 cases have been previously described in the literature.[7,8] Of these, only 10 have been diagnosed after angiography.[1,4,6–9,13] Herein is reported the case of a 53 year old woman who had abdominal pain and a chance roentgenogram of the abdomen which disclosed a small rounded density in the right lower thorax which was mistaken for a coin lesion. Another young girl with congenital heart disease who developed a huge mediastinal tumor and a right lower pulmonary varix was thought to have a metastatic lesion from the mediastinum. A third woman with a right hilar mass was suspected of having tuberculous lymphadenopathy, and when antituberculous therapy failed to affect the lesion, angiocardiography was performed, and showed a right upper lobe pulmonary varix.

## REPORT OF CASES

CASE I. *Pulmonary Varix Mistaken for Coin Lesion.* A 53 year old woman came to the emergency clinic on December 1, 1966, complaining of the sudden onset of nausea, vomiting, and diarrhea. These soon subsided and were followed by severe left lower quadrant pain in the abdomen. On examination, she was tender in the left flank and lower abdomen, and diverticulitis was suspected. A roentgenogram of the abdomen also showed the lower thorax and revealed a small rounded density in the right lower lung field (Fig. 1*A*); the barium enema study was normal. The conventional roentgenogram of the chest was also interpreted as being normal (Fig. 1*B*). However, because of the earlier study, a specially-coned view (Fig. 1*C*) and serial laminagrams of the lower right thorax were made (Fig. 1*D*) and on the basis of these, she was referred for admission into the hospital. Without further consultation with the radiology department, the patient had a right exploratory thoracotomy on January 6, 1967; no abnormality was discovered.

Following operation, in a review of the roentgenograms with more seasoned roentgenologists who were not informed about the negative thoracotomy findings, the diagnosis of a right lower lobe pulmonary varix was made. Angiocardiography on January 16, 1967 confirmed this diagnosis (Fig. 1*E*).

CASE II. *Pulmonary Varix Simulating Metastases from Mediastinal Tumor.* A 9 year old girl, with common origin of both great vessels from the right ventricle, and previously reported,[9,11] had a large mediastinal tumor (Fig. 2, *A* and *B*). A rounded density of the right lower lobe appeared to be related to the mediastinal mass, but on angiocardiography on April 7, 1959, a pulmonary varix of the right lower lobe was demonstrated (Fig. 2*C*).

CASE III. *Right Upper Lobe Pulmonary Varix Simulating Hilar Tuberculosis.* A 48 year old woman had had 3 months' treatment with antituberculous drugs which did not alter the size of the right hilar mass (Fig. 3, *A* and *B*). Angiocardiography was then performed on August 14, 1962, and showed a large pulmonary varix of the right upper lobe (Fig. 3*C*).

## DISCUSSION

Good[3] at the Mayo Clinic in 1961, and Bryk and Levin[1] in 1965 were apparently the first to publish the roentgenograms of patients with pulmonary varices that were diagnosed during thoracotomy. Also at the Mayo Clinic, Vengsarkar and colleagues[13]

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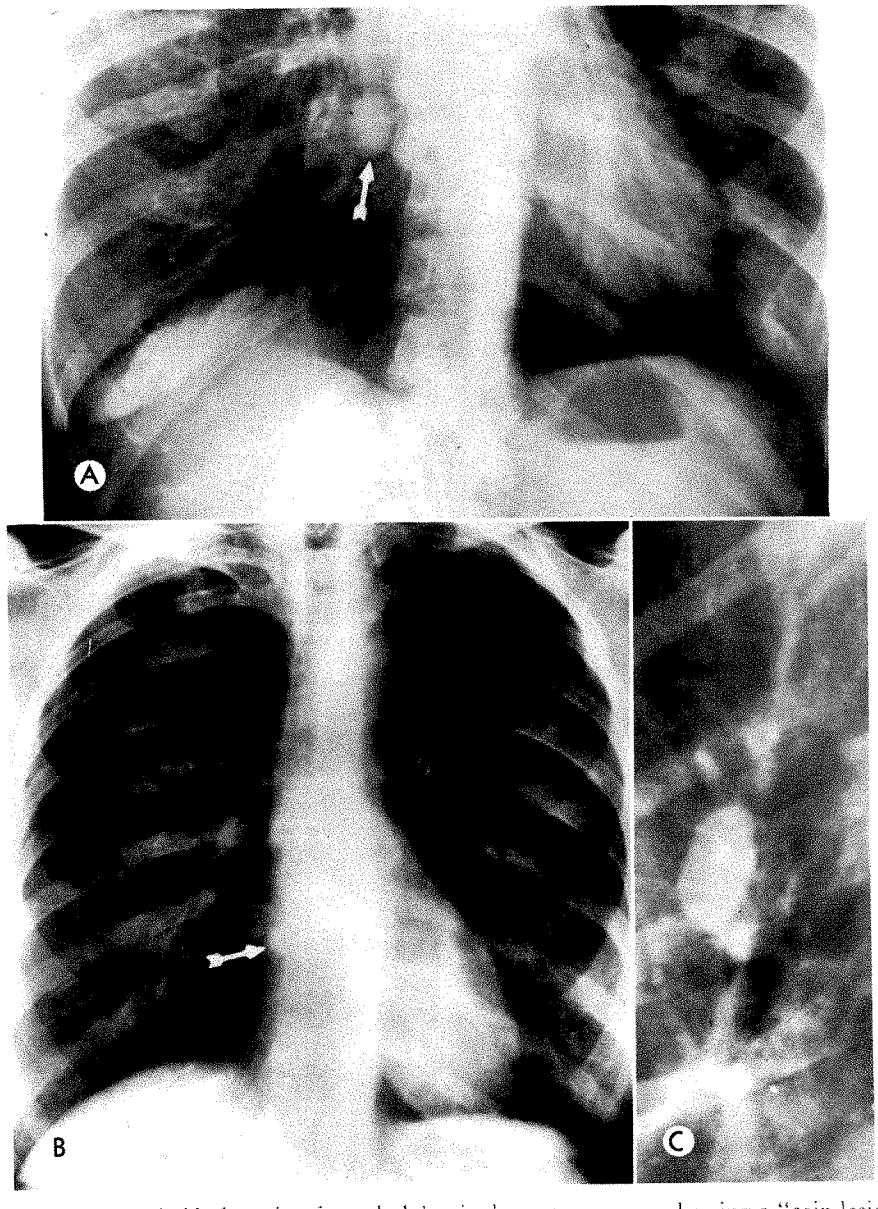



FIG. 1. Case 1. (*A*) Upper half of routine frontal abdominal roentgenogram showing a "coin lesion" (arrow). (*B*) Teleroentgenogram of the chest showing the lesion vaguely (arrow). (*C*) Special frontal view of the pulmonary mass.

in 1963, made the diagnosis of a pulmonary varix after angiocardiography in a 7 year old patient who also had a patent ductus arteriosus. The angiograms of a similar case with a huge right-sided pulmonary varix were shown recently at this center by Dr. Edgar A. Riley. However, this was found in another hospital upon thoracotomy exploration of a suspected mediastinal tumor; the diagnosis was later confirmed by angiocardiography.

The importance of angiocardiography for diagnosis of cardiovascular abnormalities became evident in 1939.[12] Mouquin and colleagues[6] in 1951, and Gottesman and Weinstein[4] in 1959, were among the first to use angiography for diagnosis of varicosities of the pulmonary veins. In 1959, in this center, while studying a mediastinal mass that developed in a 9 year old patient with both great vessels arising from the right ventricle,[10,12] a pulmonary varix was also demonstrated (Fig. 2, *A–C*). In 1962, Dr. Harold A. Lyons, who had also visualized the right upper lobe pulmonary varix, referred the asymptomatic patient who had originally been suspected of having right hilar tuberculosis (Fig. 3*A*). After a trial treatment with antituberculous drugs failed to affect the hilar mass, angiocardiograms showed a right upper lobe pulmonary varix (Fig. 3, *B* and *C*). Nelson *et al.*[8] have used angiocardiography to differentiate pulmonary varicosities from pulmonary arteriovenous fistulas.

Nash and colleagues[7] have proposed that pulmonary varices be called aberrant insertion of pulmonary veins. However, in Dorland's Medical Dictionary[2] the pulmonary varix is defined as an enlarged and tortuous vein, and this concise definition plus the long and well established usage of the term[4,6,8,9,13] would make this the preferred designation.

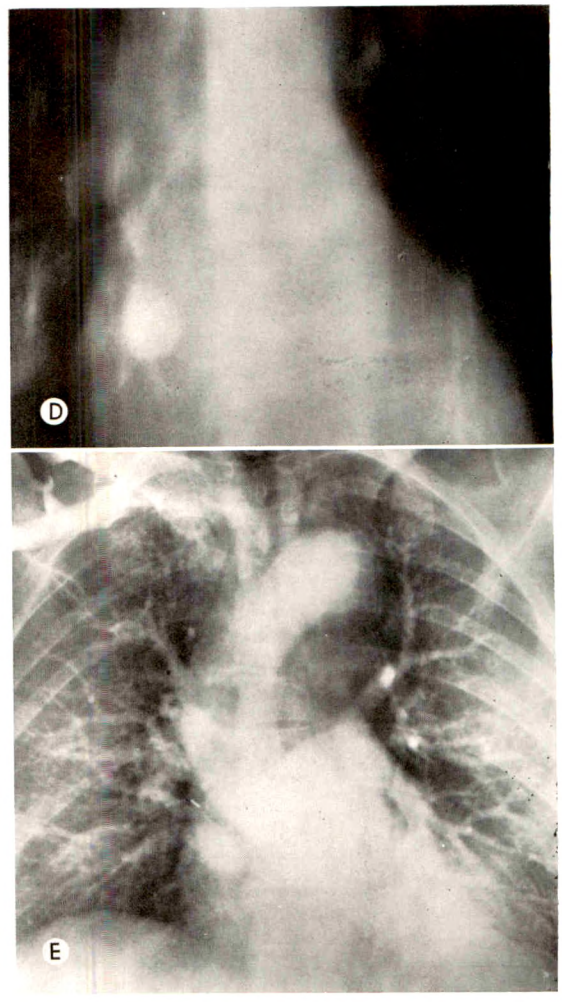



FIG. 1. (*D*) Laminagram showing the vascular nature of the pulmonary shadow. (*E*) Frontal angiocardiogram showing the lower lobe pulmonary varicosity. The metallic clip above is a marker inserted following the "negative" exploratory thoracotomy.

## SUMMARY AND CONCLUSION

Patients with varicosities of the pulmonary veins are usually asymptomatic and the lesions are discovered after roentgenographic examination of the thorax. Pulmonary varicosities may appear as rounded shadows in the lung due to the varicosity being seen end-on, and thereby simulate a coin lesion. Larger pulmonary varicosities may be more elongated or fusiform-shaped and be mistaken for hilar masses.

Conventional roentgenography, especially laminagraphy, may show the vascular nature of the abnormality; angiocardiography, however, establishes the definitive

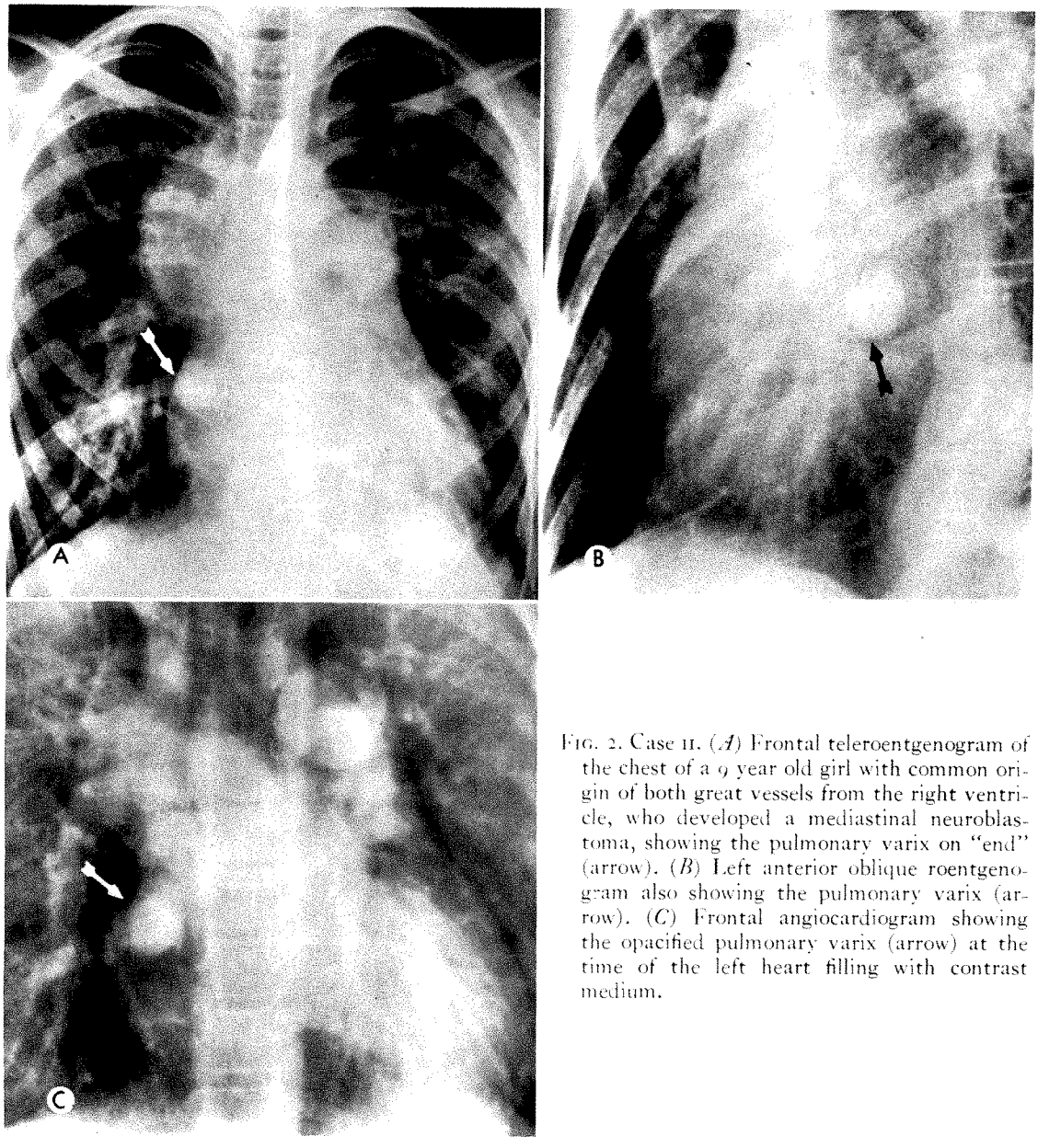



FIG. 2. Case II. (*A*) Frontal teleroentgenogram of the chest of a 9 year old girl with common origin of both great vessels from the right ventricle, who developed a mediastinal neuroblastoma, showing the pulmonary varix on "end" (arrow). (*B*) Left anterior oblique roentgenogram also showing the pulmonary varix (arrow). (*C*) Frontal angiocardiogram showing the opacified pulmonary varix (arrow) at the time of the left heart filling with contrast medium.

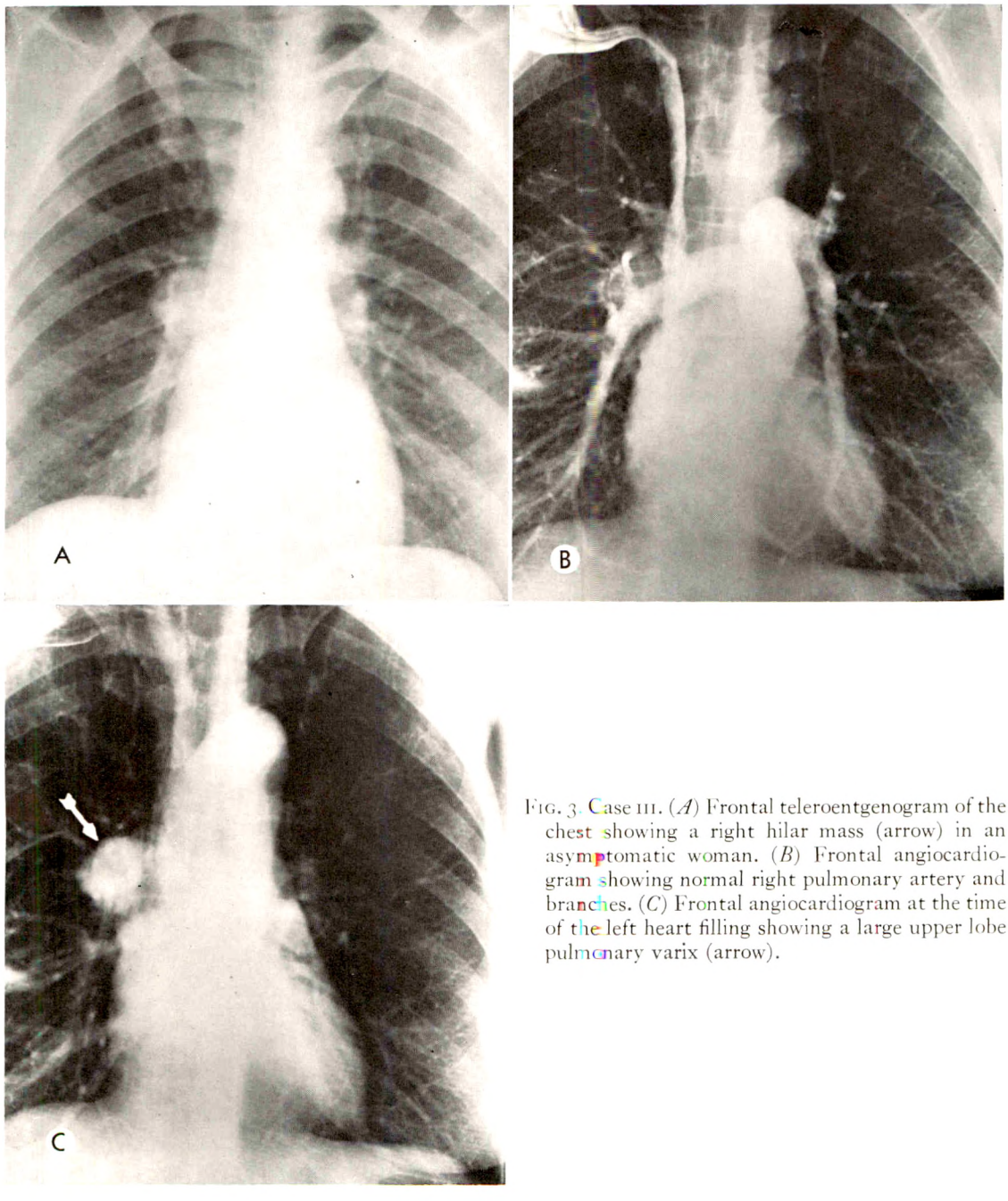



FIG. 3. Case III. (*A*) Frontal teleroentgenogram of the chest showing a right hilar mass (arrow) in an asymptomatic woman. (*B*) Frontal angiocardiogram showing normal right pulmonary artery and branches. (*C*) Frontal angiocardiogram at the time of the left heart filling showing a large upper lobe pulmonary varix (arrow).

diagnosis and thereby makes exploratory thoracotomy unnecessary.

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# MASSIVE SUBCUTANEOUS EMPHYSEMA COMPLICATING TRANSCRICOTHYROID BRONCHOGRAPHY*

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TRANSCRICOTHYROID bronchography has been used widely in England and on the Continent but not in the United States until recently. Few complications have been reported and these have been of a relatively minor nature.

Recently, the authors observed a case with severe generalized subcutaneous emphysema, mediastinal emphysema and unilateral pneumothorax following transcricothyroid bronchography.

## REPORT OF A CASE

An 18 year old male was admitted to the Harrisburg Hospital on March 4, 1967 for investigation of recurrent episodes of pneumonia over a 4 year period. The remainder of his history was noncontributory and the family history was unremarkable. There was no history of allergy or chronic cough.

Physical examination revealed a healthy young male with no abnormal findings. The most recent episode of pneumonia had left no residual pulmonary changes. Laboratory findings were unremarkable except for a white blood cell count of 8,000 per cubic mm. with 6 per cent eosinophils. Prior to bronchography, chest roentgenography was normal and roentgenograms of the paranasal sinuses revealed thickened membranes in the frontal, sphenoid and right maxillary sinuses. Bronchoscopy showed mild diffuse bronchitis. A specimen of mucoid material obtained during bronchoscopy was unremarkable.

Transcricothyroid bronchography was performed under local anesthesia using a double catheter technique, the outer catheter of which had an outer diameter of 0.083 inches.[3] A total of 12 ml. of 2 per cent Xylocaine for bronchial anesthesia and 30 ml. of oily dionosil as contrast material was used. Bilateral bronchography was carried out without incident.



FIG. 1. Normal bronchogram.

The bronchogram was interpreted as within normal limits (Fig. 1).

Approximately 2 hours following bronchography, the patient complained of slight pain and swelling in his neck and was found to have slight subcutaneous emphysema limited to the neck. He was not coughing excessively. Four hours after bronchography, he complained of nasal stuffiness and interscapular pain. The subcutaneous emphysema had increased. There was dysphonia but no laryngeal stridor. Chest roentgenography revealed subcutaneous emphysema and pneumomediastinum. Over the next several hours, further subcutaneous emphysema developed with extension to the face, entire trunk, scrotum and upper extremities to the finger tips. At this time he had respiratory distress, cyanosis and generalized cutaneous tenderness.

The patient was taken to the operating room where the cricothyroid membrane was explored under local anesthesia. There was no visible laceration. A 1 cm. area of discoloration from hemorrhage was present. A probe could be admitted into the trachea through the puncture site in the cricothyroid membrane. The orifice

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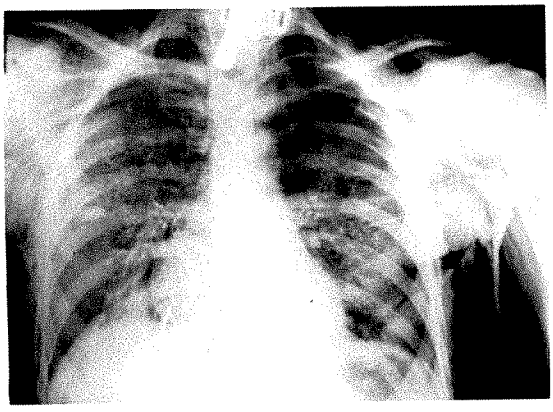


FIG. 2. Roentgenogram of the chest shows pneumothorax on the left, mediastinal emphysema, and marked subcutaneous emphysema.

was estimated to be about the size of the catheter used. Tracheostomy was performed in the upper trachea. Following this, the patient's cyanosis and respiratory distress diminished.

The next day roentgenography of the chest revealed 40 per cent pneumothorax on the left, in addition to the marked subcutaneous and mediastinal emphysema previously noted (Fig. 2). A Clagett needle was inserted and the pneumothorax improved. The subcutaneous emphysema gradually improved, and had completely disappeared in 3 weeks. The patient's facial appearance the day following tracheostomy and his appearance 3 weeks later are shown in Figure 3, *A* and *B*.

## DISCUSSION

Transcricothyroid bronchography was first described by Sicard and Forestier in 1922.[9] The first paper in the American literature was published in 1933 when Amberson and Riggins[1] described only one abscess formation in their 6 years' experience. There was a paucity of published reports in the American literature until 1958 when the first detailed procedure of transcricothyroid bronchography was pub-

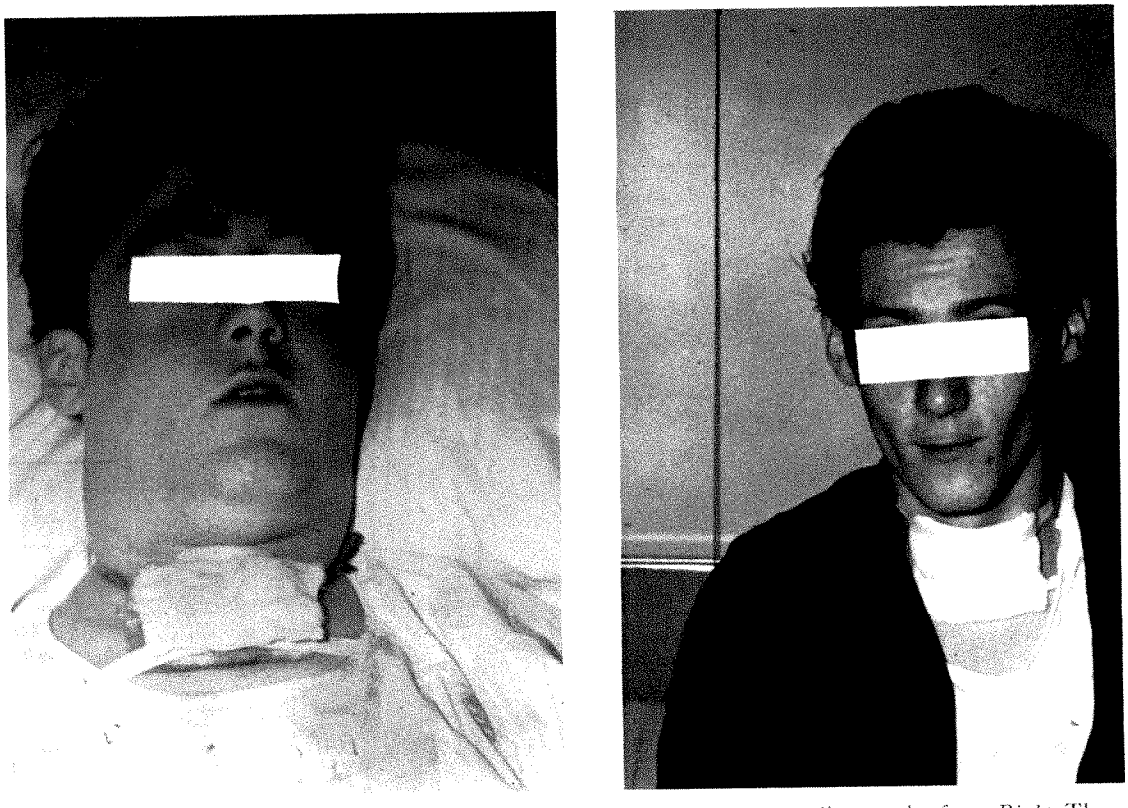


FIG. 3. *Left.* The day following tracheostomy. Note severe emphysema extending to the face. *Right.* Three weeks later, the patient's facial appearance returned to normal with complete disappearance of emphysema.

lished.[2] In 1959, Willson[11] reviewed the literature and found only 3 cases of serious complications—1 fatal mediastinal emphysema and air embolism, 1 transitory air embolism, and 1 fatal anaerobic cellulitis. These cases had been reported in the European literature. A review of published material relating to transcricothyroid bronchography in the American literature yielded 10 cases with complications.[2,4,5,7,11] These include extravasation of contrast material, cellulitis, severe cough, transient bradycardia with hypotension, hoarseness, hemoptysis, and subcutaneous emphysema. All of these complications required no treatment except for infection.

In transtracheal bronchography, 4 cases of subcutaneous emphysema were reported,[10] but were minor and required no treatment. Rossi *et al.*[8] reported 1 case of subcutaneous emphysema lasting 24 hours from the transtracheal method. Zucherman and Jacobson[12] described 7 patients with redness and swelling of the neck or thyroid following injection of contrast material outside the trachea when using a needle technique. Transcricothyroid puncture has recently been proposed as a method of obtaining pure lower respiratory tract specimens for bacteriologic examination.[6] Five cases of subcutaneous emphysema in this series of 94 patients were all selflimited, although 1 was moderately severe with extension to the face.

It is interesting to speculate on the cause of the marked subcutaneous emphysema in the reported case. Theoretically, transcricothyroid bronchography could result in laceration of the cricothyroid membrane. The orifice in this patient's cricothyroid membrane was about 2 mm. in diameter, obviously considerably smaller than the diameter of a normal open glottis. It is conceivable that laryngeal edema or spasm was present forcing air through the cricothyroid membrane. The patient did complain of nasal stuffiness and his voice was altered, but there was no laryngeal stridor. He did have a mild eosinophilia prior to the examination. Unfortunately, the patient's larynx was not inspected while the subcutaneous emphysema was developing. If laryngeal edema or spasm were to result from either local anesthesia or contrast material, the complication of severe subcutaneous emphysema might be anticipated.

Patients with severe cough should have transnasal rather than transcricothyroid bronchography. To diminish air exchange following transcricothyroid puncture, it has been suggested recently that the patients be placed at bed rest for 4 to 8 hours.[6]

SUMMARY

A case of severe subcutaneous and mediastinal emphysema complicated by pneumothorax following transcricothyroid bronchography is reported. Exploration of the cricothyroid membrane revealed an opening unlikely to have been solely responsible for the amount of subcutaneous emphysema observed. The possibility of an associated drug reaction with laryngeal edema is entertained as a complicating factor.

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# ANGIOGRAPHIC FINDINGS IN ARTERIOVENOUS FISTULAS FOLLOWING LUMBAR DISK SURGERY*

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THE possibility of the development of a postoperative arteriovenous fistula between the aorta and its iliac branches and the vena cava and its iliac tributaries has been known since the report by Linton and White in 1945.[3] In the last 20 years approximately 27 cases have been reported and summarized in the medical and surgical literature.[1,2,4,5]

The diagnosis of such a postoperative arteriovenous fistula is not difficult. A palpable thrill is often present and a continuous bruit may be audible in the region of the fistula. Enlarged pulsating veins may appear in the vicinity of the lesion and if the circulation to the extremity is seriously involved, the increased venous pressure may produce edema. If the lesion is large, cardiac failure may supervene and its symptoms bring the patient to medical attention.

The pathogenesis of the lesion is probably related to the following facts. The right common iliac artery fixes the vena cava and its tributaries against the lower vertebral column. If degeneration of an intervertebral disk involves the anterior part of the annulus, then the anterior longitudinal ligament of the spine may fail to offer a firm barrier to the surgeon's rongeur. If penetration of the ligament should occur, the instrument may tear the posterior wall of one or both of the great vessels which lie beyond. This risk is probably increased by the fixation of the vessels to the fibrous coverings of the spine by the inflammatory tissue which may have formed around the diseased disk.[5] Thus, in some operations for disk lesions the great vessels are in a vulnerable position. Many of the reported vascular complications of disk surgery occurred while experienced surgeons were operating, and no means of consistently avoiding this complication are expressed in recent literature.

Some authors have commented upon the difficulty of obtaining satisfactory vascular contrast studies of the fistula preoperatively. In the 2 cases reported by Taylor and Williams[5] aortography was unsuccessful. In the first case, the contrast material was said to be swept away so quickly that an aortogram of the vessels could not be obtained, and in the second, faint outlines of the pelvic vessels were obtained but the site of the fistula could not be demonstrated. Spittell *et al.*[4] point out that the location of such a fistula is limited anatomically to the aorta or iliac arteries and the inferior vena cava or iliac veins. They suggest that aortography is unnecessary in the preoperative evaluation of the patient.

Because of the ease with which the lesion can be demonstrated by retrograde aortography and because we are convinced that the surgeon is a better operator if he is familiar in advance with what he is likely to encounter at operation, we present the angiographic findings and clinical histories in the following 3 cases.

## REPORT OF CASES

CASE 1. D.M., U.H. #3060-E, a 47 year old Negro male, was operated upon on October 18, 1965 for herniated nucleus pulposus at the L4–L5 level. At surgery, degenerated fibrocartilage was removed using pituitary instru-

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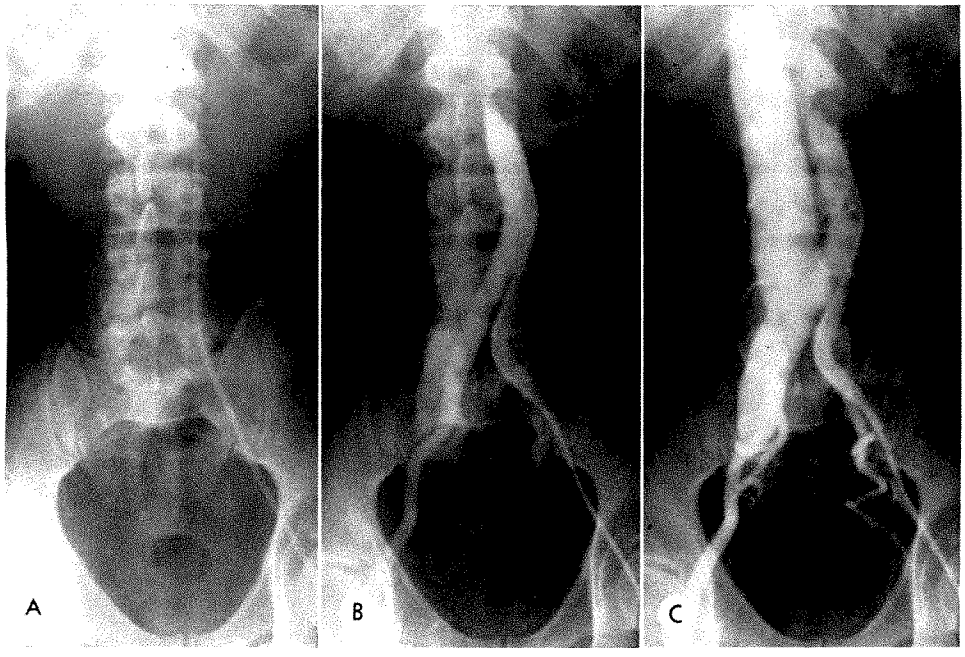



FIG. 1. Case I. (*A–C*) A gray kifa catheter with end and side holes was introduced percutaneously into the left common femoral artery and advanced into the abdominal aorta. Forty milliliters of 69 per cent Renovist were injected under a pressure of 500 lb. per square inch, followed by serial filming at the rate of 2 per second. The aortograms reveal a fistulous connection between the right common iliac artery and vein, with immediate filling of the inferior vena cava. The right common iliac vein is enlarged at the point of the fistula, and the vena cava bulges on the left at the point where its tributaries combine just anterior to the 4th lumbar interspace.

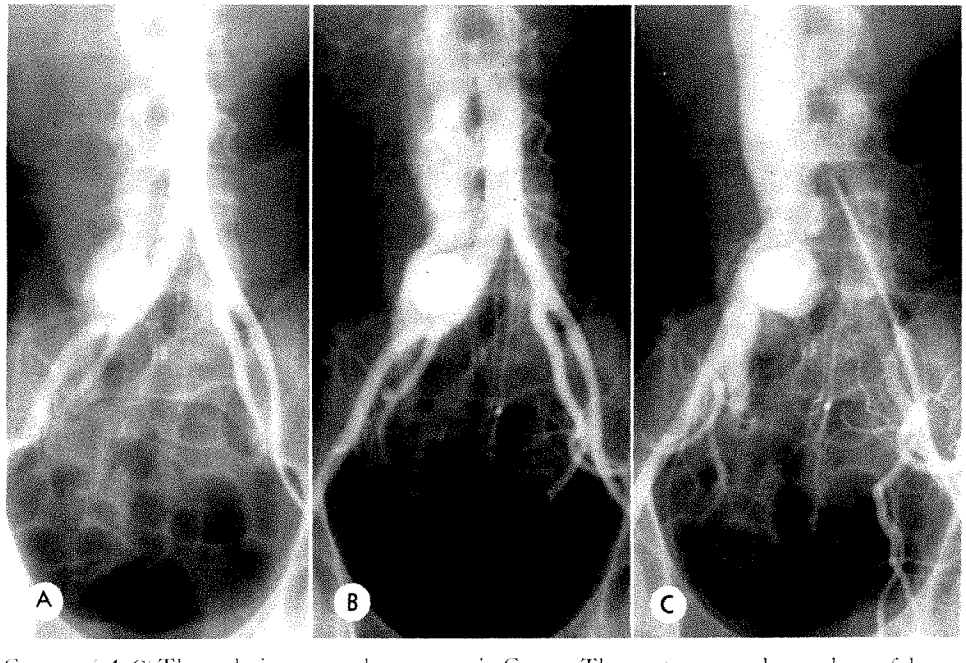



FIG. 2. Case II. (*A–C*) The technique was the same as in Case I. The aortograms show a large false aneurysm arising from the right common iliac artery, from which a fistulous communication with the right common iliac vein arises. The vena cava fills immediately. Late aortograms in the series show pooling and retention of the contrast material in the false aneurysm of the iliac artery.

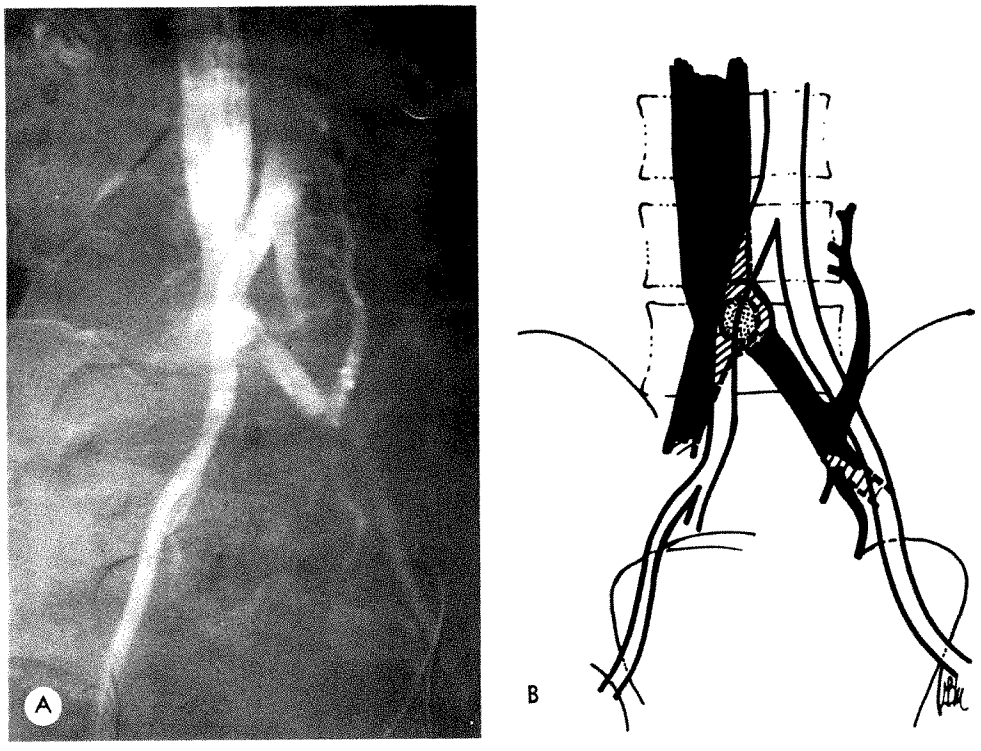



Fig. 3. Case III. A teflon needle was introduced percutaneously into the right common femoral artery and advanced until its tip lay in the right common iliac artery. Thirty milliliters of 69 per cent Renovist were injected under a pressure of 250 lb. per square inch followed by serial filming. (*A*) The subtraction technique was employed to enhance detail. A fistula is present between the right common iliac artery and the left common iliac vein. There is immediate filling of the inferior vena cava. (*B*) Line drawing of *A*.

ments, ring curettes and cup-shaped curettes. Abundant epidural venous hemorrhage was present, but no arterial bleeding was encountered. The total estimated blood loss was 600 cc., 500 cc. of which were replaced. The patient was discharged on October 30 in good condition.

He returned 2 months later complaining of the gradual onset of abdominal and leg swelling, dyspnea on exertion and paroxysmal nocturnal dyspnea. He was treated for cardiac failure with improvement. On February 28, 1966 a continuous bruit was heard over the abdomen and over the lower part of the back and a thrill was palpable in the abdomen. His left lower extremity was warmer than the right, and the pulse was stronger on the left. An arteriovenous fistula involving the right iliac artery and the adjacent vein was considered likely. He was admitted to the hospital a few days later, at which time a to-and-fro bruit was found over the right lower quadrant, loudest over the right iliac artery at its origin. A thrill was felt over the right iliac artery and the bifurcation of the aorta. Edema of the right lower extremity was present. Following aortography (Fig. 1, *A–C*) on March 9, 1966, the patient was operated upon and the arteriovenous fistula corrected by lateral arteriorrhaphy and venorrhaphy. Following the successful repair of the fistula his heart size returned to normal, and he was discharged on April 5, 1966 in good condition.

Case II. W.I., U.H. #72955-P, a 34 year old white male, underwent an operation for herniated lumbar intervertebral disk on December 15, 1966. He did well postoperatively, but soon he began to experience a dull aching pain in the right lower quadrant of the abdomen. Subsequently, his physician noted an abnormal sound over the abdomen and he was then referred to the University of Texas Medical Branch Hospitals for evaluation.

On physical examination, there was a machinery-like bruit heard over all quadrants of the abdomen but loudest over the right lower quadrant. A palpable thrill was felt over the

right femoral artery. There were good peripheral pulses in both lower extremities. The impression was arteriovenous fistula secondary to disk operation, and he was admitted to the hospital on December 26, 1966. Retrograde femoral abdominal aortograms (Fig. 2, *A–C*) demonstrated the presence of the fistula between the right common iliac artery and vein. A false aneurysm was present at the level of the fistula.

He was operated upon a month later. The fistula and aneurysm were found; the common iliac vein was closed and an unsuccessful attempt was made to repair the common iliac artery. The vessel was therefore replaced with a dacron graft. The patient made an uneventful recovery and was discharged from the hospital on February 4, 1967.

CASE III. J.H., U.H. #28672-M, a 48 year old Negro male, was operated upon on October 28, 1966 for herniated intervertebral disk at the L4–L5 level. A large amount of degenerated disk material was removed using rongeurs.

On May 22, 1967 he was seen in the Emergency Room complaining of shortness of breath and swelling of his leg. A loud to-and-fro murmur was heard over the entire abdomen, loudest in the right lower quadrant. There were roentgenographic signs of left ventricular enlargement, and it was thought that he was in congestive heart failure. His edema and dyspnea improved with medical therapy. A retrograde femoral abdominal aortogram displayed the presence of an arteriovenous fistula between the right common iliac artery and left common iliac vein (Fig. 3, *A* and *B*). On June 14, 1967 he was operated upon and the fistula repaired by direct excision and closure. Recovery was uneventful.

SUMMARY

The development of an arteriovenous fistula between the aorta and its iliac branches and the vena cava and its iliac tributaries is an uncommon complication of surgery for herniated intervertebral disk. The diagnosis is usually obvious by physical examination. Retrograde femoral abdominal aortography accurately displays the anatomic character and the location of the communication.

Three cases are reported.

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# SUBPHRENIC ABSCESS*

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SUBPHRENIC abscess continues to plague the surgeon despite the ever-increasing armamentarium of antibiotics. The radiologist plays a major role in suggesting, confirming, and localizing a subphrenic abscess, but surprisingly little has been written in the radiologic literature concerning this important problem. For this reason, a review of all cases of subphrenic abscess seen at the Hospital of the University of Pennsylvania between 1950 and 1966 was made.

Forty-eight cases were reviewed with associated roentgenographic examinations. Three cases without roentgenographic examination were not included in this series. Also eliminated were all cases in which the diagnosis was suspected on clinical grounds alone. Surgical or autopsy proof of an abscess was required for inclusion in the series. Subphrenic abscess was considered to be any abscess which was found between the diaphragm and the transverse mesocolon. Subhepatic abscess was thus included. This is a commonly accepted definition,[8,12,14,15] although Boyd[4] feels that subhepatic abscess should be considered as a separate entity.

## CLINICAL MATERIAL

In the 48 cases reviewed, 18 abscesses were found on the right side, 25 on the left, and 5 were bilateral. Twenty-eight cases were seen following initially clean intra-abdominal surgery (Table I). In contradistinction to this presumptively iatrogenic group, 20 cases were considered to be spontaneous. We have defined a spontaneous abscess as any abscess resulting from peritoneal contamination that existed prior to surgical intervention. In this group, 18 cases followed rupture of an intra-abdominal viscus. In 2 cases, no ruptured viscus could be identified so that the abscess was considered primary (Table II).

TABLE I

SURGICAL PROCEDURES FOLLOWED BY SUBDIAPHRAGMATIC ABSCESS

| | |
|---|---|
| Subtotal gastrectomy | 4 |
| Total gastrectomy | 2 |
| Esophagogastrectomy | 1 |
| Subtotal gastrectomy and vagotomy | 1 |
| Resection of jejunal tumor, *subtotal gastrectomy*, and partial colectomy | 1 |
| Splenectomy, *subtotal gastrectomy*, and right colectomy | 1 |
| Splenectomy, and *subtotal gastrectomy* | 1 |
| Splenectomy | 2 |
| Splenectomy and hiatal hernia repair | 1 |
| Splenectomy and bypass of cavernous transformation, portal vein | 1 |
| Splenectomy, vagotomy and pyloroplasty | 1 |
| Splenectomy, cholecystectomy, and portocaval shunt | 1 |
| Cholecystectomy | 3 |
| Cholecystectomy and duodenotomy | 1 |
| Common duct exploration | 1 |
| Partial hepatectomy | 1 |
| Partial colectomy | 2 |
| Total colectomy | 1 |
| Dilatation and evacuation of uterus | 1 |
| Suspension of kidney | 1 |

The age of the patients varied from 3 to 77 years. The average age of the postoperative patient was 52 years, while the average age of the patients with spontaneous abscess was 42 years. Eighteen of the 48 patients died giving an over-all mortality rate of 37.5 per cent. Four of the spontaneous cases died, giving a mortality rate of 20 per cent and 13 of the 28 postoperative cases died giving a mortality rate of 46 per cent.

Bacteriologic identification was not available in all instances, but the offending organisms most commonly encountered

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TABLE II

SUBDIAPHRAGMATIC ABSCESS

| | |
|---|---|
| Perforation of the appendix | 6 |
| Perforated duodenal ulcer | 5 |
| Perforated gastric ulcer | 1 |
| Perforated gallbladder | 1 |
| Perforated Salmonella abscess of the spleen | 1 |
| Perforated diverticulitis of the colon | 1 |
| Traumatic lacertaion of the liver | 1 |
| Unknown | 2 |
| Pancreatitis with perforation of the pancreas | 1 |
| Perforated carcinoma of the colon | 1 |

were *Staphylococcus*, *Streptococcus*, *E. coli*, *B. proteus*, and *Pseudomonas*. Often there was a mixture of organisms found on culture.

Forty-two of the 48 abscesses were identified at operation, and 6 at postmortem examination. Only 1 of those discovered at postmortem examination was an unsuspected spontaneous abscess. The remainder occurred following some form of intra-abdominal surgery.

## ROENTGENOLOGIC FINDINGS

Three major roentgenologic findings were observed (Table III). The first and probably the most important is reactive change in the lungs, pleura, or diaphragm. Specifically diagnostic when present is the second roentgenologic sign—air in the abscess cavity. A third very helpful sign is displacement of intra-abdominal viscera, seen either on the plain roentgenogram of the abdomen or in assorted barium studies. These findings are amply documented in the 6 Cases selected for illustration (Fig. 1–11).

TABLE III

ROENTGENOLOGIC FINDINGS IN SUBDIAPHRAGMATIC ABSCESS

| | | |
|---|---|---|
| *Reactive changes in the lungs, pleura, and diaphragm* | | |
| Limitation of diaphragmatic motion | 33/36 | 92% |
| Fixed hemidiaphragm | 19/36 | 53% |
| Elevation of diaphragm | 44/47 | 95% |
| Pleural effusion | 37/47 | 79% |
| Pneumonia or atelectasis | 33/47 | 70% |
| *Air in the abscess cavity* | 13/48 | 30% |
| *Displacement of intra-abdominal viscera* | 17/48 | 35% |

### REACTIVE CHANGES IN THE LUNGS, PLEURA, AND DIAPHRAGM

Four characteristic findings were variably present on roentgen examination of the chest. These included: (1) limitation of diaphragmatic motion on the involved side in patients who were fluoroscoped; (2) elevation of the hemidiaphragm on the involved side; (3) pleural effusion; and (4) pneumonia or atelectasis in the lung above the elevated diaphragm.

Thirty-six of the 48 patients were fluoroscoped for diaphragmatic motion, and 33 (92 per cent) showed definite limitation of motion. Nineteen of the 36 (53 per cent) showed a fixed diaphragm. Forty-seven patients had chest roentgenograms. Forty-four (95 per cent) of these showed elevation of the hemidiaphragm on the side involved. This was minimal in 8

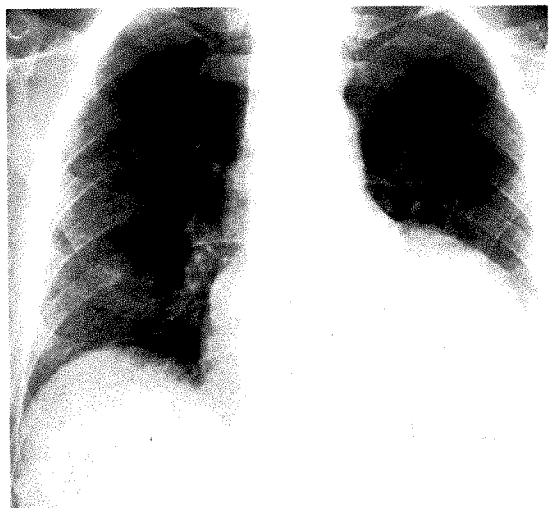


FIG. 1. Case 1. A 46 year old Negro male alcoholic with cough, fever, abdominal pain and rectal bleeding.

Chest roentgenogram shows reactive changes about the left hemidiaphragm.

The patient expired 4 days after admission without a diagnosis. Autopsy revealed an abscess of the head of the pancreas with perforation into the lesser sac and a lesser sac abscess.

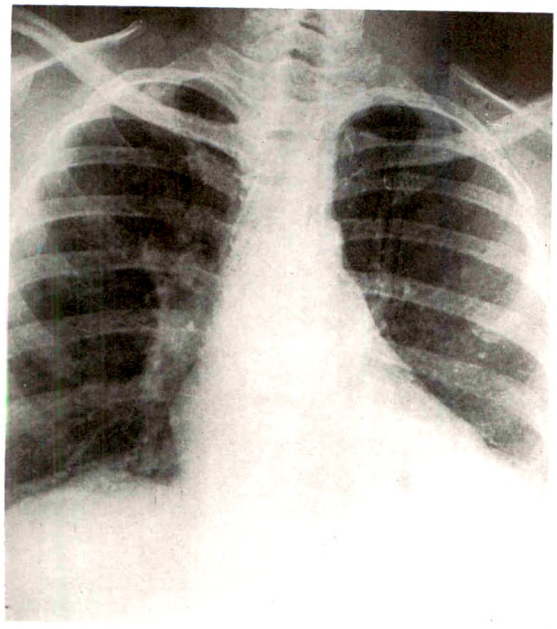


Fig. 2. Case 11. A 43 year old Negro female with a 6 week history of malaise, fever, and cough unresponsive to penicillin therapy.

Chest roentgenogram shows elevation of the left hemidiaphragm. Fluoroscopy revealed this diaphragm to be fixed.

cases, while 36 (77 per cent) exhibited a moderately or markedly elevated hemidiaphragm. Thirty-seven of the 47 patients (79 per cent) showed some degree of pleural effusion, and 33 of the 47 patients (70 per cent) showed pneumonia or atelectasis in the lower lung field on the involved side.

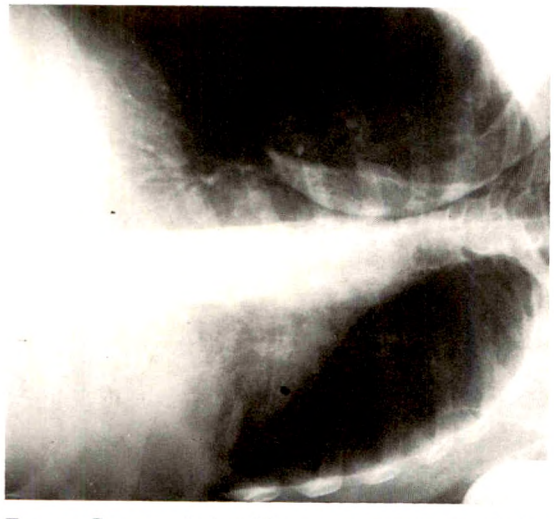


Fig. 3. Case 11. A decubitus roentgenogram of the chest reveals no free fluid.

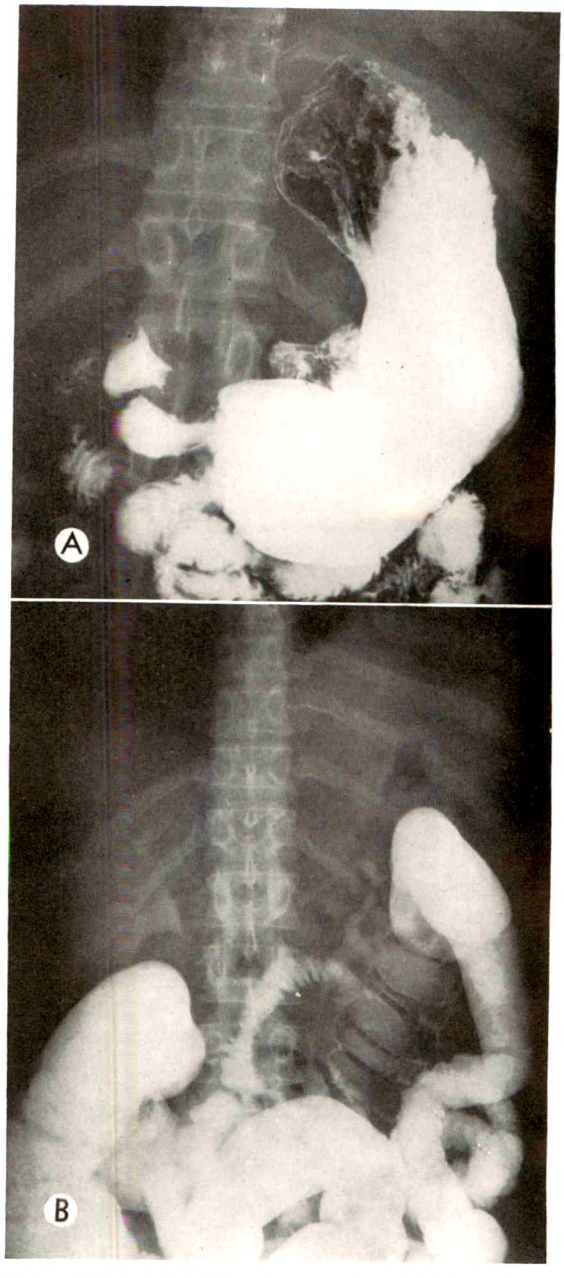



Fig. 4. Case 11. (*A*) Upper gastrointestinal series and (*B*) barium enema examination reveal displacement of the stomach and splenic flexure of the colon by a suspected subdiaphragmatic abscess.

### Air in the Abscess Cavity

Air in the abscess cavity is a specifically diagnostic sign when present. Thirteen of



FIG. 5. Case II. Pneumoperitoneum confirms the presence of a left subdiaphragmatic abscess. Exploration revealed a subphrenic collection secondary to a ruptured Salmonella abscess of the spleen.

48 patients (30 per cent) showed this finding.

### DISPLACEMENT OF INTRA-ABDOMINAL VISCERA

Displacement of intra-abdominal viscera is commonly seen in left-sided abscess, and sometimes is seen in those on the right side. Seventeen of 48 patients (35 per cent) showed some form of displacement of intra-abdominal viscera. This occurred most commonly about the stomach. Seven of these were noted on the plain roentgenogram of the abdomen. Ten were seen in upper gastrointestinal series, and 3 on barium enema examination.

## DISCUSSION

Clinically, the possibility of a localized intra-abdominal collection of pus should be considered in any patient with unexplained fever, tachycardia, or leukocytosis. Such an abscess may arise from a perforated viscus and, on rare occasions, from a remote extra-abdominal source, presumably by hematogenous spread. However, most of the cases in current practice follow initially clean intra-abdominal surgery where there has been some element of peritoneal contamination introduced at the surgical procedure.

This has not always been so. In a review of 3,372 cases in the collected literature in 1933, Ochsner and Graves[12] found that 31 per cent were secondary to perforated appendix, and 29 per cent secondary to perforated stomach or duodenum, giving a total of 60 per cent spontaneous abscess. Since the advent of antibiotics, spontaneous subphrenic abscess is less common, and postsurgical occurrence has become relatively more common. Our series showed 58 per cent of subphrenic abscess as postsurgical complications. Wetterfors[15] found that 60 per cent of his 101 cases followed recent surgery.

Males were considerably more common than females in our group, and this is true of the other large published series. The cause for this is not completely apparent,

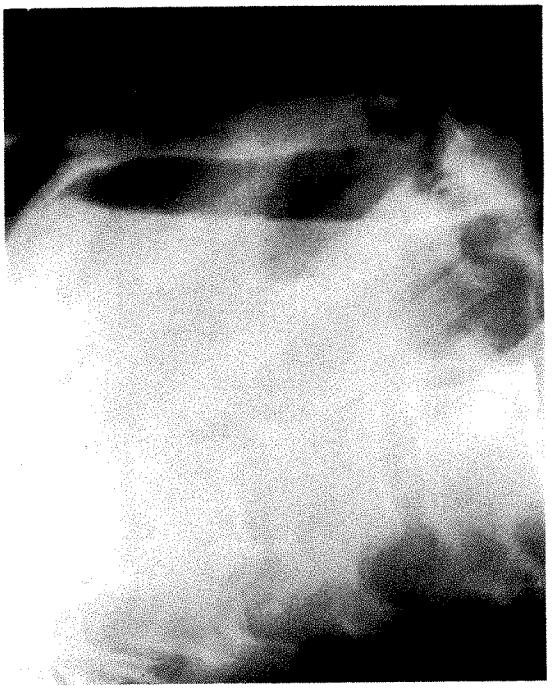


FIG. 6. Case III. A 36 year old white female with unexplained fever and abdominal pain following dilatation and evacuation of uterus for incomplete abortion.

A supine cross-table lateral roentgenogram of the abdomen reveals an air-fluid level anterior to the liver.



Fig. 7. Case III. Upper gastrointestinal series reveals downward displacement of the gastric bubble and a fistulous communication between an abscess in the right upper quadrant and the small bowel.

The patient had bilateral subphrenic abscesses at exploration.

but it is quite likely related to the higher incidence of duodenal ulcer in the male.

The average age in our series was 48 years. This is somewhat higher than in

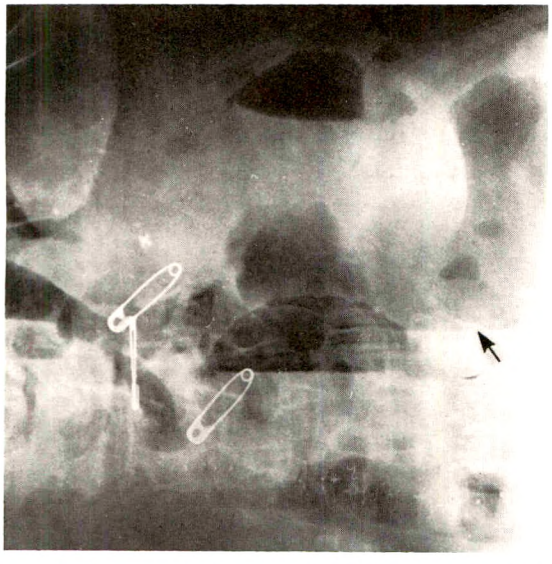


Fig. 8. Case IV. A 63 year old white male 36 days after total gastrectomy. The patient had low-grade fever and abdominal pain.

Decubitus roentgenogram of the abdomen reveals an air-fluid level in the right upper quadrant.

A multiloculated subphrenic abscess was subsequently drained.

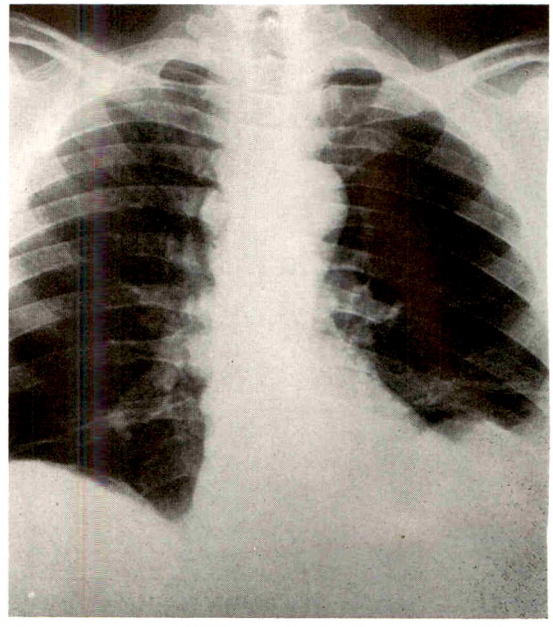


Fig. 9. Case V. A 61 year old Negro male with fever of unknown origin and abdominal pain.

Chest roentgenogram reveals elevation of the left hemidiaphragm with a small pleural effusion. Fluoroscopic study showed restriction of motion.

Ochsner and Graves' large series,[12] but it probably reflects the higher incidence of major surgery in older patients in recent years.

In several previous reports mention has been made of the increasing percentage of subphrenic abscess occurring in postoperative patients, but in none have the operations involved been tabulated, other than to state that the surgery most commonly involved is of the stomach and duodenum or biliary tract. In our series, subphrenic abscess frequently followed prolonged complicated procedures involving multiple organs (Table 1). Gastrectomy and splenectomy, especially when associated with other procedures, are high on the list of operations followed by abscess.

Of particular interest is the length of time transpiring between the initial operation and subsequent discovery of a subphrenic abscess in the postoperative patient. This varied from 5 to 131 days with an average duration of 34 days. Unexplained fever or leukocytosis, even as long



FIG. 10. Case v. Upper gastrointestinal series reveals downward displacement of the gastric fundus, an irregularity of the fundal shadow.

Exploration showed a left subphrenic abscess secondary to a perforated appendix. No abscess was found on the right.

as several months after surgery, should make one suspect the possibility of subphrenic abscess.

Mortality of an undrained abscess is two times that of abscess following surgical drainage,[6] making recognition and drainage of abscess extremely important. Roentgenologic findings and clinical experience sometimes suggest a subphrenic process in an occasional patient who subsequently improves on antibiotics alone and does not require surgery. This patient quite likely has a subphrenic *cellulitis* which does not progress to abscess formation. Such patients emphasize the importance of an intensive trial of antibiotic therapy prior to surgical drainage of any suspected abscess. On the other hand, utilization of antibiotics undoubtedly masks the presence of subphrenic collections in some instances and causes a delay in diagnosis. Therefore, after an adequate trial, it may be necessary to ultimately withhold antibiotics to confirm the presence of an abscess that requires drainage.

The over-all mortality rate of 37.5 per cent in our series is indicative of the severity of this disease. Surprisingly, this mortality rate has decreased very little over the past 40 years. In 1921, Lockwood[10] reported a mortality rate of 42.8 per cent in his own series, and a mortality rate varying from 33 to 56 per cent in other series. Ochsner and Graves[12] reported a 32 per cent mortality in their personal series of 50 cases. Comparison is not completely appropriate, however, because the majority of these patients develop subphrenic abscess following a ruptured viscus. Our own mortality rate for patients following ruptured viscus was 20 per cent. This shows some improvement over the years, but is still indicative of the serious nature of the problem.

Our group shows a slightly greater incidence of left-sided subphrenic collections than right. This is unlike most other collected series. Ochsner and Graves[12] found that 70 per cent of their cases were on the right side. Wetterfors[15] found a preponderance of right-sided abscess, 2 to 1 over left-sided abscess, and Moore[11] found all of his 10 cases on the right side. The higher incidence of left-sided abscess in our series may reflect the frequency of gastric surgery at this institution.

Roentgenologic findings as an aid to diagnosis have been emphasized in a number of reports. LeWald[9] and Pancoast[13] contributed early articles which emphasized the elevated diaphragm and the presence of an air-fluid level in the abscess. Dineen and McSherry[7] felt that the diagnosis was established by roentgenologic examination approximately 80 per cent of the time. Carter and Brewer[6] noted that every patient whom they had fluoroscoped showed positive findings about the diaphragm.

*Retrospective review of our 48 cases showed at least one positive roentgenologic sign in every instance.* Preoperative and antemortem diagnosis of subphrenic abscess was suggested in only 38 of the 48 cases (79 per cent), however. This could well have been higher with a higher index of suspicion of this disease.

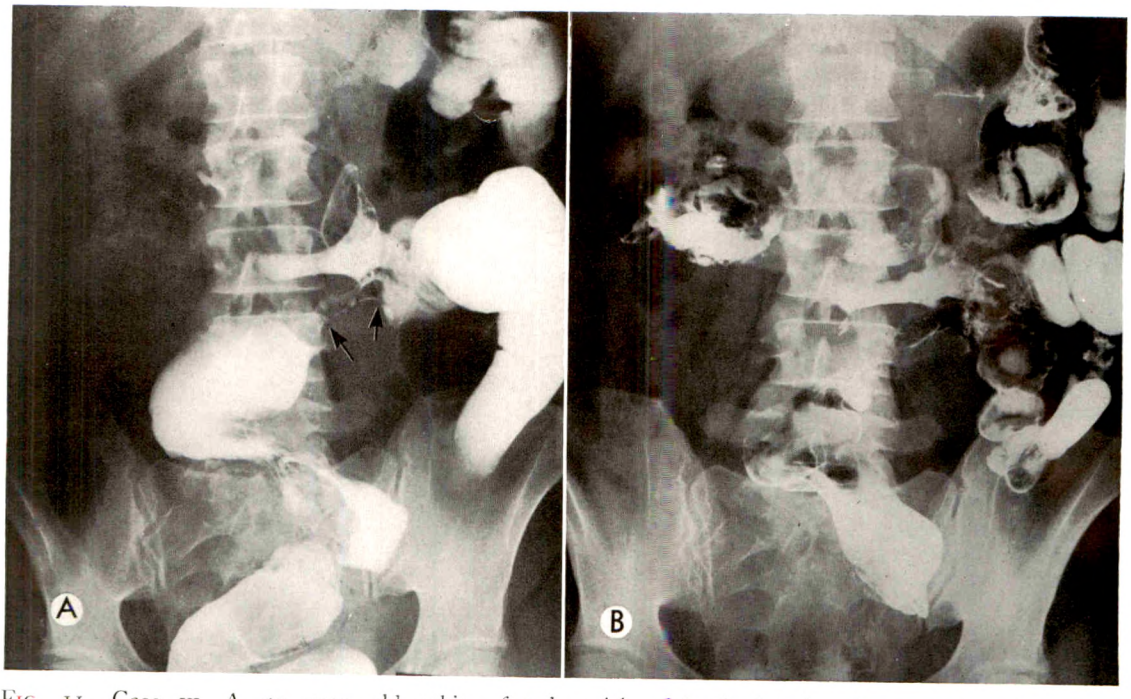



FIG. 11. Case VI. A 50 year old white female with abdominal pain, fever, and weight loss. (*A* and *B*) Barium enema examination reveals a carcinoma of the sigmoid which had perforated and created a subhepatic abscess on the right.

The most frequently present finding in subphrenic abscess is the reactive change about the diaphragm and pleura. Pleural effusion may be attributed to dilatation of the minute pleural vessels resulting in an out-pouring of transudate or to spread of infection through the diaphragm via the diaphragmatic lymphatic vessels. Atelectasis is quite likely the result of compression of the lung by an elevated diaphragm, and basilar pneumonia can result from poor ventilation of the lower lobe above an elevated diaphragm with retention of infected secretions.

Pleural effusion was seen in 79 per cent of our cases. This compares with 89 per cent reported by Wetterfors,[15] 90 per cent by Moore,[11] and only 33 per cent by Dineen and McSherry.[7] Recognition of a small amount of fluid blunting the posterior costophrenic sulcus on the lateral film, and confirmation by decubitus roentgenograms, will certainly raise the percentage of pleural effusions found. In 1933, Ochsner and Graves[12] felt that pleuritis was the result of late diagnosis of subphrenic abscess, and would not be present with early diagnosis. Our series, and more recent series, suggest that this is an early finding. Better roentgenologic detection methods and increasing awareness of the importance of pleural effusion has undoubtedly caused greater recognition of this finding.

Elevation of the hemidiaphragm was present in 95 per cent of our cases. This sign is not always of great aid because there may be elevation of one or both hemidiaphragms after abdominal surgery uncomplicated by subphrenic abscess.

Limitation of motion of the hemidiaphragm on the side of the abscess was present in 33 of 36 patients fluoroscoped. It is interesting to note that 2 of the 3 patients who did not exhibit limitation of the diaphragmatic motion had subhepatic abscesses. The diaphragm was fixed in 19 of 36 patients. This finding is a very strong indication of subphrenic collection. Fluoroscopy of the diaphragm is not generally done in the routine postoperative

chest examination; however, any clinical or roentgenologic evidence suggesting the presence of a subphrenic collection makes fluoroscopy mandatory.

Pneumonia or atelectasis was present in 33 of 47 cases. This particular feature often tends to obscure the real diagnosis by misleading the radiologist to suspect pneumonia or pulmonary infarct and to miss the primary condition. *Changes occurring about the diaphragm and the lung bases in the postoperative period should always alert the radiologist to the possibility of a subphrenic process.*

Thirteen of 48 patients (30 per cent) exhibited air in the abscess cavity. Thorek[14] postulated 3 possible sources of air in the abscess: (1) communication between the abscess and a hollow viscus; (2) communication between the abscess and a bronchus; and (3) infection with gas-forming organisms. Wetterfors[15] lists another possibility: (4) air admitted at the time of operation which is subsequently loculated. When present, loculated extraluminal air is positive proof of an abscess cavity. This was found in 41 per cent of patients by Wetterfors,[15] 37.5 per cent by Carter and Brewer,[6] and only 15 per cent by Ochsner and Graves.[12] This finding may be overlooked and should be searched for diligently. In 2 of our 13 patients, air in the abscess was originally missed but was discovered on subsequent roentgenograms.

Displacement of intra-abdominal viscera was seen in 17 of the 48 patients (35 per cent). Wetterfors[15] found displacement of viscera in 32 per cent of his patients. A higher incidence of this finding is present in left-sided collections and would presumably have been even higher in our current study if more barium examinations had been done. An upper gastrointestinal series is extremely helpful in determining displacement of viscera in the left upper quadrant and to a lesser extent, in the right upper quadrant. Whenever subphrenic abscess is suspected, upper gastrointestinal and barium enema examinations should be routinely considered.

One abscess was demonstrated by fistula injection. Carter and Brewer[6] emphasized the importance of injection of any fistulous tracts which are present. Unfortunately, fistulous tracts may not appear at all, and sometimes appear only when the patient is moribund.

Asch[2] pointed out the value of pneumoperitoneum in the diagnosis of suprahepatic abscess. Our own experience with this procedure is limited to 4 cases. In every instance, this was helpful but the diagnosis had already been strongly suspected by other studies. One instance of a false-positive pneumoperitoneum was encountered where exploration of the right upper quadrant failed to disclose an abscess. This patient had had multiple previous surgical procedures in the right upper quadrant, and obliteration of the subphrenic space was apparently due to adhesions.

Combined liver and lung scanning has proved helpful to some in the diagnosis of subphrenic abscess.[3,5] It may be an adjunct in localizing the anterior and posterior extent of the abscess, as suggested by Bodon and Holzwasser.[3]

Ariel[1] has suggested that radioactive iodinated albumin may be picked-up in an abscess in its formative stage. This is not helpful in right-sided abscesses, and we have no personal experience with it.

The importance of repeated roentgenologic examination cannot be overemphasized. Many of the patients who exhibited no roentgenologic signs early in the course of the disease subsequently developed a full-blown picture of subphrenic abscess. Repeated chest roentgenograms should be made, including overpenetrated Bucky roentgenograms and decubitus roentgenograms, when indicated. Upper gastrointestinal studies and barium enema examinations can be performed simply and easily, and may be invaluable in establishing the diagnosis. Fistula injection is mandatory when a fistulous tract is present. Pneumoperitoneum may confirm the diagnosis and combined liver and lung scans may be helpful in localizing the abscess. The most im-

portant factor in the diagnosis of subphrenic abscess is an alert radiologist with a high index of suspicion for this disease.

SUMMARY

Forty-eight cases of subphrenic abscess have been reviewed and their roentgenologic findings analyzed.

The important signs are (1) reactive changes in the lungs, pleura, and diaphragm; (2) air in the abscess cavity; and (3) displacement of intra-abdominal viscera.

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# BARIUM ENEMA EXAMINATION IN THE EVALUATION OF LARGE PELVIC MASSES*

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IN A recent paper on large pelvic masses[1] the author described a new sign which had been noted on the barium enema examination in many cases of large myomatous uteri. This sign consisted of flattening and straightening of the superior contour of the apex of the rectosigmoid in the filled frontal roentgenogram. Since that study, we have had the opportunity to examine additional cases of myomatous uteri and have noted this sign in a total of 17 cases.

As a part of this continuing study, the barium enema findings in other commonly seen large pelvic masses, the ovarian mass and the distended bladder, have been evaluated, with emphasis on the profile lateral studies.

Little has been written on the findings in barium enema examinations performed for evaluation of such masses. Taveras and Golden[7] stated that, while all lower abdominal intraperitoneal tumors displaced loops of small intestine and sigmoid, there were no specific features about the displacement of these loops which would permit a distinction between different types of tumor. Marshak[6] described no specific differential features between compression of the sigmoid by myomatous uteri and ovarian tumors, except in the case of ovarian malignancies which invade the rectosigmoid. Khilnani *et al.*[4] further elucidated the roentgen features of extracolonic malignancies invading the rectum and sigmoid. Levene and Bragg[5] described the normal anterior mobility of the rectosigmoid after evacuation, which is prevented by the presence of a pelvic mass.

The purpose of the author is to describe the significant findings in such cases on barium enema examination, emphasizing the value of the lateral roentgenogram, with an attempted postulation of the mechanisms involved.

## MATERIAL AND METHODS

All cases studied had barium enema examinations performed, including lateral roentgenograms before and after evacuation. Twenty cases of uterine myomata, 30 cases of ovarian masses and 10 cases of distended bladder in the female constitute the material for the present investigation. For inclusion in this evaluation, the mass had to be of sufficient size on roentgenography to extend into the abdominal cavity to the level of the 5th lumbar vertebra. Small, completely intrapelvic masses may be difficult to detect on roentgenograms and their effect on adjacent structures may be minimal. The larger masses, *i.e.* those which fill the pelvic cavity and extend into the abdomen, are more easily detected and evaluated roentgenographically. Clinical evaluation may be somewhat difficult because of the problem of separating these masses by manual palpation from the uninvolved pelvic structures.

All the uterine myomata and ovarian masses had proved diagnoses by laparotomy and pathologic study. The operative and pathologic findings were also studied from the standpoint of the shape, location and fixation of the masses. The diagnosis of distended bladder was proven by catheterization with disappearance of the mass. In the cases evaluated, the marked bladder distention was neurogenic or psychogenic in origin. Ten of the ovarian masses were non-neoplastic cysts, 10 were neoplastic cysts and 10 were ovarian malignancies. All the myomata were benign on histologic study.

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ANATOMIC AND PATHOLOGIC DISCUSSION

The roentgen terminology of the distal intrapelvic large intestine has been somewhat confusing. This has been due to the lack of precision in determining the junction between the rectum and the sigmoid colon on the barium enema examination. For this reason, the portion of the intrapelvic large intestine passing posteriorly and inferiorly along the upper portion of the sacrum has been referred to as the rectosigmoid.[3,5,6] This terminology was used in our previous paper on pelvic masses.[1] In a recent paper, Clark *et al.*[2] have shown that the rectosigmoid junction determined sigmoidoscopically and marked by clips is usually located on barium enema examination at the 4th segment of the sacrum, at which point, there is noted in the lateral view, a change in direction of the bowel from posteroinferior to anteroinferior. For greater precision, therefore, in describing this portion of the large intestine, the bowel which follows the proximal curve of the sacrum will be called the distal sigmoid (Fig. 1).

The true pelvis is a space limited in volume due to its surrounding bony walls. When the rectum and sigmoid are distended with barium, they tend to displace any space occupying pelvic structures into the distensible abdominal cavity. Pelvic masses will be thus displaced with the type and extent of the mass and its fixation in the pelvic cavity. Since the pelvic inlet delimits the narrowest pelvic diameters, it is at this level that the effect of the mass passing from the pelvis into the abdomen will be most evident. On the lateral barium enema roentgenogram, the severity and configuration of this compression will depend on the anteroposterior diameter of the pelvic inlet, the prominence of the sacral promontory, and the size, shape, consistency and mobility of the mass.

Of the 3 pelvic organs from which arise the masses described in this report, the bladder is the one with the greatest normal intrapelvic fixation. Anatomically, it is extraperitoneal in location with only its

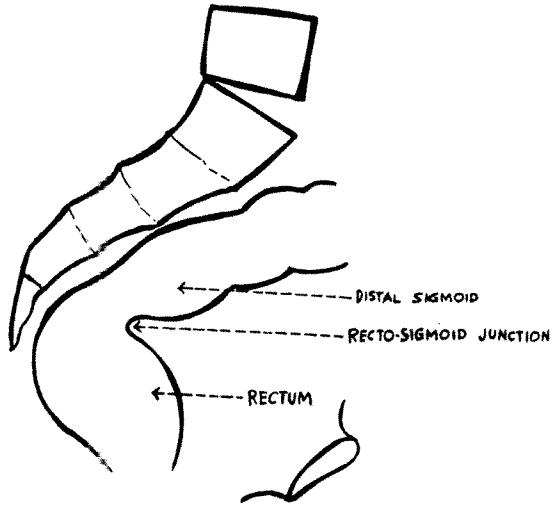



Fig. 1. Diagrammatic representation of the intrapelvic large intestine—based on Clark *et al.*[2] Note that the rectosigmoid junction is located at the level of the 4th sacral segment. The bowel which follows the proximal curve of the sacrum is labeled the distal sigmoid.

superior surface and a short segment of its inferoposterior surface covered by peritoneum. It is enveloped by vesical fascia and anchored inferiorly to the pelvic floor by the urethra.

The fixation of the uterus in the pelvic cavity allows for greater mobility than does the fixation of the bladder. The uterus is also located more superiorly lying above and behind the bladder. It is predominantly an intraperitoneal organ anchored laterally by the broad ligaments, which consist of a broad double sheet of peritoneum extending from the lateral surface of the uterus outward to the pelvic walls. It is also anchored by the round ligaments, the uterosacral ligaments and the uterovesical ligaments.

The ovary is the most mobile of the pelvic organs discussed. It is a completely intraperitoneal structure fixed only to the uterus by the ovarian ligament and to the pelvic wall by the infundibulopelvic ligament.

ROENTGEN FINDINGS

The markedly distended bladder because of its firm pelvic fixation remained

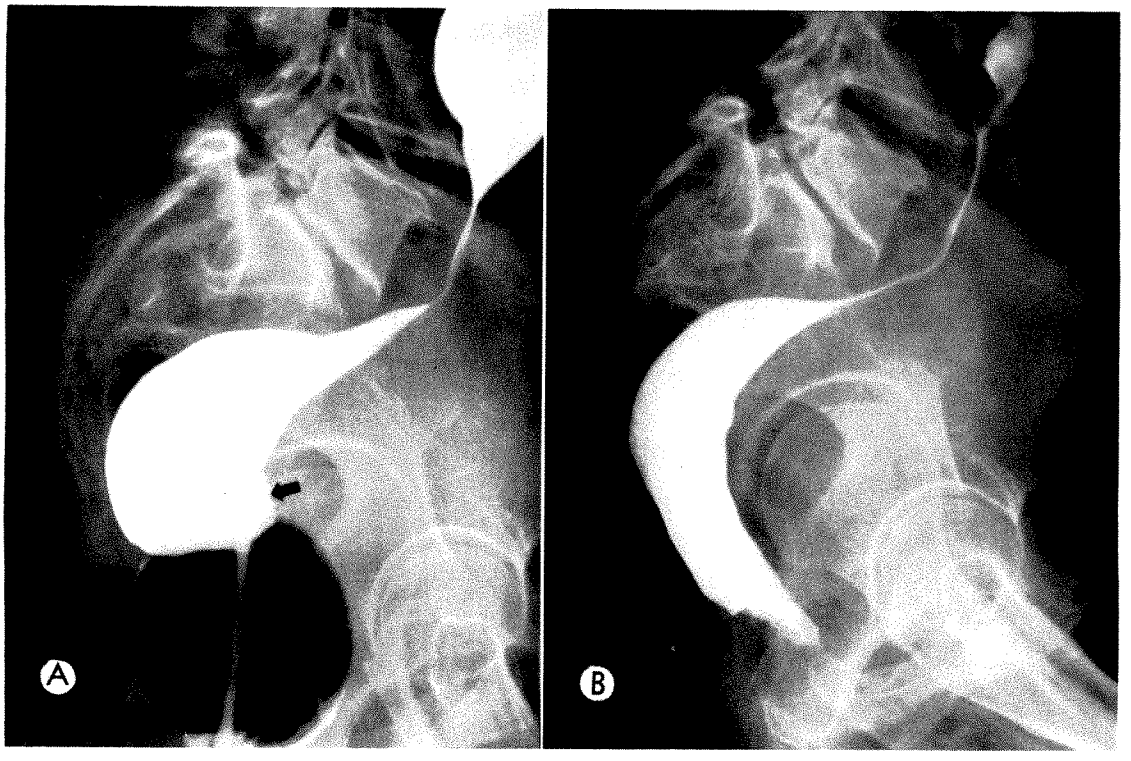



FIG. 2. Distended bladder in the female. (*A*) Barium enema roentgenogram. There is only slight flattening of the rectum in the portions not distended by the balloon catheter (arrow). The distal sigmoid is severely compressed, especially at the pelvic inlet. This is accentuated by the spondylolisthesis present in this case. (*B*) Post evacuation roentgenogram. Note the increased compression of the rectum following removal of the balloon and partial evacuation of the barium.

partially intrapelvic. However, since it was fluid-filled, it could change in shape to accommodate itself to extrinsic pressure. During the filled stage of the barium enema examination, distention of the rectum was only slightly prevented by the dilated bladder, apparently because of shift in its fluid content as well as minor displacement of the bladder into the abdomen. More pressure on the anterior wall of the rectum and distal sigmoid than produced by other pelvic masses was usually detectable, probably related to the lower position of the bladder in the pelvic cavity and its fixation inferiorly by the urethra. At the pelvic inlet, where the sagittal diameter was the smallest, the bladder severely indented the anterior aspect of the sigmoid as it passed into the abdomen (Fig. 2*A*).

After evacuation, the dilated bladder filled the pelvic cavity flattening and compressing the rectum and distal sigmoid against the sacrum, eliminating their normal post evacuation anterior mobility (Fig. 2*B*). In the abdomen, the sigmoid was displaced either superiorly or to the left.

The large myomatous uterus retained fair mobility unless adhesions fixed it to the intrapelvic structures. In the filled stage of the barium examination, the mobile myomatous uterus was displaced toward the abdominal cavity. For description of its effect on the distal sigmoid in the lateral view, the cases could be divided into 2 categories: (1) those in which the myomata arose from the fundus of the uterus and extended into the abdomen; and (2) those with more diffuse uterine enlargement. In the first category, about one-third of this series of cases, there was no effect of the mass on the rectum or distal

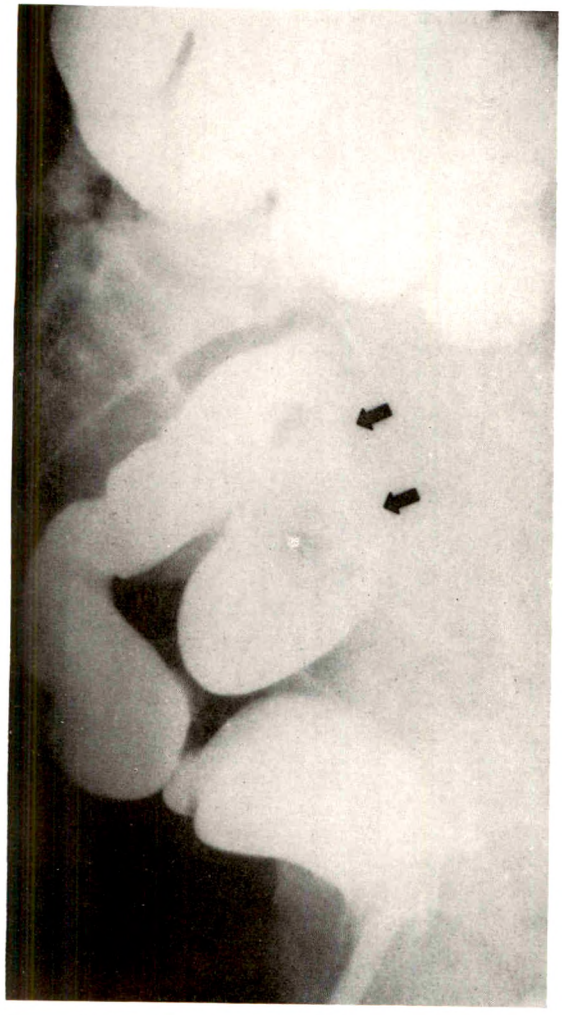


Fig. 3. Myomatous uterus with the myomata arising from the fundus of the uterus and extending into the abdomen (Category 1). Note the segment of the midsigmoid depressed into the pelvic cavity and flattened from above (arrows).

sigmoid in the lateral view, but a segment of the midsigmoid was usually pushed into the pelvic inlet and compressed from above. This appearance in the lateral roentgenogram appeared to be fairly characteristic for this type of myomatous uterus and was not seen in the intra-abdominally located ovarian masses (Fig. 3). In the second category, about two-thirds of this series of cases, there was slight flattening of the anterior contour of the distal sigmoid at the pelvic inlet (Fig. 4*A*).

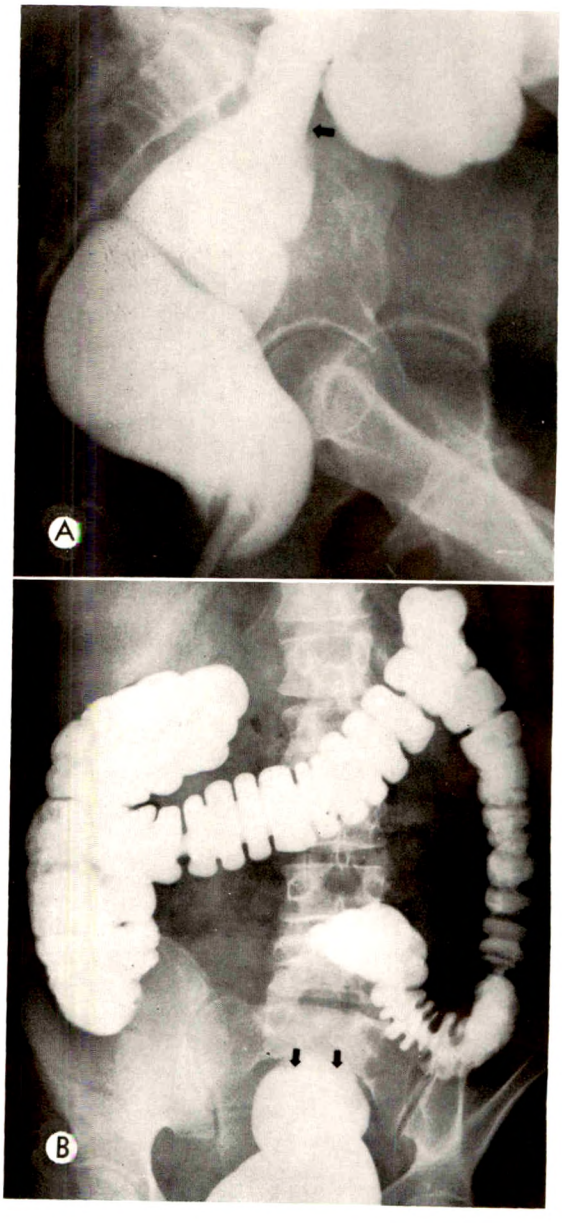



Fig. 4. Myomatous uterus with diffuse enlargement (Category 2). (*A*) Lateral roentgenogram. There is flattening of the anterior contour of the distal sigmoid at the pelvic inlet (arrow). The rectum and the remainder of the distal sigmoid are unaffected. (*B*) Frontal prone roentgenogram. Note the marked flattening of the superior aspect of the distal sigmoid (arrows). Comparison of the frontal and lateral roentgenograms indicates that this flattening is due to a vise-like effect on the sigmoid entrapped between the mass above and below the sacral promontory.

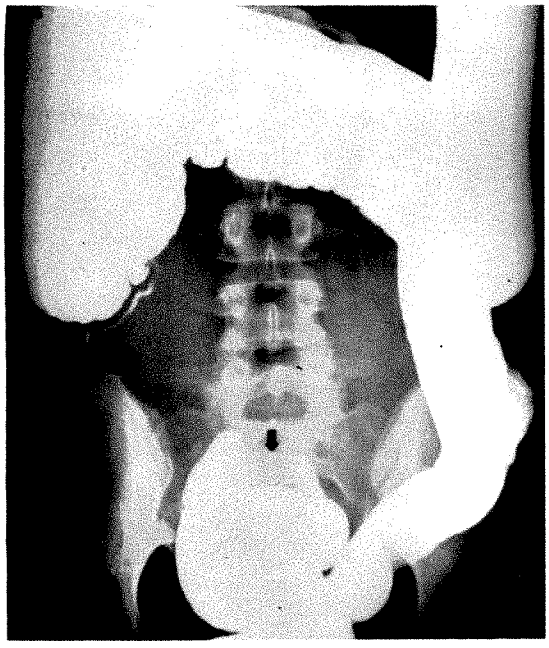


FIG. 5. Myomatous uterus extending to the level of the 1st lumbar vertebra. Frontal supine roentgenogram. Note the marked extrinsic pressure and flattening of the superior aspect of the sigmoid (arrow). The midsigmoid is depressed.

However, in both types of cases, as the mass extended into the abdomen, it was pulled inferiorly by its attachment to the intrapelvic structures. This apparently produced rather characteristic compression of the superior aspect of the distal sigmoid as it exited from the pelvic cavity (Fig. 4*B*; 5). The multilobulated nature of the masses appeared to aid in causing this flattening, since it led to the production of a vise-like effect upon the sigmoid entrapped between the portion of the mass above and below the sacral promontory. This flattening was seen in 17 of the 20 cases of myomatous uteri. The cases in which it was not present usually showed symmetric smooth uterine enlargement. It was not seen in any of the ovarian masses or cases of distended bladder. The sign when present thus appears to be fairly specific for the large myomatous uterus. In the previous study[1] this sign was described as being seen in the supine frontal roentgenogram. A few subsequent

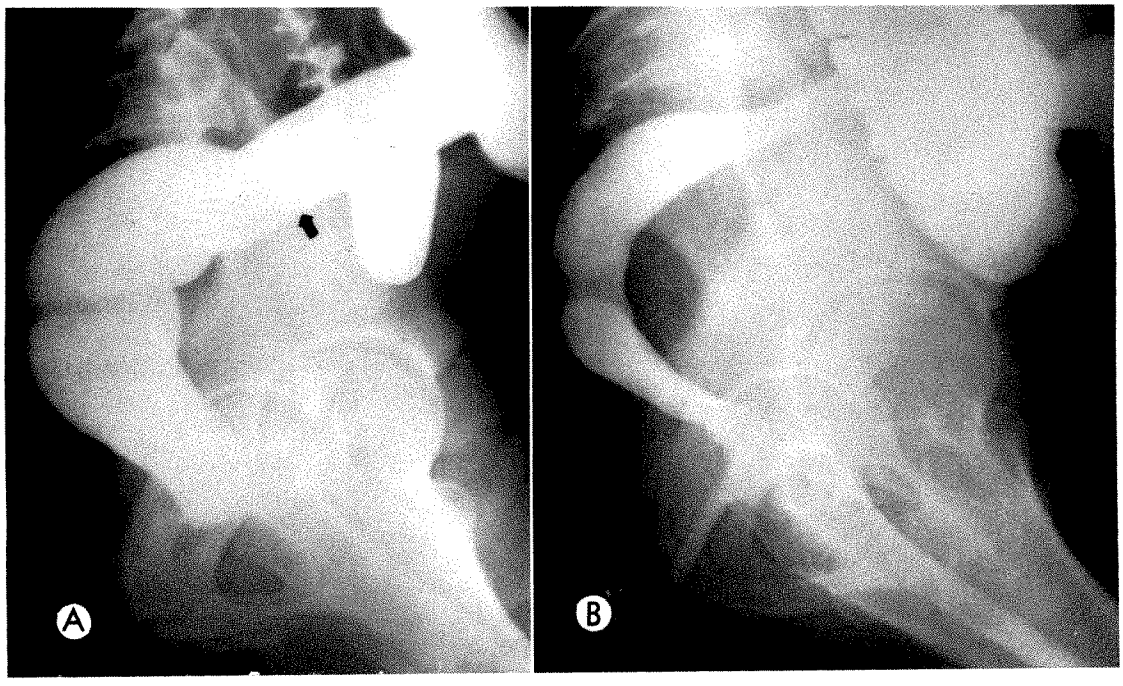



FIG. 6. Myomatous uterus with diffuse enlargement. (*A*) Barium enema roentgenogram. There is only minimal flattening of the anterior wall of the distal sigmoid at the pelvic inlet (arrow). (*B*) Post evacuation roentgenogram. Note the increased compression of the distal sigmoid resulting from return of the mass to the pelvic cavity. This prevents the normal post evacuation anterior mobility of the distal sigmoid.

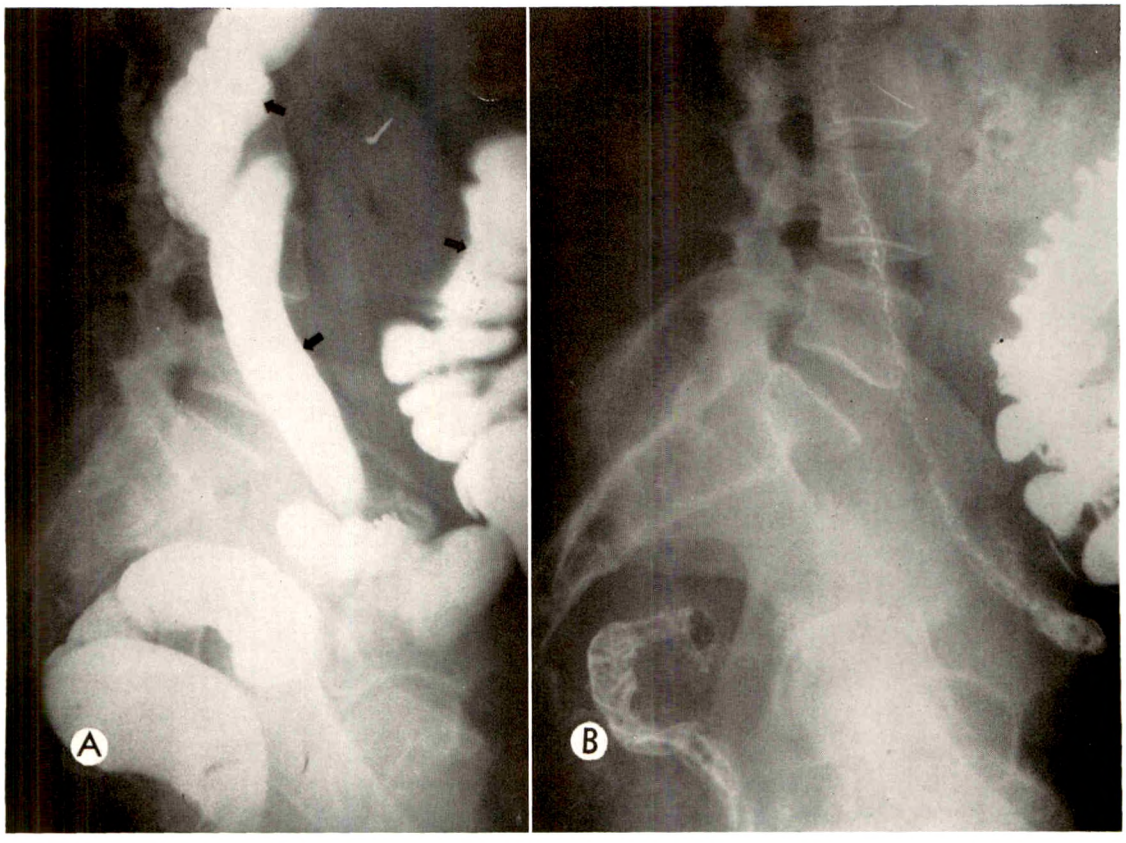



FIG. 7. Large serous cystadenoma of the ovary. (*A*) Barium enema roentgenogram. The mass is completely intra-abdominal in location and does not affect the rectum and distal sigmoid. Note the intra-abdominal compression of the descending colon and ascending colon (arrows). (*B*) Post evacuation roentgenogram. The mass remains intra-abdominal and does not prevent the normal anterior mobility of the distal sigmoid.

studies, however, also demonstrated it in the prone frontal roentgenogram, so that both positions of the patient are suggested for its demonstration.

After evacuation, increased compression of the rectum and distal sigmoid by the myomatous uterus was seen (Fig. 6, *A* and *B*). This again varied with the shape and structure of the mass, depending upon the size of its intrapelvic portion. When the myomatous uterus was fixed in the pelvic cavity by adhesions, or when myomata were impacted into the pelvic cavity, greater sigmoid compression was evident.

Intra-abdominally, the smaller masses, that is those extending to the level of the 4th lumbar vertebra, elevated the sigmoid or deviated it to the left (Fig. 4*B*), while the larger masses, that is those extending above the 4th lumbar vertebra, depressed the sigmoid (Fig. 5). This sigmoid depression was most evident in masses with a predominant intra-abdominal component, in which the midsigmoid was occasionally considerably depressed into the pelvic cavity.

Because of their large size and relative mobility, most of the benign ovarian masses in this series were completely intra-abdominal in location. They therefore had no effect on the rectum or distal sigmoid both on the filled and post evacuation roentgenograms (Fig. 7, *A* and *B*). A few dipped into the pelvic cavity at the pelvic inlet, flattening the anterior aspect of the distal sigmoid, especially on the post evacuation roentgenogram (Fig. 8).

Marked compression of the rectum and



FIG. 8. Large serous cyst of the ovary. Lateral post evacuation roentgenogram. The mass dips into the pelvic cavity after evacuation and produces moderate compression of the sigmoid at the pelvic inlet (arrow).

distal sigmoid indicated either invasive malignancy or infarction with fixation of the mass in the pelvic cavity. The roentgen features of malignant invasion of the rectum and sigmoid were, as described by Khilnani *et al.*:[4] (1) nodular contour defects, (2) limited extensibility with fixed thickened transverse folds, and (3) mucosal ulceration (Fig. 9). The infarcted masses showed severe compression with larger nodular contour defects (Fig. 10).

None of the ovarian masses produced the flattening of the apex of the distal sigmoid seen with myomata. The deviation of the intra-abdominal sigmoid depended upon the size of the mass as described for the uterine myomata.

## DISCUSSION

The barium enema examination is frequently requested by clinicians as part of the work-up of large pelvic masses. The information usually reported by the radiologist is the presence or absence of invasion of the colon by a suspected ovarian malignancy.

This study indicates that additional useful information is provided by the barium enema examination, especially if lateral roentgenograms of the rectum and sigmoid are obtained. Demonstration of flattening and straightening of the superior contour of the distal sigmoid in the frontal view strongly suggests the diagnosis of a myomatous uterus. This is further suggested by the presence of a mobile mass with both intrapelvic and extrapelvic components, whose effect on the rectum and sigmoid is more pronounced in the post evacuation lateral roentgenogram. The distended bladder produces more severe

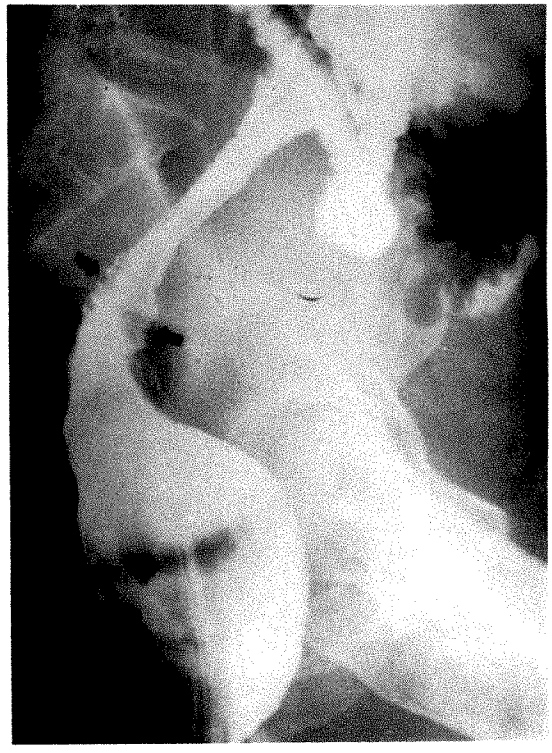


FIG. 9. Ovarian adenocarcinoma. Lateral barium enema roentgenogram. There is severe compression and narrowing of the proximal rectum and distal sigmoid. Note the limited extensibility with small nodular contour defects and fixed thickened transverse folds (arrows).



Fig. 10. Infarcted ovarian cyst. Lateral barium enema roentgenogram. The distal sigmoid is severely compressed. Note the large contour defects on the anterior wall of the distal sigmoid (arrows).

compression due to its greater intrapelvic fixation as well as more rectal compression due to its lower pelvic position. The large benign cystic ovarian masses tend to be completely intra-abdominal in location with little effect on the intrapelvic colon. Marked rectal and sigmoid compression by a large ovarian mass suggests pelvic fixation due to invasive malignancy but is occasionally associated with an infarcted benign mass.

Additional useful information is provided by the plain roentgenogram and intravenous pyelogram. The findings in these studies have been analyzed previously.[1]

SUMMARY

The findings on barium enema examination were evaluated in a series of commonly seen large pelvic masses—the myomatous uterus, the distended bladder and the ovarian mass. The masses studied showed significant extension into the abdominal cavity. The study emphasizes the utility of the lateral roentgenogram in evaluating the mobility or fixation of the masses and the probable pelvic organ from which they originate. A sign suggestive of the large myomatous uterus—flattening of the superior contour of the distal sigmoid in the frontal projection—is discussed and its probable mechanism postulated.

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# A SIMPLIFIED CANNULATION TECHNIQUE FOR LYMPHANGIOGRAPHY*

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ALTHOUGH lymphangiography as developed by Kinmonth and associates[3] has evolved into a clinically useful procedure, its performance by the radiologist remains rather tedious and time-consuming.

A modification in the cannulation technique used to perform lymphangiography on 137 patients will be described here because it has helped to reduce markedly the radiologist's time in performing this procedure.

The radiologist's time can be reduced to that necessary to (1) explain the procedure to the patient, (2) inject the blue dye solution, and (3) dissect and cannulate a lymph duct. The most time-consuming aspect of the procedure for the radiologist is the accomplishment of leakproof cannulation, and it is to this aspect of lymphangiography to which recent reports on modification of technique have been directed.[1,2,4,5] An assistant* can be trained to (1) assemble equipment and instruments, (2) prepare the skin of the patient's feet with antiseptic solutions, and, most important, (3) comfort and observe the patient while the contrast medium is being injected by the Hobb's injector. During the injection of contrast medium (usually a 3/4 to 1½ hour period), the radiologist can be working elsewhere in the department where he can be immediately summoned by the assistant should any untoward reactions occur.

## TECHNIQUE

The lymphangiography technique used is that of Kinmonth and associates[3] as it is currently utilized,[6,7] but with the following modifications of the cannulation technique

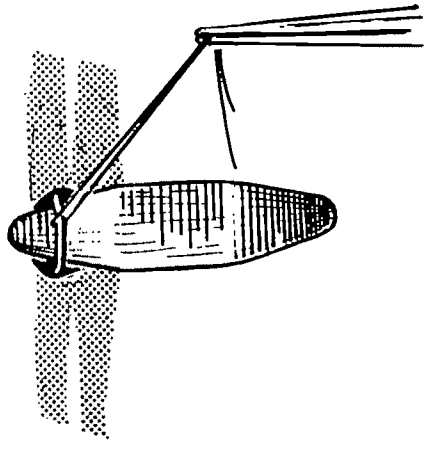


FIG. 1. A thin metal blade is inserted beneath the lymph duct to support it at the level of the skin surface before cannulation.

(Fig. 1 to 4): A flat area on the dorsum of the foot is the site chosen for the skin incision. This site can be either above or below the hump located above the first metatarsal-tarsal joint. The flat surface will allow the cannulating needle to lie in direct alignment with the lymph duct to be cannulated (Fig. 2*A*). A thin metal blade has been designed for the purpose of supporting the lymph channel at the level of the skin surface (Fig. 1 to 4). These blades can be made from a Davis Retractor† and have dimensions in the order of 1 1/6 by 1/4 by 1/20 inch. The ends are tapered from the maximal width of about 1/4 inch. Although the blade need not be of these exact dimensions, it should be of a width that allows insertion beneath a lymph duct even when a very small incision has been made, and yet wide enough to support a segment of lymph duct of sufficient length to allow cannulation with the needle. The

* My thanks to Mrs. Mildred Jorgenson for her able assistance.

† Obtained from V. Mueller and Co.

* Presented at the Meeting of the Minnesota Radiological Society, Minneapolis, Minnesota, January 28, 1967.
From the Mayo Clinic and Mayo Foundation: Section of Roentgenology.

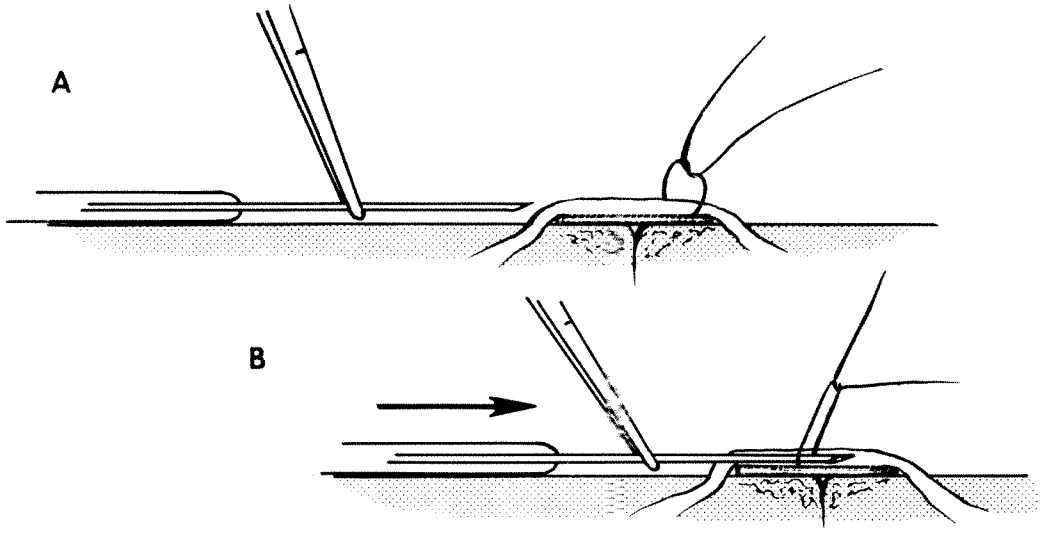



Fig. 2. (*A*) If a flat skin surface has been chosen for the incision site, the cannulating needle can be stabilized by holding it with an eye needle holder flat against the skin. It will be in direct alignment with the lymph duct when the latter is supported by the thin metal blade. (*B*) To cannulate, with the needle held pressed against the skin to minimize or eliminate unwanted motion, it need only be advanced proximally to puncture the lymph duct.

most important feature of the blade is that it be extremely thin so that the duct is supported at the level of the skin surface, where it will thus be held in direct alignment with the needle to be used in cannulation (Fig. 2, *A* and *B*). If the blade is too thick, angulation of the needle can occur during cannulation and contribute to the development of a leak. The blade is malleable and bends slightly to conform to the curvature of the dorsum of the foot. Ordinarily the blade does not need to be secured in position beneath the lymph duct, but occasionally sterile tape* is used to hold it in position. A small needle holder allows the cannulating needle† to be held flat against the skin and in direct alignment with the lymph duct, which is supported by the metal blade (Fig. 2, *A* and *B*). It is important to use a needle holder such as the Paton eye needle holder, which allows the needle to be released in a motionless fashion once it has been inserted into position within the lymph duct lumen. The needle can be held with the fingers, but it is easier to hold the needle flat against the skin and in direct alignment with the lymph duct by use of a needle holder.

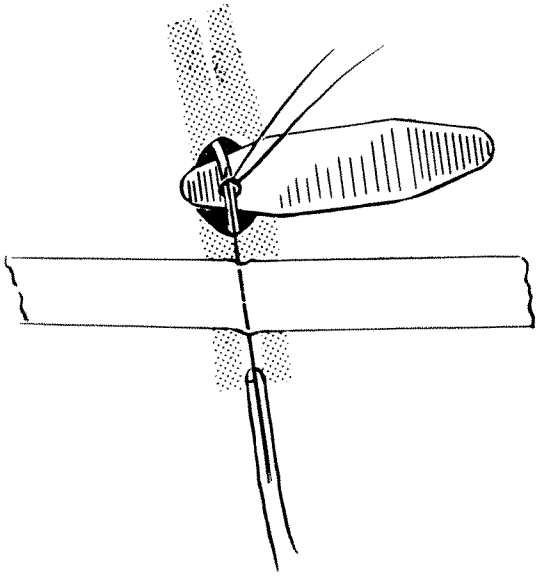


Fig. 3. Once the needle has punctured the lymph duct, which is supported by the metal blade, it can be stabilized in this position by use of sterile tape. Both hands are then free to tie the needle in position by a ligature placed around the lymph duct before cannulation.

* 3-M Steri Strips are available from Minnesota Mining & Manufacturing Co., St. Paul, Minnesota.

† 27 G needles on plastic tubing as sterile disposable lymphangiography sets are made by Becton Dickinson Co., Rutherford, New Jersey.

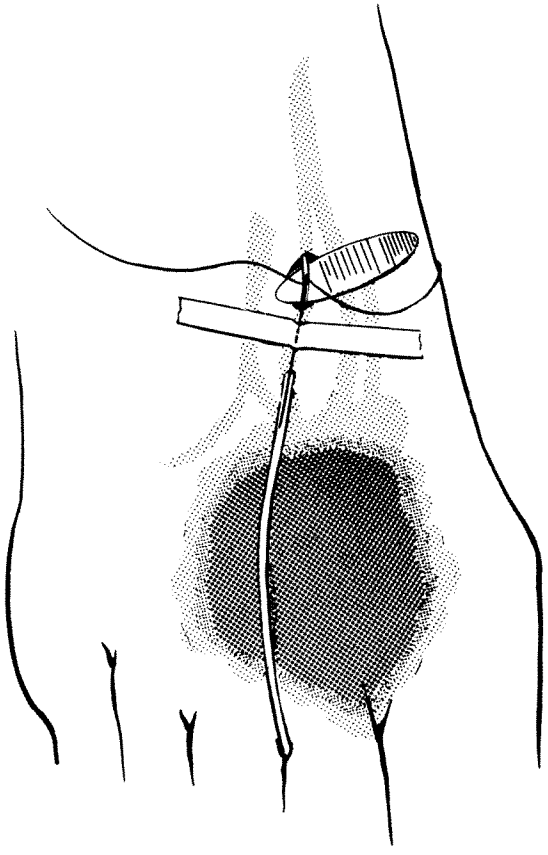


FIG. 4. Appearance of lymph duct after the needle is tied in position at the completion of cannulation.

To cannulate, the needle need only be advanced proximally until the tip can be seen to lie within the lumen of the lymph duct (Fig. 2, *A* and *B*). Magnifying glasses aid in inserting the needle so that the tip can be directed into the center of the lumen of the lymph duct. Holding the needle pressed against the skin to stabilize it during cannulation minimizes or eliminates unwanted motion that can cause leaks. Once the needle lies within the lumen, one hand is free to use sterile tape to secure it in position. The needle can be released from the needle holder without movement; both hands are then free to tie the needle securely by use of a No. 6–0 silk ligature previously placed around the lymph duct to complete the cannulation procedure (Fig. 3 and 4).

SUMMARY

In this simplification of the technique for lymphangiography, a thin metal blade can be used to elevate the lymph duct to the level of the skin surface. If a flat skin surface has been chosen for the incision site, the cannulating needle can be held flat against the skin surface, where it will be in direct alignment with the lymph duct. In this stabilizing position, the needle need only be advanced proximally to insert it into the lumen of the lymph duct. Sterile tape is used to hold the needle in this position so that both hands are free to tie the needle in place.

This modified technique has considerably reduced the radiologist's time needed to cannulate lymphatic ducts for lymphangiography.

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# MOBILE RADIATION PROTECTION BARRIER FOR USE IN ARTERIOGRAPHY*

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RADIATION exposure to the hands is a potentially serious problem to the radiologist or surgeon performing numerous arteriographic procedures, utilizing hand injections. This is particularly true in procedures where the hands are likely to be close to the radiation field as in neuroradiologic or selective abdominal procedures. In some of these procedures, if at all possible, mechanical injectors should be employed in preference to hand injections; however, in many cases mechanical injectors are not felt to be sufficiently safe for routine use. We have found the use of lead gloves, lead ceiling aprons, and other such devices unnecessarily cumbersome and have instead devised a mobile lead protection barrier which can be easily constructed, allows complete freedom of the hands, and is extremely effective in reducing radiation exposure.

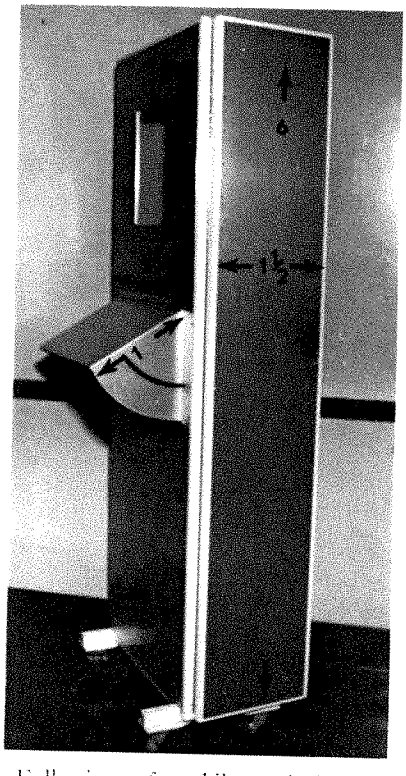


FIG. 1. Full view of mobile radiation protection barrier. Hinged hood in partially open position.

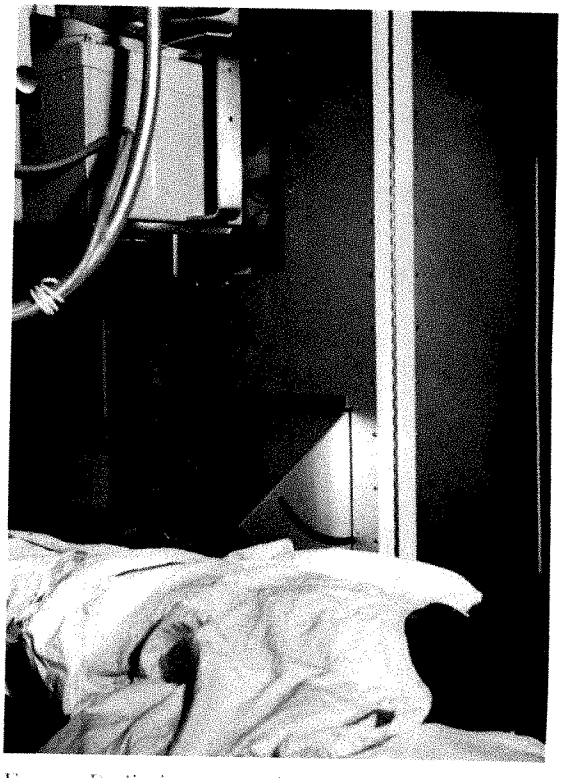


FIG. 2. Radiation protection barrier in use during pelvic arteriography. The hands are shielded by hinged hood during filming.

## DESIGN AND CONSTRUCTION

The 6 × 2½ foot vertical barrier is constructed in three layers, a front surface of .091 inch sheet aluminum mounted on .062 inch lead which is affixed to a .250 inch pressdwood base (Fig. 1). There is a 1½ foot wide hinged side extension of the vertical barrier providing protection from

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lateral scatter. The barrier has a 1×1¾ foot hinged hood extending 1 foot beyond the barrier when in a fully opened position of 90°. This allows the hood to be extended close enough to the injection area so that the hands can be safely covered by the hood, at the same time, allowing unencumbered manipulation of the syringe (Fig. 2). A 5/16 inch leaded glass port allows visualization of the patient during the injection procedure. The barrier is mounted on swivel casters allowing good mobility and is made quite stable by two 11 lb. lead counter weights on the base. Recorded exposure doses to a pocket dosimeter placed on the outside of the hood have ranged from 10 to 60 mr during various types of selective procedures with serial rapid filming in pelvic or head regions. Exposure dose to the underside of the hood in the position of the hands, as recorded by pocket dosimeter has been 0. The radiation protection provided by the mobile barrier has made it an extremely important and useful piece of equipment in our angiographic laboratory.

SUMMARY

A simply constructed mobile radiation protection barrier, shielding the hands and body of the operator during arteriographic procedures utilizing hand injections, is described.

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We wish to thank Allen Dannenbrink, R. T., Kenneth Duncan, Fergus T. O'Foghluda, Ph.D. and Thomas W. Nooney, M. S. for aid in the design and construction of the mobile radiation barrier.

VOL. 101, No. 4

# RADIATION BEAM ANALYZER FOR DIAGNOSTIC RADIOLOGY*

*By* E. M. HIGGINS,† HAROLD D. ROSENBAUM, M.D.,‡
*and* WALDO D. DEVORE, M.S.§
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MOST radiology departments have several diagnostic x-ray machines which are used interchangeably. It is highly desirable that these machines be calibrated so any particular set of technical factors produces a roentgenogram of identical density and contrast on all machines within the department. Present calibration methods employ voltage and current indicating meters. These meters, in conjunction with factory-supplied data, are used to make the initial calibration of pkv. or ma. This does not mean that two generators calibrated according to factory specifications will have the same output since variation in output can be caused by a number of factors. These factors include line, transformer and x-ray tube characteristics, which are not controlled in the calibration procedure. Kilovoltage variations among x-ray machines in a department must be minimized not only because of the resulting variation in quality of the beam but also because of the variations in radiation output. The latter is more important because output is approximately a function of the square of the voltage.

While it is ideal to calibrate all machines in terms of absolute values of pkv. and average milliamperage, it is more important that output of various generator-tube combinations be uniform. An error in measurement of pkv. may be acceptable provided the same error is operative on all units.

Fortunately, the slope of the straight line portion of the Hurter-Driffield curve exhibited by x-ray film is such (2 to 3.5) that a small change in exposure does not significantly change the film density.[5] The resulting exposure latitude makes it possible for several generators within a diagnostic department to vary somewhat in output and still produce films of diagnostic quality. Variations among x-ray machines will, however, reduce the latitude of exposure settings which result in acceptable films. This factor tends to increase the number of repeated examinations.

## METHOD OF CALIBRATION

With these considerations in mind, it seemed desirable to develop an instrument which would measure parameters relevant to the attainment of uniform output from different x-ray machines. Such an instrument should be easily transportable from room to room, should display the information concerning the radiation beam in easily interpretable form and should be simple to operate. If the distance from the x-ray tube target to the radiation detector is standardized, four variables must be considered: pkv., ma., time and filtration.

A fixed amount of aluminum filter is usually added to the inherent filtration of each x-ray tube. Consequently, the filtration variable can be assessed by inspecting the added filtration and by consulting the manufacturer's data regarding inherent filtration in the tube and collimator. The remaining three factors (pkv., ma., and time) must be measured by the instrument.

Several factors prevent a digital readout system, based on integration of the area under a curve (for output) together with a pulse height readout (for pkv.) from giving

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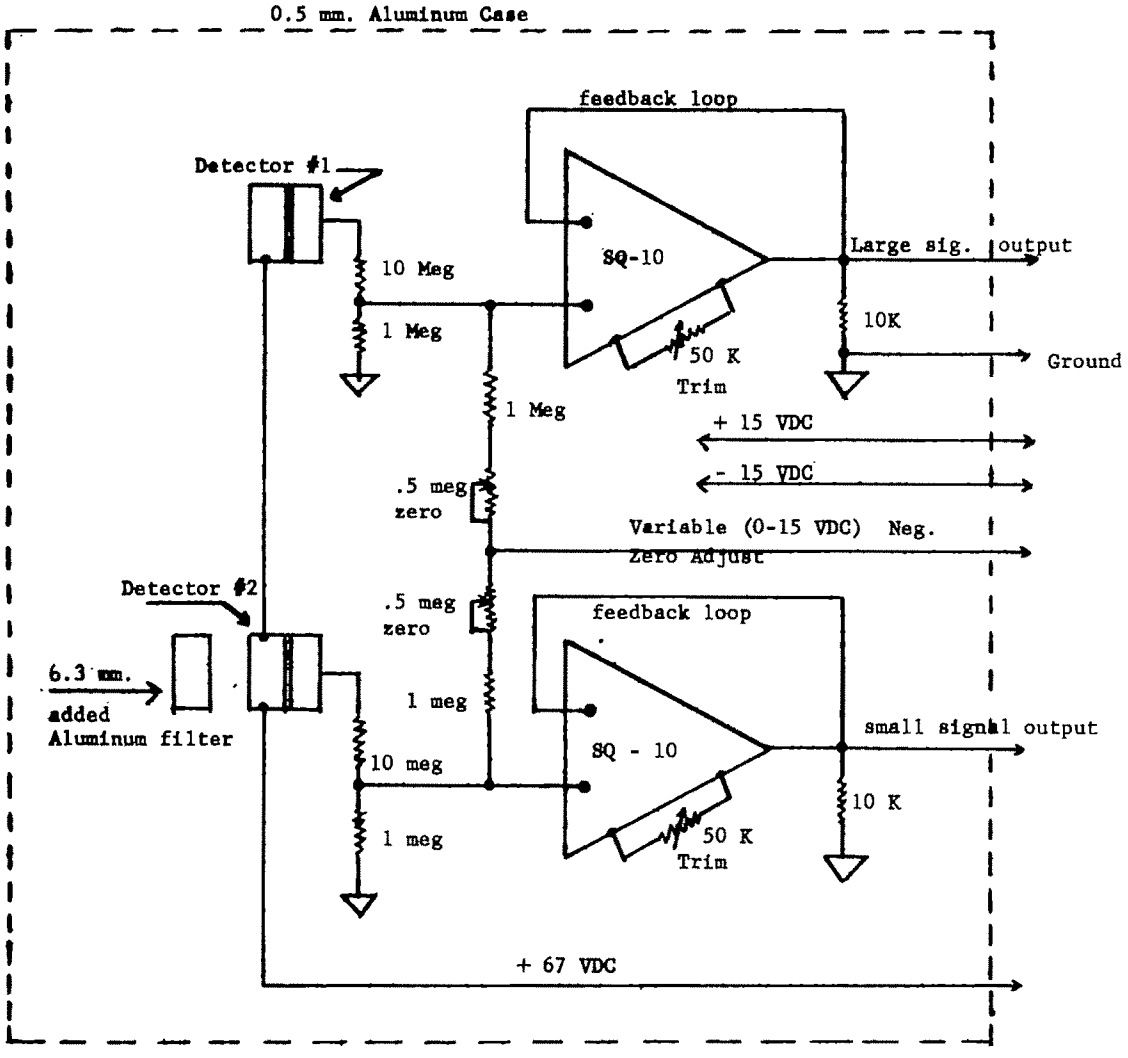



FIG. 1. Schematic diagram of solid state detector assembly.

adequate information. Peak kilovoltage and voltage waveform do not necessarily remain the same from one impulse to another. Milliampere stabilizer difficulties[3] can enter into a moderately long exposure. Contactor bounce may become an appreciable problem on short exposures. Finally, timer variations from one exposure to the next, which cause considerable variation in output, cannot be detected without examination of several consecutive exposures. The need for additional information obtained through the use of an oscilloscope was immediately apparent.

The resulting instrument consists of three sections: the solid state detector preamplifier assembly, the power supply section and the readout section.

Newell and Henry[4] demonstrated that absorption of radiation in a known thickness of aluminum filter could be used to measure peak generating voltage. This concept can also be utilized with solid state detectors. Lithium-ion-drift solid state detectors[2] have a depletion layer hundreds of times thicker than ordinary solid state semiconductor radiation detectors and, accordingly, have a much greater sensitivity to energies in the diagnostic range. As radiant energy is absorbed in the depletion layer of these

devices, their resistance is decreased. Accordingly, these detectors can be used to develop an electrical signal proportional to the absorbed radiant energy.

Two detectors are placed adjacent to one another in the beam with an aluminum filter modifying the radiation incident on the active surface of one of the detectors. The quiescent impedance of each detector is 10 megohms; therefore, a voltage divider (10–1 megohm string) was inserted in the load circuit of each detector to reduce the spurious 60 cycle signal which is almost unavoidable with such high impedance. The signal developed at the 1 megohm load resistor is coupled directly into an operational amplifier* connected as a voltage follower (Fig. 1). The signal-to-noise ratio thus obtained is in the order of 100/1. Depending upon the distance from the source to the detector, a signal ranging from 0.1 volt for low pkv., low ma. radiation to 2.5 volts at high pkv. and high ma. can be developed. These values contrast favorably with the micro- and millivoltages produced by most radiation detectors. The infinite feedback loop used in the operational amplifier provides extremely linear, consistent, and distortion-free amplification of signals. Provision has been made for the individual zeroing of each operational amplifier. Output with no radiation signal is at ground potential. Direct coupling throughout avoids shift of the baseline in the output signal and minimizes frequency loss.

According to manufacturer's specifications, lithium-ion-drift detectors have a response time in the order of nanoseconds† and are, therefore, able to follow easily the 60 cycle sine wave envelope of the radiation. No provisions are shown for balancing the output of the two detectors before amplification, as no appreciable difference in output between the two was noted. We are uncertain whether these detectors are inherently closely matched.

* Cat. #SQ-10a, Nexus Research Laboratory, Inc., Canton, Massachusetts.

† Cat. #5×5-LID-1000-100A, Solid State Radiations, Inc., Los Angeles, California.

Radiation produced at low pkv. is strongly absorbed in the aluminum filter resulting in a large difference in intensity registered by the filtered and unfiltered detectors. As the peak generating voltages are increased the amount of absorption in the aluminum filter becomes proportionally less. A 6.3 mm. aluminum filter ahead of one detector reduces the registered intensity of the filtered detector to approximately 20 per cent of the unfiltered detector reading at 30 pkv. At 120 pkv., approximately 65 per cent of the radiation is transmitted to the filtered detector.

The use of the instrument is simple and straightforward. It is not necessary to remove panels from the x-ray control or transformer. The detector is placed at a known distance from the tube target, exposure factors are selected, an exposure is made, and the signals displayed upon the oscilloscope photographed. A polaroid oscilloscope camera allows viewing of the photographs 10 seconds after the exposure.

Principal observations include the relative amplitudes of the filtered and unfiltered detector signals, the number of impulses in the exposure and irregularities, if any, in the wave shape. The time is determined by counting the pulses and by the decade readout tubes which show the number of impulses in any exposure. It is possible to select a time such as 1/20 of a second and make a dozen or more exposures in rapid sequence. If the timer is not varying, the counting tubes will show an even multiple of 6 impulses. The instrument is equipped with a reset switch so that even on relatively long exposures it is possible to make rapid repeat measurements by resetting the counter to zero between exposures, thereby checking the consistency of the timer. Series of exposures, such as are used with film changers which normally dictate exposure times of 1/20 second or less, can be checked for accuracy and consistency at high recycling rates.

Measurements of the relative amplitude of the signals are made on the graticule of the oscilloscope which is superimposed

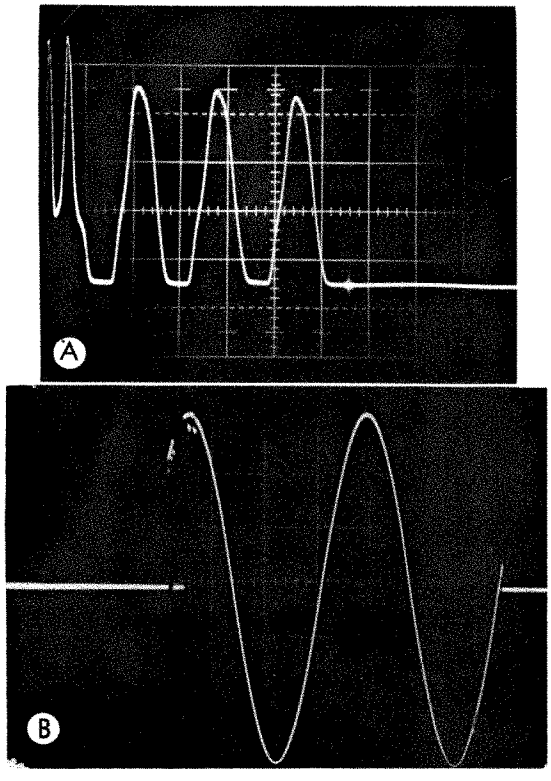



FIG. 2. (*A*) Effect of contactor bounce on radiation output. Radiation output is plotted on the Y axis. Time is plotted on the X axis. Each centimeter equals 5.0 milliseconds. (*B*) Voltage waveform applied to primary of high tension transformer showing voltage interruption due to contactor bounce.

upon waveforms of the two signals. The ratio of the amplitudes of the two waveforms (filtered to unfiltered) is used to calculate peak kilovoltage. Resolution is within one pkv. at midrange. The amplitude of either signal can be used to determine the milliamperage.

Initial calibration for pkv. is standardized using the "K" edge radiation from known absorbers.[1] Identical pkv., ma. and time settings result in identical waveforms if the output of the generators is, indeed, identical.

A slight bounce as the contactors close creates two separate peaks of different intensity in the first impulse of the radiation output (Fig. 2, *A* and *B*). These peaks are read as two timing impulses on our timing monitor section. Although this indication is false, it provides valuable information regarding the closure of contact points and indicates the need for immediate correction to prevent application of excessive surges to the x-ray tube.

An example of deficient valve tube filament emission is shown in Figure 3*A*, where roentgenographic output dropped with increased exposure time. The valve filaments were energized at the recommended setting of 6.0 volts rms. The percentage of transmission through the 6.3 mm. aluminum filter dropped to a barely measurable value

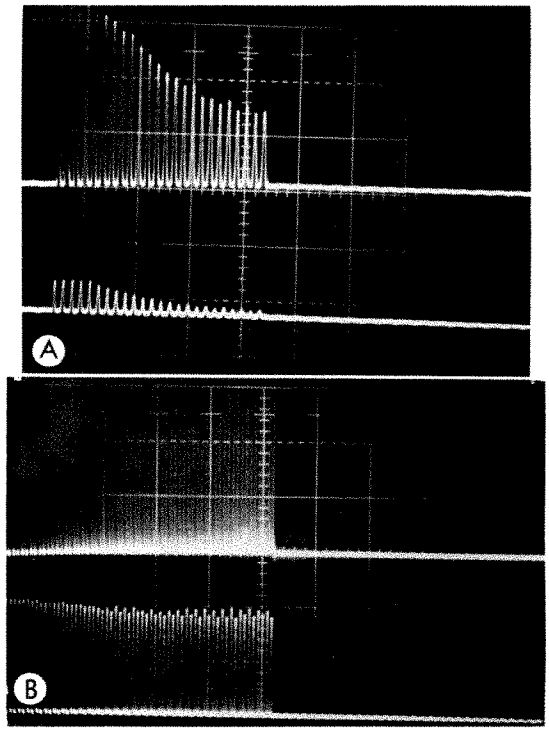



FIG. 3. Faulty valve filament energization. (*A*) Radiation output with thoriated tungsten filament set at 6.0 volts AC. Output decreases in quality and quantity as exposure progresses. Factors used: 300 ma, 40 pkv., 0.2 sec. and no added filter (mammography range). Each centimeter equals 0.05 seconds. Top trace is developed by detector with no additional aluminum filter. Bottom trace is developed by detector after radiation passes through 6.3 mm. additional aluminum. (*B*) Radiation output with same technical factors after valve filament voltage is increased to 6.4 volts AC. Each centimeter equals 0.1 second. Top trace is developed by detector with no additional aluminum filter. Bottom trace is developed by detector after radiation passes through 6.3 mm. additional aluminum.

even though the control milliameter and the transformer primary voltage read correctly. Figure 3*B* shows the improvement in output when the valve filament voltage was boosted to 6.4 volts rms.

Once the machine is properly calibrated and is without contactor bounce or time deficiencies, it is desirable to file a photograph of the waveform of a particular technique for service information. If at any time in the future the calibration or performance of the machine becomes questionable, the output can be re-photographed for comparison to see whether the deficiency is related to pkv., ma. or time.

SUMMARY

A method of x-ray machine calibration based on analysis of the emitted x-ray beam is described. An instrument has been developed and tested which measures pkv., ma., output waveform, and time. By examining the final product of the generator-tube combination, the radiation output, one has the best opportunity to detect alterations of the factors which may lead to significant change in the resulting radiation beam.

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# PANOGRAPHY*

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PHILADELPHIA, PENNSYLVANIA

PANOGRAPHY is a radiographic technique recently described by Blackman[1] for use predominantly in dentistry.[1,2] John E. Cullinan, Jr., chief radiologic technician at the Albert Einstein Medical Center of Philadelphia, suggested the possibilities of a wider field of usefulness, which we have explored with promising results.

The main departure from conventional roentgenographic equipment lies in the miniaturization of the x-ray tube and the ability of the extended anode of the tube to be placed into body openings and crevices to secure unique roentgenograms of structures heretofore not easily studied.

The miniature tube is part of a mobile, compact apparatus called "Panoramix."* It is 37 1/2 inches high, 31 inches long, and 21 inches wide (Fig. 1). The tube arm can be rotated 360° about the supporting column; the tube itself can be extended 6 feet from the column. The tube mount can be rotated 180° in both the horizontal and vertical planes.

The anode of the x-ray tube is composed of copper with a tungsten apex. It is set at the end of a rod which extends about 10 cm. from the tube head. Because this anode can be placed very close to the tissues, the energy required to expose adequate films is achieved with very low kilovoltages and with short exposure times—*i.e.*, on the order of 50 to 60 kv. and 0.1 mas. for nonscreen film, and less these amounts for screened film. Either nonscreen or fast films in flexible cassettes may be used. The focal spot of the tube is 0.1 mm.

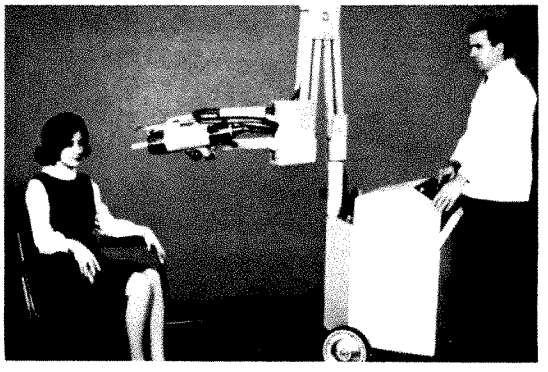


FIG. 1. The Panoramix equipment.

Panoramix roentgenography should not be confused with the laminography of the Orthopantomograph or the Panorex. The source of radiation from the anode of the Panoramix tube is held stationary during exposure, the beam extending outwards in all directions from within the body cavity or crevice into which the anode has been placed, such as the mouth, rectum, axilla, or joint fossa. In laminographic apparatus, the x-ray beam moves in a rotary fashion, sequentially exposing a rotating film.

The Panoramix x-ray beam emerges in a 270° angle in two planes and in a 360° angle in a plane perpendicular to the anode rod. Constant potential current is derived from a 120 volt AC line. The power supply can furnish up to 70 kv. in steps of 2.5 kv. each, and exposures can be obtained from 0.05 to 1 mas. in steps of 0.05 mas. The full capacity of the tube is 1 milliampere per second. Rectification is solid state and capacitors are filtered.

Because of the short film-focus distance and the wide divergence of the x-ray beam, Panoramix radiography depicts structures with exceptional clarity and with striking

* Westinghouse Electric Corp., X-ray Division, Baltimore, Md.

* Presented at the Sixty-eighth Annual Meeting of The American Roentgen Ray Society, Washington, D. C., September 26–29, 1967.
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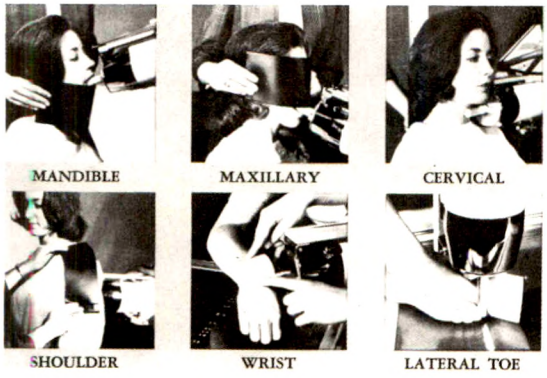



Fig. 2. Examples of some methods of application of the Panoramix apparatus.

enlargement of detail. The wide angle roentgenograms which are achieved with this equipment, and the magnification it affords, add dramatic dimensions to roentgenography in areas of the body not readily accessible before (Fig. 2). The following examples indicate the range of some of its clinical applications.

## ILLUSTRATIVE CASES

### DEMONSTRATION OF BONES OF FACE AND ALL TEETH BY SINGLE EXPOSURE

Case 1. An 82 year old man developed looseness of a dental bridge on the left side of the mandible. A single panographic examination projected all the teeth of the maxilla and the mandible. A fracture of the lower left second molar at the level of the metallic cap was seen (Fig. 3*B*). The anode portion of the x-ray tube for this examination was inserted into the mouth through a central perforation in the film, which encircled the face (Fig. 4). By comparing the Panoramix roentgenogram (Fig. 3*B*) with

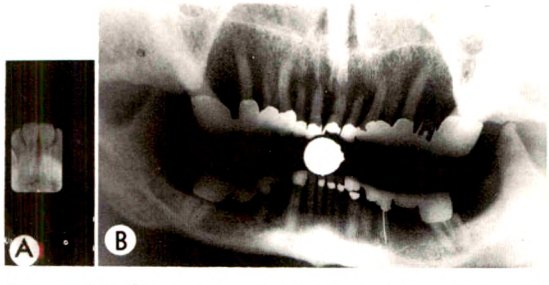



Fig. 3. (*A*) Conventional dental roentgenogram. (*B*) Panoramix view of entire mouth. A fracture of the lower molar on the left side is seen just below the metallic crown.

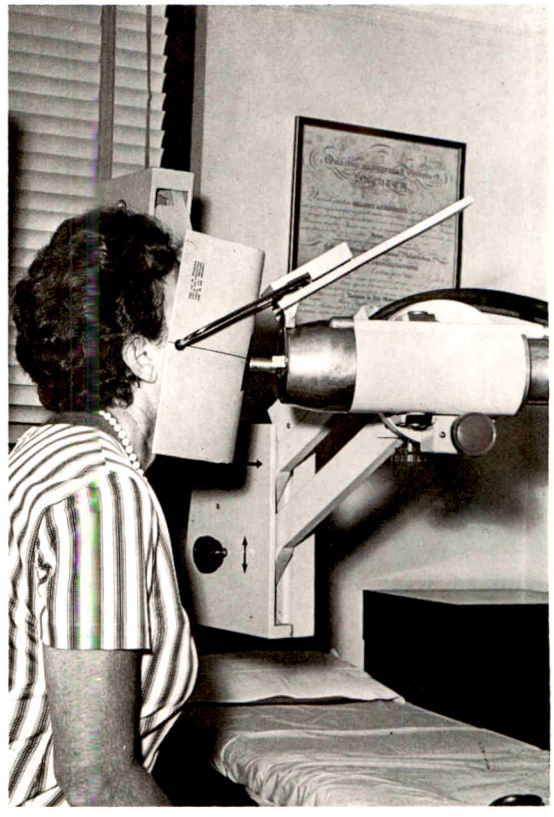


Fig. 4. The anode is inserted through a perforation in an 8×10 inch film encircling the front of the face.

the conventional dental roentgenogram (Fig. 3*A*), it can be seen that the size of the teeth is automatically magnified about twice, while all the teeth in the maxilla and mandible are visible in this singly exposed film. No superimposed shadows or significant distortion have occurred.

Case 11. A 47 year old male developed a painless swelling of the right side of his jaw and face. Panoramix examination disclosed the entire mandible, revealing an expanding cystic

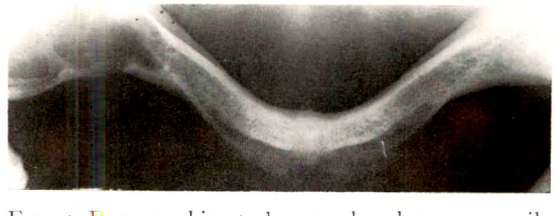


Fig. 5. Panographic study reveals a large expansile cystic lesion in the right mandible, proven to be a dental cyst.

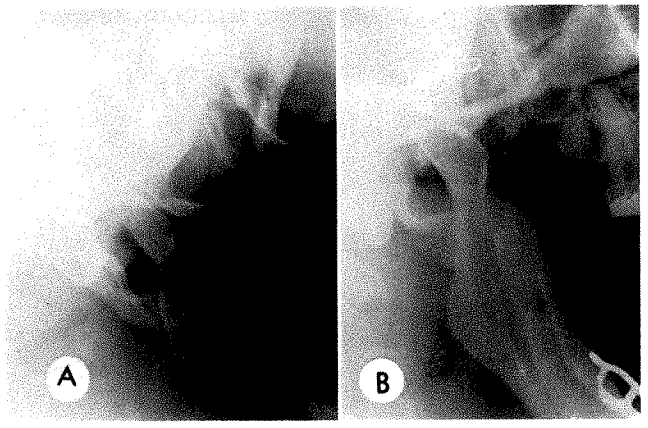



FIG. 6. Panoramix views of a normal temporomandibular joint with the mouth slightly opened (*A*) and fully opened (*B*).

lesion (Fig. 5). Operation uncovered a large, simple dental cyst, whose pathologic nature was confirmed by histologic study.

CASE III. A 73 year old housewife complained of pain in the region of the right temporomandibular joint. The partially opened joint was examined with the Panoramix unit, using intensifying screens and Parspeed x-ray film. Excellent detail can be seen in the ascending ramus, the coronoid and condyloid processes of the mandible, and the structures of the normal temporomandibular joint (Fig. 6, *A* and *B*).

ANATOMIC FOSSAE AND INTERSPACES

CASE IV. A 26 year old male fell on his outstretched hand and immediately experienced pain in the region of the right elbow. A conventional roentgenogram disclosed a rather typical fracture of the radial head (Fig. 7*A*). The Panoramix examination clearly identifies the slight tilt of the lateral fragment, with excellent magnification of detail (Fig. 7*B*).

CASE V. A 45 year old male had a history of intermittent locking of the knee joint, with repeated effusions. Conventional roentgenography did not reveal the osteochondromatous bodies adjacent to the medial tibial spine nearly so well as did the magnified Panoramix view (Fig. 8).

CASE VI. A 53 year old male was referred for a painful shoulder, which proved to be due to adhesive capsulitis. A Panoramix study was done so as to exclude the possibility of roughening and irregularity of the bicipital groove. The examination was made by placing the Pano-

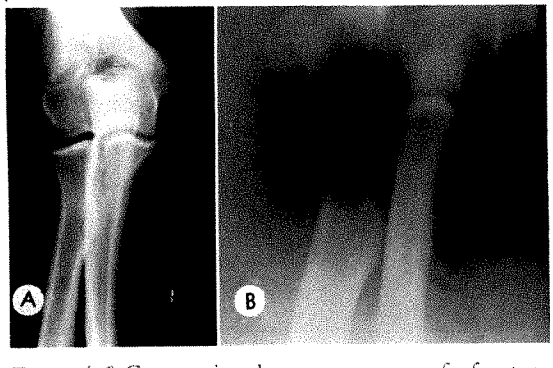



FIG. 7. (*A*) Conventional roentgenogram of a fracture of the radial head. (*B*) Panoramix view of same area, showing magnification of detail.



FIG. 8. Osteochondromatous bodies in knee joint, not readily seen with conventional roentgenography, but quite evident in Panoramix view.

ramix anode in the axilla and wrapping the film about the shoulder. The resultant exposure was obtained with considerably more ease than the conventional roentgenograms of this area (Fig. 9).

INTERSPACE PROJECTIONS BETWEEN SMALL JOINTS

Case VII. An old, ununited fracture of the proximal phalanx of the left index finger was treated surgically with insertion of a bone graft and fixation by two metallic pins (Fig. 10*A*). The Panoramix study demonstrates the graft and the bone architecture with amazing clarity (Fig. 10*B*). The bone graft is seen on the lateral aspect of the fracture.

DISCUSSION

The conception of an intra-oral source of radiation is by no means new, having been conceived simultaneously in 1897 by Rollins in the United States and by Bouchacourt in France, according to Blackman.[1] However, in those days, bulky gas tubes with focal targets too large to produce a sharp image were used. Consequently, very little progress was made until the mid 1940s, when discovery of a large range of radioactive isotopes made renewed experimentation feasible. The results obtained were promising, but isotopes were costly, they were relatively short-lived, and the radiation risks were forbidding.

About 1938, industry found uses for long-rod anode radiographic tubes in the examination of tubular constructions such as the pipes of a steam boiler or the cylinder bores of an internal combustion engine. These x-ray tubes were constructed so that an arm extended from the center of the x-ray bulb, with the anode target placed remotely at the free end of the extension. The Chaoul radiotherapy tubes, of a similar design, followed and were used for rectal and vaginal irradiation. The Panoramix tube is similar to the Chaoul therapy tube, except that it has an extremely fine focal area and gives a 270° angle of radiation divergence in two perpendicular planes.

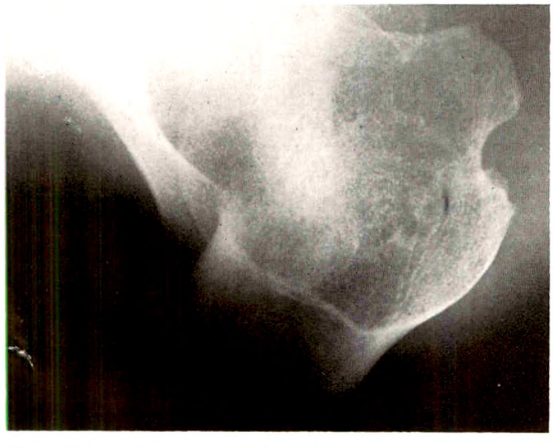


Fig. 9. Striking view of the bicipital groove, easily obtained with Panoramix technique.

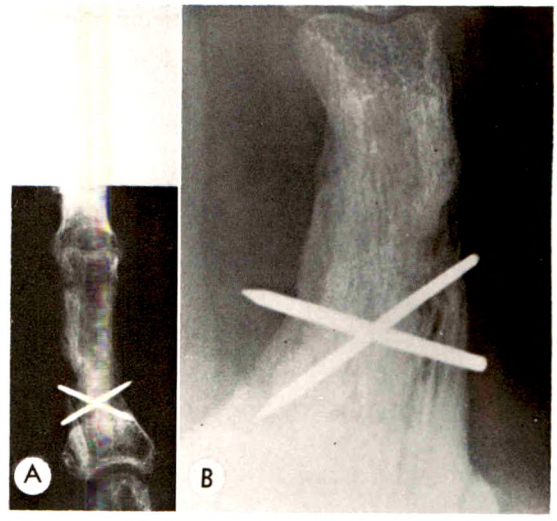



Fig. 10. (*A*) Conventional and (*B*) Panoramix views of old fracture of the proximal phalanx of the left index finger following bone grafting and fixation by two metallic pins.

The radiation dose to the patient's tissues has been carefully measured and found to be well within safe limits. In many cases, it is less than the dose required for a corresponding x-ray examination. The apparatus has been inspected by the U. S. Bureau of Standards, and the radiation output has been carefully measured by several investigators, among them personnel at the Jefferson Medical College of Philadelphia, the University of Pittsburgh, and Sir Sidney Blackman of the Royal Dental Hospital of London.

Using measurements made 4.7 cm. from the tube axis at 70 kv., 0.47 mas. exposure,

and at points 3 cm. and 5 cm. from the end of the tube, the half-value layer was calculated to be approximately 2.1 mm. Al and 2.5 mm. Al. The proximity of the anode to the patient's tissues results in an exposure dose of 1 to 1.4 rad with the average exposure time of 0.1 mas. It is estimated that the total absorbed energy to tissues from a single intra-oral exposure is less than that of a conventional exposure. For example, instead of the 10 to 14 single exposures usually necessary for a full mouth dental roentgenographic study one Panoramix examination, using less radiation, furnishes a diagnostic roentgenogram of all the teeth, as well as the structures of the maxilla and mandible.

When the apparatus is used to explore other parts of the body, the energy absorbed by the tissues in securing a diagnostic roentgenogram is of the same order as that necessary to obtain conventional roentgenograms, the difference being essentially that of the distribution of the energy. This energy is naturally greater in tissues close to the anode of the tube than in those more distant.

As with any radiation, due regard for the safety of the patient and the operator must be maintained. A metal shield on the anode rod can be positioned so that virtually all radiation except that which emanates in the desired direction is excluded. Since the principal radiation hazard is posed by the beam which emerges in a 360° arc, the operator should take the precaution of remaining behind a protected control panel and should, as an additional safety measure, wear a protective apron when the tube is angulated away from the axis parallel to the floor. When the anode rod is to be inserted into any body cavity, a light-weight rubber finger cot makes a satisfactory and hygienic disposable cover.

SUMMARY

Radiologic apparatus called "Panoramix" is described.

The unique feature of Panoramix is a miniature x-ray tube with an extended anode, 10 cm. long and 15 mm. in diameter, which can be placed into body cavities and crevices. Its use in obtaining new projections of the bones of the face, pelvis, hips, anatomic fossae, and small bones and joints is explained and illustrated. For example, all the bones of the face, including all the teeth in the maxilla and mandible, and the temporomandibular joints can be portrayed by a single exposure. Yet the tissue absorption of radiation is less than that obtained with 14 dental roentgenograms. In addition, the increased detail afforded by this apparatus in spite of the image magnification augments information gained from conventional studies.

Our experience so far indicates a promising field of usefulness for this compact, mobile, and flexible roentgenographic equipment.

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VOL. 101, NO. 4

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The Fiftieth Annual Meeting of the Society will be held at the Hotel Fontainebleau in Miami Beach, Fla., April 7–11, 1968.

## EDITORIAL

# THE SIXTY-EIGHTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Sixty-eighth Annual Meeting of the American Roentgen Ray Society was again held at the Washington Hilton Hotel, Washington, D. C., from Tuesday, September 26 through Friday, September 29, 1967.

At 8:30 A.M. Tuesday, President Clyde Alan Stevenson of Spokane, Washington, called the meeting to order and introduced Henry Dunlop Ecker, M.D., President of the Medical Society of District of Columbia, who welcomed the Society to our nation's capital, a frequently chosen site of the meeting. Dr. Ecker called attention to the fact that this year's meeting coincides with the 150th anniversary of the founding of the Medical Society of District of Columbia and in warm words invited the members and guests of the American Roentgen Ray Society to visit the new modern headquarters of this historically important organization.

Following the Address of Welcome, Dr. Stevenson gave a description, illustrated with beautiful lantern slides in color, of the symbolic Pfahler gavel, fashioned from a mastodon's tooth recovered from Alaskan glacial ice, and the working Erskine gavel made from memorabilia of Röntgen, Coolidge and Snook.

Concluding the ceremony, Dr. Stevenson installed Dr. C. Allen Good as the new President, and awarded him the traditional Gold Medal of "President of the American Roentgen Ray Society."

As his first function, incoming President Good remitted to outgoing President Stevenson the Past Presidents' Book, inscribed with his name, and containing the color photographs of the Pfahler and Erskine gavels, and a list of all Past Presidents.

President Good then gave his Inaugural Address on "Evolution in the Training of Radiologists," which is published in full as the leading article of this issue of the JOURNAL. In his Address Dr. Good skillfully described the background of the slowly evolving formal training in the science and art of radiology in the early days, leading up to the formation of the American Board of Radiology in 1934. He mentioned that Dr. B. R. Kirklin, first and longtime secretary of the Board, devoted his Caldwell Lecture before the American Roentgen Ray Society in 1948 to "Graduate Education in Radiology" and traced the development of radiologic training up to that time. In 1948 the essential requirement was 3 years of formal residency training in radiology and the Board examination consisted of oral interviews of 30 minutes each by 6 examiners. In the almost 20 years since 1948 many changes have taken place. The two most important of these are that, in 1960, a fourth year of either training or practice has been added, and now it was announced that beginning with June, 1968 a written examination will also be required. The mechanism of implementation of the latest changes was discussed and a cursory comparison with the essential requirements of other specialty Boards was given.

As an examiner for the American Board of Radiology over a long period, President Good then stated without reservation, "That today young radiologists who are ready for certification are much better trained and much more knowledgable in all phases of our specialty than they were 20, yes, even 10 years ago." He also considered some of the future possibilities in the further training of radiologists, including the role of scientific societies and various educational organizations, and concluded with the statement: "The various pieces of the

future structure of our specialty are currently available. They require little reorientation to evolve into a better method of education for more competent radiologists. Certainly that is our goal. . . ."

The First Scientific Session began at 9:10 A.M. Tuesday with President C. Allen Good in the Chair. Ten papers were presented, of which 6 dealt with pediatric and fetal problems, 2 with renal problems, 1 with a new apparatus for mass screening with mammography and 1 with thermography and vein diameter ratio in the detection of the nonpalpable breast carcinoma.

On Tuesday evening the Caldwell Lecture was given by Dr. John W. Kirklin, M.D., Professor of Surgery and Chairman of the Department of Surgery, University of Alabama Medical Center and University Hospitals and Clinics, Birmingham, Alabama. The title of his lecture was "The Tetralogy of Fallot." Doctor Kirklin, who is recognized as one of the most famous cardiovascular surgeons of the world, presented a brilliant lecture, delivered with extraordinary clarity and superbly illustrated.

Dr. Kirklin was introduced by his equally famous Harvard teacher and mentor, Robert E. Gross, M.D., Ladd Professor of Children's Surgery, Harvard Medical School and Surgeon-in-Chief of Children's Hospital, Boston, Massachusetts. The introduction of Dr. Gross and the Caldwell Lecture of Dr. Kirklin will be published in the February, 1968 issue of the JOURNAL.

At the conclusion of the lecture, President Good presented to Dr. Kirklin the Caldwell Gold Medal, the highest recognition of the Society for meritorious scientific achievement.

The Second Scientific Session was started at 8:30 A.M. Wednesday, with 1st Vice-President Arthur E. Childe, M.D., of Winnipeg, Manitoba, Canada, presiding. At this session 13 papers were presented on varied subjects, including: Basal cell nevus syndrome, shoulder arthrography, tomography of the petrous pyramid, vertebra plana in histocystosis, pseudoinfections of the intervertebral disk and adjacent vertebrae, beam quality measurement in diagnostic roentgenology, radiation beam analysis for diagnostic radiology, panography, fluorodensimetry, ultrasound, the effect of patient position on the radioisotope renogram, guided percutaneous arterial infusion chemotherapy and complications associated with arterial chemotherapy perfusion.

Wednesday at 1:30 P.M. the American College of Radiology, following the tradition of the past, presented "A Report from the American College of Radiology." The moderator was J. E. Miller M.D., President of the College, and the "report" included: (1) Presentation of a beautifully executed teaching film on "Roentgen Anatomy of the Normal Urinary System" by J. Scott Dunbar, M.D., and (2) Presentation from Commission on Public Relations, "Cooperative Programs with Organizations on X-ray Industry."

The Third Scientific Session began Thursday morning at 8:30 A.M., with J. Scott Dunbar, M.D., Montreal, Quebec, Canada, Chairman of the Executive Council, presiding. At this session 12 papers were read, again including varied topics such as: Esthesioneuroblastoma, radiation injuries of the jaws and teeth, curative radiotherapy of carcinoma of the esophagus, treatment of lung cancer with split-dose radiation therapy, prognosis in germinal testicular tumor, radium dosage patterns in treatment of uterine carcinoma, permanent interstitial therapy with low-energy long half-life sources, and 5 papers on gastrointestinal topics of timely interest.

On Thursday evening at 7:30 P.M. the Annual Banquet, preceded by a Cocktail Party at 6:30 P.M., and held in the Hilton International Ballroom, was the highlight of the social activities of the meeting. President Good, after introducing the newly elected officers and their wives, presented the highly coveted Awards to the Scientific Exhibit winners. This ceremony was followed by an entertaining floor show and a pleasant evening of dancing.

On Friday morning the Fourth and last Scientific Session, which again started at

8:30 A.M., was presided over by Edgar M. McPeak, M.D., Washington, D. C., 2nd Vice-President of the Society. At this session 12 papers were presented, 5 of which dealt with pulmonary conditions and 7 with cardiovascular diagnostic problems. The scheduling of so many related papers on one morning created unusual interest with the result that at the closing of the Sixty-eighth Annual Meeting at 12:30 P.M. still a large audience was present.

The Section on Instruction, with Dr. Harold O. Peterson as Director and Dr. Donn G. Mosser as Associate Director, comprised an outstanding Faculty of Instructors, who conducted courses in a relatively informal manner on the latest contributions to radiology. A four-period sequential course on radiotherapy was given, on special invitation, by Robert Gibb, M.D., Ch.B., F.F.R., Deputy Director of Radiotherapy, Christie Hospital and Holt Radium Institute, Manchester, England. The arrangements for the courses were excellent and they attracted large groups of interested individuals.

The Scientific Exhibits were numerically the largest in the Society's history and they were most instructive. Dr. Richard G. Lester, enthusiastic Chairman and his Committee continued the high standard for such exhibits and their success will be difficult to surpass in the future. A detailed description of the Scientific Exhibits is given in the following pages of this issue of the JOURNAL.

The Technical Exhibits likewise were numerically the largest and qualitatively the most outstanding in the Society's history. They were assembled under the expert direction of Mr. Clifford L. Sherratt, Honorary Member, and Dr. James C. Cook, Manager of the Annual Meeting. This issue of the JOURNAL also gives an account of these exhibits with a detailed list of the exhibiting firms.

The Committee on Local Arrangements, under the able Chairmanship of Dr. Charles E. Bickham, Jr., and the Ladies Committee, with charming Mrs. Charles E. Bickham as Chairman, had prepared excellent social activities. There were comprehensive lectured tours throughout Washington, An Afternoon on Embassy Row, a visit to the Museum of African Art, followed by lunch at Billy Martin's Carriage House in Old Georgetown, and a special church tour to the Episcopal Cathedral of Saints Peter and Paul, the National Shrine of the Immaculate Conception and the San Franciscan Monastery. To Dr. and Mrs. Charles E. Bickham, Jr., the members of their Committees, and all those who participated in the arrangement of this most enjoyable program, the Society is very grateful and extends its profound thanks.

On Monday, preceding the meeting, the Annual Golf Tournament was held at the Indian Spring Country Club, Glenmont, Maryland. Dr. Ralph M. Caulk, Past President of the Society and ardent golf enthusiast, with the aid of his local Committee made all the necessary arrangements. On a beautiful course, under sunny skies, competition in the tournament was for the Willis F. Manges Trophy awarded for the low net score, and the Exhibitors' Trophy awarded for the low gross score. This was followed by cocktails and the Annual Golf Dinner.

The Committee on Press Relationship under the Chairmanship of Dr. Karl C. Corley, with the splendid assistance from Mr. Otha W. Linton, Director of Public Relations of the American College of Radiology, conducted the press and public relation activities with skill and good judgment.

To the Management and Staff of the Washington Hilton Hotel, and to the Washington Convention Bureau, the Society expresses its sincerest thanks for the excellent service, efficient registration and the many other courtesies gracefully rendered throughout the meeting.

Judging from the comments which were heard from those who attended, it is the concerted opinion that the Sixty-eighth Annual Meeting was most outstanding in every respect. President Good may take justifiable pride, for this was a meeting that will be long remembered for its excellence.

TRAIAN LEUCUTIA, M.D.

# THE SCIENTIFIC EXHIBITS

THE SCIENTIFIC EXHIBITS of the Sixty-eighth Annual Meeting of the American Roentgen Ray Society held at the Washington Hilton Hotel, Washington, D. C., September 26–29, 1967 surpassed all former records. They were nearly double in number as compared to the Scientific Exhibits assembled in the large and conveniently furnished Conventional Hall two years ago; they were of the highest visual quality and documentative value; and a surprisingly large number of them authoritatively complemented the scientific essays on the same subject presented at the Scientific Sessions.

The Society is greatly indebted to Dr. Richard G. Lester of Durham, North Carolina, Chairman, and his able Scientific Exhibits Committee for their enthusiasm and untiring effort in assembling these splendid exhibits, thus contributing significantly to the success of the Annual Meeting.

In recognition of the over-all excellence of this year's Scientific Exhibits, the Committee on Awards, after considerable deliberation, has awarded one Gold Medal, one Silver Medal, one Bronze Medal and 17 Certificates of Merit.

The following is a detailed account of the Exhibits:

## Gold Medal

*Laryngography*. Charles A. Stern, M.D., and Dan F. McFarland, M.D., Cincinnati General Hospital, Cincinnati, Ohio.

This beautifully displayed exhibit demonstrated the gross anatomy, roentgen anatomy and pathology of the larynx. Laryngograms of specimens coated with contrast material and drawings of the larynx were used to illustrate the gross and roentgen anatomy. Pathologic lesions of the larynx, particularly of the epiglottis, aryepiglottic fold, false vocal cord, true vocal cord, and subglottic region were shown and correlated with drawings in black and color of the larynx as seen on indirect laryngoscopy.

The following is an outline of the material presented:

A. *Gross Anatomy:* (1) Cartilages; (2) internal structures.

B. *Roentgen Anatomy:* Following description of the techniques of examinations, frontal and lateral laryngograms were presented during: (1) quiet breathing (inspiration); (2) phonation; (3) Valsalva maneuver; (4) modified Valsalva maneuver; and (5) inspiratory (reverse) phonation.

C. *Pathologic* examples were illustrated of: (1) Paralysis of left vocal cord; (2) carcinoma of true vocal cord; (3) carcinoma of free portion of epiglottis; (4) carcinoma of aryepiglottic fold; (5) carcinoma of vocal cord with subglottic extension; (6) carcinoma of pyriform sinus; and (7) laryngeal carcinoma with massive extension.

A list of pertinent references was appended.

## Silver Medal

*Experience with the Concomitant Use of Hydroxyurea and X-Radiation in the Management of Cancer*. Harvey J. Lerner, M.D., and Gerald L. Beckloff, M.D., Pennsylvania Hospital, Philadelphia, Pennsylvania.

Approximately 75 patients, with the majority being inoperable lung cancer and epidermoid carcinoma of the head and neck cases, have been treated with hydroxyurea and x-radiation concomitantly. Hydroxyurea is administered as a single oral dose every third day. Seven days after treatment is initiated, x-radiation is given 3 times a week for 6–10 weeks in full therapeutic doses. Hydroxyurea is continued through the irradiation and indefinitely thereafter. In the majority of cases, definitive surgery was performed 6–8 weeks following radiotherapy.

In almost all instances, patients who were originally considered to be inoperable have become surgical candidates after combination treatment.

BRONZE MEDAL

*Panography: A New Extension of Roentgenography; A Philadelphia Project.* J. Gershon-Cohen, M.D., M. B. Hermel, M.D., A. G. B. Borden, M.D., George T. Wohl, M.D., Alma Dea Morani, M.D., Herbert Goldberg, M.D., and Captain Manny H. Moser, M.D., Temple University, Philadelphia General Hospital, Pennsylvania Woman's Medical College Hospital, and Valley Forge General Hospital, Philadelphia, Pennsylvania.

This exhibit presented the characteristics of a new apparatus for obtaining new projections of various anatomic regions.

The anatomic structures displayed showed evidence of disease and trauma not previously readily obtainable about the face, temporomandibular area, orbits, temporal bones, skull, neck, shoulder, fingers, pelvis, hip joints, knees, ankles, and feet.

There was a scientific essay on the same subject.

CERTIFICATE OF MERIT

1. *Early Recognition of Rheumatoid Arthritis (X-Ray Findings).* David L. Berens, M.D., L. Maxwell Lockie, M.D., and Bernard M. Norcross, M.D., State University of New York at Buffalo and Buffalo General Hospital, Buffalo, New York.

Routine studies of hands, wrists and feet were done on fine-grain film during a 6 year period. Characteristic roentgenographic findings were often noted in asymptomatic joints which contributed to the early recognition of the disease.

Detailed, illustrated descriptions of these changes were correlated with the clinical, laboratory, and pathologic findings. Management of the early case was discussed.

2. *Congenital Scoliosis with a Unilateral Bar.* James J. Conway, M.D.,* Wallace T. Miller, M.D.,* and C. Dean MacEwen, M.D.,† Hospital of the University of Pennsylvania,* Philadelphia, Pennsylvania, and Alfred I. duPont Institute,† Wilmington, Delaware.

A series of 86 cases of scoliosis, secondary to congenital anomalies of the spine, was reviewed.

In many instances of congenital scoliosis, the curvature is stable and progresses little during growth.

One group consistently resulted in severe progressive deformities. A characteristic roentgenographic appearance was noted even in infancy in this group. A unilateral bar secondary to unilateral failure of segmentation of the posterolateral masses of the vertebrae provides a point of fixation about which severe progressive curvature of the spine occurs during growth.

Cases were presented demonstrating early and late manifestations of this anomaly, with and without treatment. Emphasis was placed on early recognition and treatment.

3. *Selective Arteriography of Spinal Arteriovenous Aneurysms.* John Doppman, M.D., Giovanni Di Chiro, M.D., and Ayub Ommaya, M.B., F.R.C.S., Clinical Center, NIH and NINDB, Bethesda, Maryland.

Selective catheterization of the feeding arteries has been performed in 11 cases of spinal cord arteriovenous aneurysms. Surgical ligation was done in 9 cases.

The radiographic findings in pre- and postoperative arteriographic studies were presented and the results of surgical treatment were summarized.

4. *Laryngomalacia.* J. S. Dunbar, M.D., M. B. Nogrady, M.D., and D. W. MacEwan, M.D., Department of Radiology, The Montreal Children's Hospital, Montreal, Quebec, Canada.

Laryngomalacia is a common abnormality of infants in the first 6 to 12 months of life, and produces inspiratory stridor. While the clinical picture is usually sufficiently characteristic to permit the diagnosis to be made, it is not always possible to be certain from clinical information alone about the cause of the infant's stridor. In such cases, indirect or direct laryngoscopy was formerly considered necessary.

This exhibit showed that positive diagnosis of laryngomalacia can be made in almost every case by radiographic and

cinefluorographic examination of the infant's larynx in lateral projection. A cinefluorographic film was projected.

5. *Normal and Pathologic Roentgen Anatomy of the Paranasal Sinuses*. Lewis E. Etter, M.D., The Falk Clinic, Presbyterian-University Hospital, University of Pittsburgh, Pittsburgh, Pennsylvania.

Anatomic material and opacification techniques of the paranasal sinuses were shown to demonstrate the occurrence of lateral extensions of the sphenoid sinuses in approximately 45 to 50 per cent of skulls, where this anomaly is not often considered in radiologic and rhinologic interpretation.

This is important because one may have involvement of the lateral recesses of the sphenoid sinuses and, since these are projected over the maxillary sinuses in the Waters' position, a diagnosis of inflammatory change within the maxillary sinus may be made when, indeed, the complication is in the recesses of the sphenoid sinuses.

6. *Megasigmoid and the Megasigmoid Syndrome: Roentgen Aspects*. Nathaniel Finby, M.D., and Ernest Kraft, M.D., St. Luke's Hospital Center, NYC and Veterans Administration Hospital, Northport, New York.

Megasigmoid denotes an abnormally dilated sigmoid colon differing from megacolon by the extent of involvement. It can be related to a congenital abnormality (Hirschsprung's disease) or a wide variety of acquired conditions.

The megasigmoid syndrome is an acquired disease, predominantly but not exclusively observed in neuropsychiatric patients. It manifests itself as a chronic progressive localized rectosigmoid dilatation which becomes massive due to accumulation of feces and intestinal gas. It is uncommon since only 18 cases were found in the exhibitors' hospitals during the past 30 years.

The characteristic roentgen features were demonstrated.

7. *Gastro-Intestinal Lesions in Mastocytosis*. Gerald Fishbone, M.D., A. Everette James, M.D., Arthur R. Clemett, M.D., Murray L. Janower, M.D., and Robert J. Levine, M.D., Yale University School of Medicine, New Haven, Connecticut.

Gastrointestinal symptoms are very common in systemic mastocytosis but are only partially explained by the increased frequency of peptic ulcer in this disease. The Exhibitors found significant small bowel abnormalities in about one-third of these patients. The principal abnormalities—small nodular lesions and a diffuse mild infiltrative process—may occur alone or in combination. These were illustrated with correlative biopsy material.

The small bowel pathology is variable but can be explained by the unique biochemistry of the mast cell and by analogy to the better known lesions occurring in skin, bone, liver and spleen.

There was a scientific essay on the same subject.

8. *Historical Synopsis of American Radiology* (1896–1965). E. R. N. Grigg, M.D., Cook County Hospital, Chicago, Illinois.

The captions and the photographs (of men and organizations, of machines and their makers, and of ideas and resulting trends) illustrated the growth of the specialty in this country from (1) The Era of the Roentgen Pioneers through (2) The Golden Age of Radiology, and into (3) The Atomic Phase.

This was an elaborate exhibit, magnificently displayed.

9. *Selective Coronary Arteriography*. Melvin P. Judkins, M.D., University of Oregon Medical School, Portland, Oregon.

"Coronary seeking" tip configurations set in a moldable positive control catheter implement rapid, consistent selective coronary catheterization. When properly positioned, the catheters will remain in place for anatomic and dynamic filming in multiple projections. The technique can be quickly learned by the resident, fellow or practicing angiographer.

10. *Shoulder Arthrography*. Paul J. Killoran, M.D., Ralph C. Marcove, M.D., and Robert H. Freiberger, M.D., Hospital for Special Surgery affiliated with New

York Hospital-Cornell University Medical Center, New York, New York.

Shoulder arthrograms were used primarily to demonstrate traumatic or degenerative rupture of the rotator cuff. The degree of capsular laxity following shoulder dislocation, as well as the abnormally tight capsule of periarthritis ("frozen shoulder"), was also shown.

The technique of shoulder arthrography using an image intensifying fluoroscope was demonstrated. Normal and abnormal arthrograms were shown correlated with anatomic illustrations.

11. *Study of Dissecting Aneurysm with Retrograde Catheterization.* William Molnar, M.D., and Vivian J. Harris, M.D., Ohio State University Hospital, Columbus, Ohio.

The diagnosis and accurate evaluation of dissecting aneurysm require direct injection of the aorta which can be safely accomplished through retrograde catheterization utilizing a soft tip catheter. This approach was used in 21 consecutive patients with proven dissecting aneurysms at the Ohio State University during the last 4 years.

The display demonstrated the characteristic angiographic findings and their correlation with plain chest film diagnosis.

There was a scientific essay on the same subject.

12. *The Roentgenographic Features of Homocystinuria.* Charles L. Morreels, Jr., M.D., Barry D. Fletcher, M.D., and John D. Dorst, M.D., Johns Hopkins Medical Institutions, Baltimore, Maryland.

The roentgenographic and clinical features of 23 individuals with homocystinuria were presented.

The roentgenographic findings include: microencephaly, sternal deformities, dolichostenomelia, and cupping of the distal radial and ulnar metaphyses with stippling of the adjacent physes. Generalized osteoporosis with vertebral biconcavity and scoliosis is present. Roentgenographic evidence of pulmonary infarction is also observed.

These patients may be mentally retarded, and ectopia lentis is a constant clinical feature. There is a tendency to develop central and peripheral vascular thrombosis.

13. *Roentgen Significance of the Transverse Thoracis Muscle.* Charles E. Shopfner, M.D., Richard T. O'Kell, M.D., and Carl Jansen, M.D., Children's Mercy Hospital, Kansas City, Missouri.

An elliptic density measuring approximately $2 \times 5$ cm. is present behind the sternum in over 50 per cent of all pediatric chest roentgenograms. Clinical experience has shown that the structure causing this density has been misinterpreted as pneumonia, loculated pleural effusion, pleural tumor and mediastinal mass.

Correlation of roentgen and gross anatomy revealed this pathologically simulating roentgen finding to be caused by the normal density of the transverse thoracis muscle.

Awareness of the anatomico-roentgen features of this muscle permits its accurate identification without resort to complicated roentgenologic and surgical procedures, and prevents needless concern, confusion and treatment.

There was a scientific essay on the same subject.

14. *The Seldinger Catheter in Radiation Therapy.* Richard J. Steckel, M.D., University of California at Los Angeles, Los Angeles, California.

Work performed at the National Cancer Institute (Radiation Branch), Bethesda, Maryland; James D. MacLowry, M.D. (Pathology Branch), James M. Holland, M.D. (Surgery Branch), Ralph E. Johnson, M.D. (Radiation Branch), and David F. Paulson, M.D. (Surgery Branch).

A method has been developed to protect the canine kidney from irradiation by the infusion of microgram quantities of epinephrine into the renal artery during irradiation. A percutaneous catheter (Seldinger) is used.

This technique might be employed to prevent radiation nephritis following high-dose radiation therapy to abdominal tumors.

The results of animal studies, as well

as of preliminary clinical studies, were presented.

15. *Roentgenographic and Clinical Manifestations of Primary TBC.* Alfred L. Weber, M.D., Kenneth T. Bird, M.D., and Murray L. Janower, M.D., Massachusetts General Hospital, Boston, Massachusetts.

This exhibit demonstrated the various roentgenographic manifestations on admission and during treatment of primary tuberculosis as they were encountered in the analysis of 85 patients from the Middlesex County Sanatorium in Waltham, Massachusetts.

Diagnosis, clinical symptoms, complications and bronchoscopic findings supplemented the detailed roentgenographic analysis.

16. *Selective Biplane Coronary Arteriography with Cesium 131 Myocardial Scanning.* William J. Wilson, M.D., Charles A. Dobry, M.D., Kurt Amplatz, M.D.,* and Richard E. Wetzel, M.D., University of Virginia Hospital, Charlottesville, Virginia, and University of Minneapolis Hospital,* Minneapolis, Minnesota.

The exact location of coronary arterial disease by selective biplane arteriography prompted a study to determine the accuracy of the cesium 131 myocardial scanning in the diagnosis of arteriosclerotic myocardial disease. All patients studied had a clinical history thought to indicate probable angina pectoris.

The technique of examination, arteriograms, myocardial scans, case materials and results were presented.

17. *Roentgenologic Contribution to the Evaluation and Management of Scoliosis.* Lionel W. Young, M.D., Louis A. Goldstein, M.D., and Alan E. Oestreich, M.D., University of Rochester Medical Center, Rochester, New York.

At the University of Rochester Center there has been an organized clinical program for the detection, evaluation and treatment of scoliosis in children since 1939.

This exhibit emphasized the roentgenologic contribution to scoliosis as a diagnostic parameter and treatment surveillance measure. Accurate curve measurement for all types of scoliosis is dependent upon this method.

The significant diagnostic and prognostic indicators of congenital, idiopathic and other types of scoliosis were demonstrated.

An etiologic classification of scoliosis was presented, together with historical background, pathologic anatomy, clinical features, treatment principles, results and complications.

OTHER EXCELLENT EXHIBITS WERE:

*Pediatric Case of the Day.* D. H. Altman, M.D., R. E. Litt, M.D., and L. A. Greenberg, M.D., Variety Children's Hospital, Miami, Florida.

A different pediatric case was presented each day. A single diagnosis was permitted on the basis of history and pertinent roentgenograms.

The answer was given the following day with detailed discussion and demonstration of the pathology in color.

Rewards were given to all radiologists making a correct diagnosis.

*Obstructive Valves of the Male Urethra.* Donald P. Babbitt, M.D., Norman B. Hodgson, M.D., and Stuart W. Fine, M.D., Milwaukee Children's Hospital, Milwaukee, Wisconsin.

This exhibit presented the spectrum of urethral valves with examples of normal anatomy, various degrees of obstructive urethral valvular lesions, and demonstration of the secondary changes in the urinary tract. It emphasized the variation in the clinical history of these children.

The diagnostic techniques were graphically illustrated, stressing the use of the voiding cystourethrogram and intravenous pyelogram.

Treatment was also discussed, using before and after treatment examples.

There was a 5 minute cinecystourethrographic movie on a continuous running Mark IV projector.

*Diagnosis of Intra-Abdominal Bleeding by Means of Selective Arteriography.* Stanley Baum, M.D., Moreye Nusbaum,

M.D., Koson Kuroda, M.D., and George Stein, M.D., Graduate Hospital, University of Pennsylvania, Philadelphia, Pennsylvania.

The clinical application of selective arteriography in the diagnosis of unknown sites of gastrointestinal hemorrhage was illustrated. In addition, experimental work utilizing controlled bleeding rates in dogs was shown.

The technique which affords a method for the control of hemorrhage by the infusion of vasoconstricting agents directly into the mesenteric bed was demonstrated.

*The Value of Prone Roentgenography of the Abdomen in Pediatrics.* Walter E. Berdon, M.D., David H. Baker, M.D., and John Leonidas, M.D., Babies Hospital, Columbia Presbyterian Medical Center, New York City, New York.

Prone (as opposed to conventional supine) roentgenography of the abdomen has proven superior in identifying free air, sites of obstruction, and in clearly showing genitourinary pathology.

In addition to examples, the physiologic basis behind this simple maneuver was presented.

There was a scientific essay on the same subject.

*$CO_2$-$O_2$ Pneumomediastinography with Polytomography in the Evaluation of Mediastinal Disease.* Alfred S. Berne, M.D., Philip Ikins, M.D., and Walter Bugden, M.D., Syracuse Memorial Hospital and Upstate State Medical Center of New York, Syracuse, New York.

The Exhibitors' technique of pneumomediastinography was described and illustrated. The indications for pneumomediastinography were given and the roentgenographic criteria for diagnosis were discussed.

The advantages and limitations of the procedure were demonstrated.

*Neoplasms and Related Conditions of the Small Intestine.* Irwin Bluth, M.D., Bruce Steger, M.D., and Leroy Garner, M.D., The Brookdale Hospital Center, Brooklyn, New York.

Roentgenograms of benign as well as of primary and metastatic malignant neoplasms were demonstrated.

The characteristic features of specific tumors were stressed when pertinent.

Non-neoplastic conditions which enter into the differential diagnosis were cited.

*Plain Film Diagnosis of Dissecting Aneurysms of the Thoracic Aorta.* John D. Brooks, M.D., Thomas S. Harle, M.D., Lois C. Collins, M.D., and John O. F. Roehm, Jr., M.D., Baylor University College of Medicine, Houston, Texas.

The early recognition of dissecting aneurysms of the thoracic aorta has become of increasing importance since the advent of readily available surgical reparative techniques.

There are several reliable plain film signs which permit early recognition of dissection.

This exhibit illustrated these signs, which may indicate the need for further studies, such as angiography, or may provide the clinician with adequate diagnostic information in those instances in which the condition of the patient makes further study impractical.

*Variation of the Scan Pattern in Middle Cerebral Artery Occlusion.* Hyo H. Byun, M.D., Marion F. Magalotti, M.D., Stasia J. Frank, M.D., and Anthony J. Raimondi, M.D., Cook County Hospital, Chicago, Illinois.

Among the interesting abnormal brain scans seen in conditions other than neoplasm are those produced by acute cerebrovascular occlusion.

In their study of abnormal brain scans produced by acute cerebrovascular accident, the Exhibitors found a wide variety of scan configurations which they attempted to classify as follows: (1) Tubular, (2) triangular, (3) wedge shaped, (4) bi-lobular, (5) tumor-like, and (6) diffuse.

Examples of each were presented.

There was also a scientific essay on the same subject.

*Remote Control Filming and Fluoroscopy Combined with High-Speed Processing; A*

*Practical Method for Gastrointestinal Tract Examination.* Robert E. Campbell, M.D., and William J. Tuddenham, M.D., Pennsylvania Hospital, Philadelphia, Pennsylvania.

Examination of the gastrointestinal tract by remote control filming and fluoroscopy is still rejected by many as unsatisfactory and impractical.

Examples of 2 years' experience with this method combined with high-speed film processing were exhibited to prove that this concept is invalid.

*Epicardial Fat: Lateral Plain Film Analysis in Normals and in Pericardial Effusions.* Edward W. Carsky, M.D., and Edward J. Lane, Jr., M.D., Upstate Medical Center and Crouse-Irving Hospital, Syracuse, New York.

Analysis of routine lateral chest roentgenograms frequently demonstrates the normal mediastinal fat and the subepicardial fat separated by a soft tissue line representing the pericardium. These two fat stripes are separated by soft tissue density in pericardial effusions and can be identified on plain lateral chest roentgenograms. In the Exhibitors' series it occurred in 16 of 27 cases. In no case of septic pericarditis was the subpericardial fat stripe visible.

*Bony Abnormalities in Gardner's Syndrome.* C. H. Joseph Chang, M.D., Edward D. Piatt, M.D., Kenneth E. Thomas, M.D., and Alvin L. Watne, M.D., West Virginia University School of Medicine, Morgantown, West Virginia.

Gardner's syndrome is a familial disease with a triad consisting of colonic polyposis, osteomas, and soft-tissue tumors. It was first described in 1950 by Gardner, who also postulated mendelian autosomal dominant inheritance.

Recently, at West Virginia University Medical Center, the Exhibitors examined 33 members of 2 families with Gardner's syndrome. A variety of bone lesions, ranging from localized cortical thickening to large osteomas, have been noted.

The purpose of this exhibit was to present the various osseous lesions in this interesting disorder.

*Amniography As an Aid in Intrauterine Fetal Transfusion.* Charles N. Chasler, M.D., and Anne N. Tessaro, Magee-Women's Hospital, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania.

The purpose of this exhibit was to review the basic problem of erythroblastosis fetalis and demonstrate the role of radiology in the treatment via intrauterine fetal transfusion.

The initial portion defined the disease and was followed by a demonstration of the method of transfusion. The final portion displayed some of the complications encountered as well as a few interesting examples of additional information obtained as a result of the procedure.

There was a scientific essay on the same subject.

*Educational Program (ACR Foundation).* Charles N. Chasler, M.D., University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania.

In this exhibit some reproductions of "Classical Monographs on Radiology" were shown in loosely bound volumes.

*Voiding Cystourethrography.* Arthur L. Coleman, M.D., and Keith Waterhouse, M.D., The Brooklyn Hospital, Brooklyn, New York.

Investigations of urologic disease with particular emphasis on the functional and anatomic considerations relative to bladder outlet obstruction have been intensively pursued. Voiding cystourethrography has been designed to evaluate bladder emptying and detect reflux, outlet obstruction and bladder deformities.

The indications, preparation, materials needed, contrast media, and cystographic technique were presented.

This simple, radiographic examination of the urinary tract in children has been performed with standard radiographic equipment in a general hospital.

The problems in diagnosis from the normal to the abnormal were discussed.

*Guided Percutaneous Arterial Infusion Chemotherapy.* Joseph L. Curry, M.D., and Willard J. Howland, Jr., M.D., Ohio Valley General Hospital, Wheeling, West Virginia.

This exhibit detailed a percutaneous catheter approach to arterial chemotherapy of tumors of diverse anatomic location. Catheter exploration angiographically locates the optimum position for tumor perfusion, and a carotid or axillary insertion site permits ambulation.

The problem of surgically locating arterial tumor "feeders" in extensive neoplasms is obviated. Catheters of moderate size permit adequate angiography, are well tolerated for several weeks and rarely obstruct. Tiny catheters, surgically inserted, do not allow adequate angiography and often obstruct.

There was a scientific essay on the same subject.

*Salivary Gland Scanning with Technetium ($Tc^{99m}$ Pertechnetate.* Giovanni Di Chiro, M.D., and Arthur S. Grove, Jr., M.D., National Institutes of Health, Bethesda, Maryland.

Technetium pertechnetate is concentrated by a variety of normal and abnormal tissues. This isotope rapidly accumulates in the salivary glands and permits their functional evaluation, delineation and localization by multiple view scanning.

This exhibit demonstrated the usefulness of technetium scanning in (a) evaluating salivary gland function, (b) determining the limits, location or infiltration of these glands and (c) differentiating among some of the tumors and masses which occur within or nearby the salivary glands.

*Diagnosability of Colon Stenoses.* M. Wendell Dietz, M.D., University of Missouri Medical Center and Burge-Protestant Hospital, Springfield, Missouri.

This exhibit comprised 23 film studies of 18 diverse etiologic categories of colonic stenosis.

Roentgenologically, the nonspecificity of benign constrictions was notable. Nevertheless, accurate evaluation was ordinarily achieved upon correlation with concomitant and prior roentgen findings, history, physical and laboratory data.

These criteria were ineffectual in the diagnosis of two stenoses secondary to pericolic abscess (one due to perforative ulcerative colitis and one of indeterminate cause) and of a case of "functional" stenosis. Both primary and secondary malignant stenoses were typically neoplastic.

Three unusual complications were displayed: megacolon, intestinal perforation and fecalithiasis.

*Infusion Drip Cholangiography.* John L. Doyle, M.D., F.A.C.S., and Peter Zanca, M.D., F.A.C.R., Department of Surgery and Radiology, Robert B. Green Memorial Hospital, University of Texas, South Texas Medical School, San Antonio, Texas.

This exhibit presented a series of cases in which infusion "drip" cholangiography proved advantageous, where routine cholangiographic examinations yielded indeterminate results.

*Arteriography of the Breast.* Frieda Feldman, M.D., David V. Habif, M.D., Richard Fleming, M.D., Ira Kanter, M.D., and William B. Seaman, M.D., Columbia Presbyterian Medical Center, New York, New York.

Angiography of the breast was done in 12 patients in whom a diagnosis of malignant tumor was made from clinical evidence, together with 1 normal control. The apparent correlation between the arteriographic findings and the surgical results has been encouraging. There were abnormal findings in all 6 cases of malignancy which were absent in the benign lesions or in the normal.

*Pediatric Liver Scans: Defects Simulating Metastatic Disease.* Kenneth E. Fellows, Jr., M.D., and Melvin Tefft, M.D., Children's Hospital Medical Center, Boston, Massachusetts.

Liver scans are often used in the pre-

operative evaluation of children with abdominal tumors. Extrahepatic masses have been found to simulate intrahepatic invasion, or metastases by causing pressure deformities in the liver. Three examples were presented.

Children who have had abdominal irradiation for malignant disease were also followed for metastases with liver scans. In some cases the original (pretherapy) scans were normal; however, 2 to 10 months after therapy, localized defects appeared and persisted. Biopsy in each case demonstrated radiation change without evidence of tumor. Three such cases were also presented.

It, therefore, seems appropriate in the Exhibitors' opinion, to consider extrinsic pressure deformity and radiation change as sources of abnormality other than metastasis, when evaluating liver scans in children with abdominal neoplasia.

*Ultrasonography: Echo Trace, Echo Scan.* Walter Francke, M.D., Colvin H. Agnew, M.D., and John Anderson, M.D., St. Vincent's Hospital, Billings, Montana.

High frequency short duration electromechanical pulses emitted by special transducers in direct or indirect contact with a part of the body under examination produce visual information of the utmost value. These rays penetrate the body and are reflected by (a) tissue interfaces, (b) changes in density, (c) differences in acoustic impedance, and (d) variations of the elasticity encountered in even slight molecular structure dissimilarity.

The exhibit included A and B scope scanning and also contact scanning (body contour).

The photographs demonstrated brain, liver and breast scans for tumor detection, cirrhosis, third ventricle displacement, etc.

*Roentgenographic Aspects of the Normal and Abnormal Vagina.* Richard M. Friedenberg, M.D., Max C. King, M.D., and Charles Ney, M.D., The Bronx-Lebanon Hospital Center, The Bronx, New York.

The Exhibitors presented a comprehensive survey of various conditions affecting the vagina which can be visualized by contrast roentgenographic examination. Included in the presentation were the normal vagina in children and adults, congenital lesions of the vagina such as intersex, fistulas, inflammatory lesions and benign tumors.

The technique of examination was given together with brief discussions of the diagnostic criteria of each entity.

*Dual-Tracer Technique—An Expansion of the Clinical Value of Scanning.* Jack K. Goodrich, M.D., Robert Wilkinson, Jr., M.D., C. Craig Harris, M.S., Juan Carranza, M.D., and A. J. Wyrick, M.S., Division of Nuclear Medicine, Department of Radiology, Duke University Medical Center, Durham, North Carolina.

Radioactivity scanning depends on selective concentration or retention of tracer substances in body organs or pools through a variety of physiologic processes. A certain radioactive pharmaceutical, biologic, or inorganic tracer may be chosen to visualize a specific organ.

Dual-tracer techniques employ different tracers, either simultaneously or in serial studies to show one organ's relationship to another and both expand and confirm marginal findings by virtue of differing tracer properties.

Organ groups that may be visualized by these dual-tracer techniques are liver-spleen-lung, heart-lung, liver-pancreas, and others. Additional choices of labelling radionuclides add value to this useful and still developing procedure.

*Roentgenographic Classification of Renal Cysts.* Herman Grossman, M.D., Patricia Winchester, M.D., Frank Chisari, B.A., and Jack Hagstrom, M.D., The New York Hospital-Cornell Medical Center, New York, New York.

Four distinct types of cysts may be distinguished according to their distribution throughout the kidney and the nature of the principal pathologic disturbance.

These are: (1) Generalized dilatation of collecting tubules; (2) cysts involving

nephrons and collecting tubules; (3) cysts involving nephrons and collecting tubules but with irregular distribution, normally formed and functioning tissue being intermixed with the abnormally dilated structures; and (4) microscopic and/or gross cysts, similar to types (2) and (3), which result from pressure secondary to distal obstruction such as urethral valves or narrowed bladder neck.

Representative examples of each type were shown.

There was a scientific essay on the same subject.

*The Systematic Double Contrast Study in Examination of the Colon.* Luc Hamelin, M.D., and Maurice Dufresne, M.D., Service de Radiologie, Hôpital Notre-Dame, Montréal, Québec, Canada.

Results of this technique based on a series of 5,000 cases studied systematically over a period of 18 months revealed remarkable improvement in statistical data.

The technique was described in detail and the documenting results obtained were presented in beautifully executed color tables and drawings.

*Pulmonary Edema Without Cardiomegaly.* Thomas S. Harle, M.D., James T. Kountoupis, M.D., Herbert L. Fred, M.D., and Max L. M. Boone, M.D., Baylor University College of Medicine, Houston, Texas.

This exhibit emphasized that pulmonary edema of both cardiac and noncardiac origin can occur in the presence of a normal sized heart. Cognizance of this fact can prevent significant diagnositic and therapeutic errors. The edema may be unilateral or bilateral in distribution and may appear interstitially, as nodules or as diffuse alveolar infiltrates.

Among representative cases shown were examples of pulmonary edema resulting from acute myocardial infarction, uremia, allergic reactions to drugs and blood, convulsions, hanging, near drowning, connective tissue disease, and exercise at high altitude.

There was a scientific essay on the same subject.

*The Effect of Grid Variations on High Kilovoltage 70 MM. Radiographic Spot Filming.* David B. Hayt, M.D., and Carl J. Collica, B.S., Misericordia and Fordham Hospitals, New York, New York.

Comparative 125 kv. 70 mm. spot roentgenograms were obtained, utilizing 6 different grids or combinations of grids to show the changes in film quality at varying patient thicknesses in the abdomen. Accompanying radiation dosage measurements were made for each combination.

It was concluded that very little difference in film quality and radiation dosage is seen regardless of the grid employed in thinner patients, but major differences are noted with heavier patients. The use of crossed grids produced the best quality 70 mm. spot roentgenograms.

*Anonymous Mycobacteria in Human Disease; Potential for Specific Diagnosis.* E. Robert Heitzman, M.D., Robert A. Bornhurst, M.D., and John P. Russell, M.D., Upstate Medical Center, Syracuse, New York.

In the relatively few reports which have appeared in the literature concerning human disease produced by anonymous mycobacteria, the possibility of a specific roentgen diagnosis has been debated.

Based on an analysis of 20 cases, all with positive cultures for anonymous mycobacteria and without *M. tuberculosis*, the Exhibitors gave criteria by which disease due to these organisms can be suggested. The importance of distinguishing between diseases due to anonymous mycobacteria and disease due to *M. tuberculosis* was emphasized.

*Complete Angiographic Evaluation of Renal Cancers.* Paul C. Kahn, M.D., Henry M. Wise, Jr., M.D., and Alan H. Robbins, M.D., Department of Radiology and Urology, Tufts–New England Medical Center, Boston, Massachusetts.

The diagnosis of renal carcinoma can usually be made by simple aortography or selective renal arteriography. The complete evaluation of such tumors requires more sophisticated techniques. We have attempted to obtain a complete evaluation of tumor size, extracapsular extension, venous

invasion and remote metastasis in 30 patients with kidney cancer. This has been done by combining aortography and selective renal arteriography with epinephrine renal arteriography, selective renal venography, and selective arteriography of suspected metastatic sites—all in one carefully planned examination.

The results of this study were presented.

*The Roentgenographic Evaluation of Ascites.* Eugene J. Keeffe, M.D., Raymond A. Gagliardi, M.D., and Richard C. Pfister, M.D., St. Joseph Mercy Hospital, Pontiac, Michigan.

In this well organized exhibit an evaluation has been made of the reliability of various roentgen findings in ascites with demonstration of each and their application to different situations.

Special emphasis has been placed on anatomic and pathologic considerations of the peritoneal flank stripes.

*Bronchogenic Carcinoma Masquerading As Inflammatory Disease Roentgenographically.* Israel E. Kirsh, M.D., Blaz Korosec, M.D., and Arnold Seitam, M.D., Veterans Administration Hospital, Hines, Illinois.

Many patients with primary carcinoma of the lung have sought hospital treatment when they had pneumonia. One has to be watchful for evidence of tumor on roentgenograms made to follow the progress of pneumonia.

Known cases of tuberculosis may likewise cause the radiologist to drop his suspicion and there can be evidence of carcinoma on the roentgenograms for some time before it is noticed. Some pathologists have found carcinoma frequently in tuberculous lungs. Longer survival of tuberculous patients with modern treatment may account for this.

Examples of such instances were presented, as were cases of skin cancer and cancer with fungus disease.

*Ultra Sound: Clinical Applications of B-Scan Ultrasonography.* J. S. Lehman, M.D., G. C. Evans, M.D., J. D. Bissell, D.D.S., M.S. in Bio. Eng., D. F. Torzewski, MS EE, M.S. in Bio. Eng., and J. G. Skucas, MS EE, Hahnemann Medical College and Hospital, Philadelphia, Pennsylvania.

The principles and techniques of B-scan ultrasonography were presented and illustrated.

Representative examples of the application of B-scanning to study of the brain and intracranial mass lesions, the liver, spleen, kidneys and pelvic organs, and other anatomic areas were displayed.

Pertinent clinical applications of B-scan ultrasonography were shown, along with a consideration of the present state of usefulness of this modality.

There was a scientific essay on the same subject.

*Arteriography of Trauma.* Leon Love, M.D., Thomas W. Braun, M.D., George B. Greenfield, M.D., Rogelio Moncada, M.D., and Carlos J. Reynes, M.D., Cook County Hospital, Chicago, Illinois.

A specialized trauma unit affords an opportunity to investigate cases of arterial injury in acute and chronic phases. Special attention was paid to abdominal and peripheral trauma.

*Chemotherapy of Metastatic Testis Cancer.* A. Ranald MacKenzie, M.D., Robert B. Golbey, M.D., and Willet F. Whitmore, Jr., M.D., Memorial and James Ewing Hospitals, New York, New York.

This exhibit gave results of chemotherapy in 180 patients with metastatic cancer of the testis. A variety of agents have been used. The best results, however, have mainly been associated with programs which have included actinomycin D.

A combination of actinomycin D, methotrexate and chlorambucil was employed as the first form of chemotherapy in 75 patients. An objective response occurred in 39 patients and in 11 of them there was complete disappearance of all evidence of metastatic disease. Five of these patients are still alive and well. This combination of drugs was used as a secondary program, *i.e.*, after some other program of chemotherapy, in 18 patients. Partial regression occurred in 2 but no patient became free of disease.

A combination of actinomycin D and chlorambucil was employed as primary

treatment in 27 patients. Partial objective regression of disease was seen in 14 patients and in 5 of them all evidence of disease disappeared. Two of these patients are still alive and well. This combination of drugs was used secondarily in 10 patients, partial objective regression being seen in 2.

Actinomycin D alone was employed primarily in 20 patients. Objective regression occurred in 15 patients with complete disappearance of all evidence of disease in 7. Six patients are still alive and free of disease. In addition, 2 patients recently treated are still undergoing regression of disease and may possibly reach a disease-free state. Actinomycin D alone has been used as a secondary treatment in a further 12 patients. Objective regression occurred in 5 and in 1 patient there was complete disappearance of disease. He is still alive and well.

The only other drugs which have caused disappearance of all evidence of metastatic disease are chlorambucil, which used alone caused disappearance of metastases in 2 patients with metastatic seminoma (1 of these patients is still alive and free of disease), and 6-mercaptopurine and 6-diazo-5-oxo-levonorleucine used in combination, which caused disappearance of disease in 1 patient.

*The Larynx: Heavy Filtration Roentgenography.* G. H. Maguire, M.D., J. D. Moir, M.D., and R. A. Beique, Ph.D., Montreal General Hospital, Montreal, Quebec, Canada.

By using the technique of heavy filtration (1 mm. brass or copper) and high kilovoltage, the roentgenographic examination of the larynx is so simplified that a wide variety of laryngeal abnormalities can be investigated fruitfully with a minimum of manipulation of the patient. More referrals from clinicians result, and the radiologist can contribute much more to the management of patients with upper airway abnormalities. The technique can even be used as a reliable means of assessing the patient who is difficult or impossible to examine adequately by mirror and often obviates the need for laryngoscopy, or actually indicates which patients should undergo laryngoscopy.

More sophisticated procedures such as contrast laryngography can be employed when indicated in specific situations.

*Spot Film—Urethrography in Infants and Children.* James A. Meadows, Jr., M.D., The Children's Hospital, Birmingham, Alabama.

Sixty roentgenographic films showing normal male and female urethras, inflammatory changes, constricting lesions, polyps, congenital anomalies, neurogenic changes, and other less frequent conditions were presented.

*Tumor Masses in Infants and Children Utilizing Angiography and Isotope Scanning.* V. G. Mikity, M.D., J. Paciulli, M.D., A. F. Turner, M.D., and M. Halpern, M.D., Department of Radiology, University of Southern California School of Medicine and Children's Division, Los Angeles County General Hospital, Los Angeles, California.

The radiographic diagnosis of abdominal masses in children is not too difficult utilizing current roentgenographic techniques.

However, with improved angiography, the preoperative diagnosis can be much more specific in a significant number of cases. Many times a tumor can be stated to be either benign or malignant. On occasion, its operability can be determined. The exact size of the neoplastic mass can be accurately outlined, which is of immeasurable value to both surgeon and radiotherapist.

This, combined with selective isotope scanning of the involved organ, can be utilized in the pre- and postoperative management.

Examples of a number of tumor masses within the abdomen and soft tissues were shown.

*Progress Report* 1967. Robert T. Morrison, American Institute of Radiology, Chicago, Illinois.

This exhibit was the formal announcement of the Institute being established in its own facilities.

The space, an area conjunctive with the American College of Radiology, was described by a floor plan and legends. The report summarized some of the acquired memorabilia and also some presently in process of negotiation.

Attention was invited to the advantages of premortal documentary donation to the Institute's collections and resources.

*Pneumatosis Intestinalis in Infants and Children.* J. Paciulli, M.D., V. G. Mikity, M.D., J. Gwinn, M.D., and J. E. Hodgman, M.D., U.S.C. School of Medicine and Children's Division, Los Angeles County General Hospital & Children's Hospital Society of Southern California, Los Angeles, California.

This rare condition of gas within the bowel wall may be seen in any part of the small bowel or colon and in children of any age.

In 16 infants and children it was found in association with intrinsic disease of the gastrointestinal tract, primarily as necrotizing enterocolitis, and rarely as vascular occlusion, or Hirschsprung's disease. The roentgenographic findings vary from a subtle localized area of involved small intestine to extensive collections with gas in the portal venous system.

When seen in the young infants, pneumatosis is usually indicative of sepsis.

Gas, when seen within the portal veins, is a grave prognostic sign.

*The Draftman's Lead Holder As a Radium Needle Inserter.* Enrique Pantoja, M.D., and Edward I. Holodny, B.S., The Jewish Hospital of Brooklyn, Brooklyn, New York.

Improved distribution of radium needles in planar and volume implants has been achieved employing the draftman's lead holder as an inserter.

A movie film in color demonstrated the actual use of the inserter during the implantation of a lesion in the operating room.

*Automatic Processor Control.* Andrew K. Poznanski, M.D., Lloyd A. Smith, B.S., and Richard A. Rideout, M.D., Henry Ford Hospital, Detroit, Michigan.

The advent of automatic processing has brought new problems of quality control, particularly in the large Radiology Department where several units are in operation.

Pre-exposed sensitometric strips do not truly reflect changes in development.

The use of daily, fresh densitometric strips produced in the X-Ray Department was found to be the most reliable method of controlling development and has been employed for more than 3 years.

Standard tests for fixation and washing can be performed at less frequent intervals.

*Radioisotope Scanning with a System for Total Information, Storage and Controlled Retrieval.* Abbas M. Rejali, M.D., Hymer L. Friedell, M.D., and Earl C. Gregg, Ph.D., Department of Radiology, Western Reserve University, Cleveland, Ohio.

This exhibit demonstrated improved clinical results obtained from a new radioisotope scanner and data presentation system.

In this system, all available information received by the detectors is stored without modification in a permanent memory. A special videcon television system permits immediate replay of the stored data with innumerable degrees of erase and contrast adjustments under carefully controlled circumstances. A profile representing the count rate through any slice in the scanned area may also be obtained to determine relative distribution of radioisotopes.

Various scans such as of the lung, bone, pancreas, cardiac blood pool, myocardium, placenta, etc., with this system were demonstrated.

*Hereditary Oculo-Dento-Osseous Dysplasia.* D. S. Rajic, M.D., and L. L. de Veber, M.D., University of Western Ontario and War Memorial Children's Hospital, London, Ontario, Canada.

A roentgenographic examination of the hands of a 3 year old girl, who was admitted to the hospital for repair of syndactyly revealed moderate widening of all phalanges and metacarpals with mottling of the bone texture, suggesting either storage of a substance in the bone marrow (reminiscent of gargoylism) or proliferation of the elements of the bone marrow (reminiscent of sickle

cell anemia). There was syndactyly of the ring and little fingers without bony fusion, and the middle phalanges of the little fingers were short (cube shaped) on both sides. The clinical examination of the child showed microphthalmia, a hypoplastic nose and hypoplasia of the enamel of the teeth, in addition to bilateral syndactyly of the ring and little fingers. The child showed normal intelligence. The skeletal survey revealed undertubulation of the long bones with slight mottling of the bone texture. The roentgenograms demonstrated hypoplasia of the teeth and particularly of the enamel of the teeth.

Similar changes were noted in the mother of the child, maternal grandmother, 1 maternal aunt and 2 younger siblings. Bone biopsies of 3 members of the family revealed "pseudopagetoid mosaic" structure of the long bones, but the bone marrow was normal.

Several similar cases are described in the literature, but without reference to the generalized bone changes.

In conclusion, the exhibitors stated that this is the first report of multiple cases in one family.

*Microradiography: A Description of Techniques and Demonstrations of Results.* R. J. Schulz, Ph.D., R. Anigstein, B.A., P. Roland, B.S., and M. Elkin, M.D., Albert Einstein College of Medicine of Yeshiva University, New York, New York.

Microradiography is the extension of x-ray analysis to the ultrashort wave length region of the x-ray spectrum. Microradiographs of tissue sections between 1 and 5 microns thick are made with x-rays produced by potentials that range from 500 to 5,000 volts. The tissues are placed in direct contact with high resolution photographic plates. No tissue stains are required because imaging results from x-ray absorption that occurs in varying amounts in different parts of the cell. Specific elements may also be localized within individual cells by the application of monochromatic beams.

The technique was described and the results obtained documented.

*Gastrocamera Photography: Correlation with Roentgenologically Demonstrated Gastric Lesions.* Bernard M. Schuman, M.D., Robert S. Ormond, M.D., Saul A. Rosenblum, M.D., and Robert J. Priest, M.D., Henry Ford Hospital, Detroit, Michigan.

The gastrocamera is a recently developed miniature camera that obtains multiple flash exposures of the stomach. Five millimeter color transparencies are obtained of all portions of the gastric mucosa. It is the exceptional patient in whom it has been impossible to obtain diagnostic pictures of suspected lesions.

The photographs obtained by the gastrocamera have proved of value in the teaching of Gastroenterology and Radiology by correlating the physical characteristics.

*The Value of Lung Biopsy in the Diagnosis of Unusual Pediatric Pulmonary Abnormalities.* Edward B. Singleton, M.D., Milton L. Wagner, M.D., Harvey S. Rosenberg, M.D., Don B. Singer, M.D., and P. M. Gray, M.D., Texas Children's Hospital, Houston, Texas.

The varied and bizarre roentgen patterns found in many pulmonary abnormalities of the pediatric patient are frequently similar and defy diagnosis by the usual clinical and roentgenographic methods. Lung biopsies are frequently invaluable in properly evaluating the underlying pulmonary disease.

The purpose of this exhibit was to show a number of unusual pulmonary diseases encountered in infants and children and to emphasize the importance in reaching a definitive diagnosis.

*Radium Dosage Distribution in Uterine Phantoms.* Jacob Spira, Ph.D., Isamettin Aral, M.D., and Richard Horn, B.A., Boston City Hospital, Boston, Massachusetts.

Presented in the exhibit was a model of a paraffin phantom simulating the uterus, which was used for radium dose measurements in the treatment of endometrial carcinoma applying Heyman packing technique. Different stages of the experimental procedures were shown on roentgenograms and photographs. These included packing of the uterine phantom with the radium capsules, systems used in dosage calculations, isodose curves resulting from differ-

ent geometric configurations of the radium capsules and, finally, the distribution of the dose to various points of anatomic importance around the uterus.

*The Solitary Aorto-Iliac Lesion.* T. W. Staple, M.D., Mallinckrodt Institute of Radiology, St. Louis, Missouri.

The clinical and roentgenographic findings of 30 patients with solitary aorto-iliac occlusive or stenosing lesions were reviewed. The first clinical sign may be embolization to a popliteal artery branch from a large proximal plaque. The pulse may be normal. Therefore, examination of a lower limb artery from the aortic bifucation to and including the branches of the popliteal artery was stressed. Accurate diagnosis is important in these young patients with surgically remediable lesions. Follow-up was made in most instances.

*Salivary Gland Scanning with $Tc^{99m}$ Pertechnetate.* Frederick C. Stebner, M.D., William R. Eyler, M.D., Lucille A. DuSault, A.B., Melvin A. Block, M.D., and Alex P. Kelly, M.D., Henry Ford Hospital, Detroit, Michigan.

The salivary glands of 6 normal subjects, 10 patients with suspected or known parotid gland disease, and 1 patient with suspected submaxillary gland disease were scanned. Correlations with operative and pathologic data have been made.

The pathologic processes identified thus far appeared as areas of decreased uptake in an otherwise functioning gland. The $Tc^{99m}$ is probably concentrated in the duct cells and any process which destroys or displaces duct cells may be shown.

Representative color scans were shown.

*Needle Aspiration Biopsy of the Lung.* G. Melvin Stevens, M.S., M.D., George P. Janetos, M.D., and Glenn A. Lillington, M.D., Palo Alto Medical Clinic, Palo Alto, California.

This exhibit demonstrated the materials, technique, indications and results of the procedure. The simplicity, safety and diagnostic accuracy were emphasized.

There was a scientific essay on the same subject.

*Mammography and Clinical Examination in Screening for Breast Cancer.* Philip Strax, M.D., Louis Venet, M.D., Sam Shapiro, M.D., and Stanley Gross, M.D., New York Hospital, New York, New York.

In a mass screening project for breast cancer involving study and control in a group of 30,000 women each, 55 cancers were found. Of these, 21 were detected on roentgenographic examination alone, 24 on clinical examination alone, and 10 on both.

Differences between the two modalities were examined in relation to age, breast type including structure and size, and type and location of the lesions.

Other areas studied in this project were thermography and 70 mm. mammography. The results of the 70 mm. mammography were presented.

*Percutaneous Aortography and Selective Arteriography in Children.* Rogert G. Sybers, M.D., Ph.D., Dale Cooper, M.D., W. H. Shuford, M.D., and Brit B. Gay, Jr., M.D., Emory University School of Medicine, Atlanta, Georgia.

The Exhibitors briefly described the technique of percutaneous catheterization, aortography, and selective arteriography in children. The value of angiography in the diagnosis of extracardiac lesions was emphasized.

Selected case material was presented to demonstrate the normal findings and pathologic changes encountered in diseases of the central nervous system, thorax and abodmen.

*The Roentgenographic Appearance of the Chest in Patients with Functional Murmurs.* John P. Tampas, M.D., and Paul R. Lurie, M.D., University of Vermont College of Medicine, Burlington, Vermont.

This exhibit evaluated the roentgenographic findings in approximately 500 normal chest examinations in the pediatric age group and in about 250 patients of comparable age who have been determined to have functional murmurs.

Comparison of the chest findings in these two groups suggested that a difference exists in their cardiothoracic relationship.

Analysis and summary of the findings were presented and the roentgenographic

appearance of typical patients with functional murmurs was displayed.

There was a scientific essay on the same subject.

*The Mannitol Pyelogram: A Simplification of the Infusion Technique.* Donald A. Taylor, M.D., Kevin L. Macken, M.D., and Americo S. Fiore, M.D., Lowell General Hospital and Columbia Presbyterian Medical Center, New York, New York.

As currently performed, drip infusion pyelography is a cumbersome and time consuming procedure requiring huge amounts of contrast medium. Since hydration of the patient is the requirement of the procedure, the concentration of contrast agent in the renal collecting system is reduced (hydration causes a decrease in the production of endogenous antidiuretic hormone).

The Exhibitors have devised a technique which gives pyelograms comparable in quality to those achieved utilizing drip infusion technique. The procedure is less cumbersome and requires much less time to perform. The patient is well hydrated and following the scout roentgenogram vasopressin is given intravenously followed immediately by 50 cc. of contrast agent together with 37.5 cc. of mannitol.

The results obtained in 44 patients are extremely encouraging.

*Combined Azygography and Superior Vena Cavography in Patients with Lung Carcinoma.* Arch W. Templeton, M.D., Carl Rinker, M.D., and Carl Jansen, M.D., University of Missouri Medical Center, Columbia, Missouri.

Sixty-three patients with suspected pulmonary carcinoma have been preoperatively evaluated by azygography and superior vena cavography.

Azygography is of significant help in determining resectability and in evaluating the progress of radiation therapy.

Superior vena cavography frequently provides additional information regarding extent of the lesions.

Six patterns of roentgeongraphic abnormality were presented. The importance of proper technique was emphasized.

*Bronchographic Signs of Lung Carcinoma.* Arch W. Templeton, M.D., Lewis Garrotto, M.D., Thomas Llewellyn, M.D., and Charles Simmons, M.D., University of Missouri Medical Center, Columbia, Missouri.

Transtracheal catheter bronchography has been performed by the Exhibitors on 91 patients with carcinoma of the lung.

Five different bronchographic sgins were presented which have been identified with, and frequently are diagnostic of, lung carcinoma.

Difficult differential diagnostic cases are often resolved following adequate bronchography.

*Case of the Day.* Cdr. Elias P. G. Theros, MC, USN, Maj. Maurice M. Reeder, MC, USA, and Maj. Joseph F. Eckert, MC, USAF, The Radiologic Registry of Armed Forces Institute of Pathology, Washington, D. C.

A different case was presented each day. On the basis of history, clinical data and the pertinent roentgenograms exhibited only a single diagnosis was accepted.

The answers were given the following day with discussion and a demonstration in color of the pathology.

*The "Aging" of Fluoroscopic Screens.* E. Dale Trout, D.Sc., and William S. Properzio, B.S., Oregon State University, Corvallis, Oregon.

From the time when fluoroscopic screens were first used, there have been scattered references to reduction in light output due to age, use, etc. References are scarce and usually devoid of quantitative data. A long-range program has been set up in an effort to arrive at some meaningful conclusion. Where possible, old screens have been studied. New screens have been subjected to x-ray exposure, exposure to sunlight, incandescent light and fluorescent light for several months with careful measurements taken monthly under controlled conditions of exposure.

*Retrograde Catheter Aortography in Dissecting Aortic Aneurysms.* Homer L. Twigg, M.D., Louis P. Kirschner, M.D., Peter W.

Conrad, M.D., and Charles E. Hufnagel, M.D., Georgetown University Medical Center, Washington, D. C.

Precise definition of the limits of dissecting aortic aneurysms is essential preoperatively.

Recent reports of complications arising from retrograde catheter aortography in this condition have discouraged many investigators from using this technique. The Exhibitors' experience in a large series of cases examined by this method has been without serious complications. The ability to clearly demonstrate the extent of double and triple lumens of dissection was presented.

Retrograde catheter aortography should not be abandoned for less satisfactory methods of investigation in dissecting aortic aneurysms.

There was a scientific essay on the same subject.

*Pseudoinfections of the Intervertebral Disk and Adjacent Vertebrae?* John L. Williams, M.D., George A. Moller, M.D., and Terrence L. O'Rourke, M.D., Geisinger Medical Center, Danville, Pennsylvania.

Following intervertebral disk surgery, there is occasionally postoperative bone destruction without clinical or bacteriologic evidence of infection. With increasing use of tomography, the Exhibitors have found similar cases of bone destruction in patients who have not had a previous surgical procedure.

The evidence suggests that many of the so-called postoperative complications were actually present prior to surgery and that the pain which led to surgery was due, at least in part, to early and undiagnosed bone necrosis, rather than to a herniated disk.

There was a scientific essay on the same subject.

*The Triangle of Reil.* Paul B. Wollschlaeger, M.D., Gertraud Wollschlaeger, M.D., and Lewis C. Garrotto, M.D., University of Missouri Medical School, Columbia, Missouri.

Postmortem cerebral angiography was employed to demonstrate the correlation between the roentgenographic appearance and the pathologic anatomy of mass lesions which affect the group of middle cerebral arteries within the island of Reil or so-called triangle of Reil. Specific distortion of the tridimensional shape in any relation to the triangle of Reil anteriorly, superiorly, posteriorly, inferiorly, medially or laterally, aids in the exact topographic localization of intracerebral lesions.

The force of impact on the triangle of Reil was pointed out by additional sketches to better advantage of correct interpretation.

*The Sphincters of the Esophagus: Comparative Studies.* Constantino Zaino, M.D., Thomas C. Beneventano, M.D., and Harold G. Jacobson, M.D., Montefiore Hospital, The Bronx, New York.

The normal comparative gross, histologic and roentgen anatomy of the upper and lower sphincters of the esophagus were reviewed by diagrams, transparencies and tables.

The comparative changes in involutional, functional and organic disturbances were chiefly illustrated by roentgenograms.

*Diagnostic Value of Infusion Pyelography.* Peter Zanca, M.D., John L. Doyle, M.D., and Carroll A. Peabody, M.D., University of Texas, South Texas Medical School, San Antonio, Texas.

Infusion "drip" pyelography has proved to be an accurate diagnostic and practical clinical urographic technique. It is especially recommended when the routine intravenous pyelographic examination is contraindicated or indeterminate. This technique produces few side reactions and is easy to perform with the new self contained infusion kit.

When the routine intravenous pyelographic examination is uniformative, the "drip" infusion pyelography will produce a urogram which will allow an accurate diagnosis in more than 95 per cent of these cases.

# THE TECHNICAL EXHIBITS

THE TECHNICAL EXHIBITS of the Sixty-eighth Annual Meeting of the American Roentgen Ray Society held at the Washington Hilton Hotel, Washington, D. C., September 26–29, 1967 exceeded all previous records, not only in Washington—its most favored meeting place—but in all other famous cities where the Society has convened in the past. They were truly a reflection of the magnificent scientific accomplishments of our time.

A kaleidoscopic view of these accomplishments revealed a dazzling array of new diagnostic, therapeutic and isotopic equipment, films, screens, cameras, projectors, radiopharmaceuticals, latest books and many other items displayed in a most artistic fashion.

The continuation, by President-Elect C. Allen Good and the Program Committee, of the policy of holding all Scientific Sessions in the morning, afforded ample time for study of these splendid exhibits. This arrangement proved highly rewarding, both to the exhibitors and to the visiting members and guests of the Society, who availed themselves of this opportunity in full measure.

Another attraction of great convenience was the fact that the Technical Exhibits were placed in the central part of the spacious Exhibit Hall of the Convention floor, surrounded by the unexpectedly large number of Scientific Exhibits, and located in the immediate vicinity of the International Ballroom, where all the Scientific Sessions were held.

Based on their experience of 2 years ago, Mr. Clifford L. Sherratt, Honorary Member and Dr. James C. Cook, Manager of the Annual Meeting, made ample use of the ultramodern facilities of the Hotel and spared no time or effort in organizing this year's exhibits, which reaped such an outstanding success.

The Society expresses its sincerest thanks and deep appreciation to the following exhibiting firms which contributed immeasurably to this outstanding success:

Abbott Laboratories, North Chicago, Illinois: Aerojet Delft Corporation, Plainview, New York; Agfa-Gevaert, Inc., Teterboro, New Jersey; Albert Acan X-Ray, Detroit, Michigan; Americana Encyclopedia, Bethesda, Maryland; Atomic Energy of Canada, Limited, Ottawa, Canada; Automatic Seriograph Corporation, Division of Litton Industries, Des Plaines, Illinois; Barber-Colman Company, Rockford, Illinois; Barnes-Hind Barium Products, Sunnyvale, California; Bar-Ray Products, Inc., Brooklyn, New York; Bell-Craig, Inc., Long Island City, New York; The Bethlehem Corporation, Bethlehem, Pennsylvania; Cenco X-Ray Company, Chicago, Illinois; Controls for Radiation, Inc., Cambridge, Massachusetts; Cook Inc., Bloomington, Indiana; Cordis Corporation, Miami, Florida; Dunlee Corporation, Bellwood, Illinois; Eastman Kodak Company, Rochester, New York; E. I. du Pont de Nemours & Company, Inc., Wilmington, Delaware; Electro-Catheter Corporation, Rahway, New Jersey; Encyclopaedia Britannica, Inc., Chicago, Illinois; Eureka X-Ray Company, Port Washington, New York; General Aniline & Film Corporation, New York, New York; General Electric Company, Milwaukee, Wisconsin; Gordon Consultants, Inc., New York, New York; Grune & Stratton, Inc., New York, New York; Halsey X-Ray Products, Brooklyn, New York; Hoechst Pharmaceutical Company, Cincinnati, Ohio; Hoffrel Instruments Inc., Norwalk, Connecticut; Hogan X-Ray Company, Philadelphia, Pennsylvania; Ilford Inc., New York, New York; Keleket, Waltham, Massachusetts; Lea & Febiger, Philadelphia, Pennsylvania; Leibel-Flarsheim Company, Cincinnati, Ohio; LogEtronics, Inc., Springfield, Virginia; The Machlett Laboratories, Inc., Springdale, Connecticut; Magnaflux Corporation, Chicago, Illinois; Mallinckrodt/Nuclear, St. Louis, Missouri; Mallinckrodt Pharmaceuticals, St.

Louis, Missouri; Medigraph Inc., Nashville, Tennessee; Metrix Inc., Denver, Colorado; Micro X-Ray Recorder, Inc., Chicago, Illinois; Naca Equipment Company, Woodside, New York; Nicholas Laboratories, Limited, White Plains, New York; North American Philips Company, New York, New York; Ohio-Nuclear, Inc., Cleveland, Ohio; Pako Corporation, Minneapolis, Minnesota; Picker X-Ray Corporation, White Plains, New York; Physicians Technical Equipment Company, Inc., New York, New York; Profexray, Division of Litton Industries, Des Plaines, Illinois; W. B. Saunders Company, Philadelphia, Pennsylvania; Schick X-Ray Company, Inc., Wilmette, Illinois; Frank Scholz X-Ray Corporation, Boston, Massachusetts; Siemens Medical of America, Inc., Union, New Jersey; Smith Kline Instrument Company, Philadelphia, Pennsylvania; Sperry Products, Danbury, Connecticut; E. R. Squibb & Sons, Inc., New York, New York; Standard X-Ray Company, Chicago, Illinois; Summit Laboratories, Inc., Hackensack, New Jersey; Technical Associates of New Orleans, Inc., New Orleans, Louisiana; Charles C Thomas, Publisher, Springfield, Illinois; U. S. Catheter & Instrument Corporation, Glens Falls, New York; United States Radium Corporation, Morristown, New Jersey; Westinghouse Electric Corporation, Pittsburgh, Pennsylvania; The Williams & Wilkins Company, Baltimore, Maryland; Winthrop Laboratories, New York, New York; Wolf X-Ray Corporation, Jamaica, New York; X-Ray Identification Corporation, Dearborn, Michigan; Year Book Medical Publishers, Inc., Chicago, Illinois.

# NEWS ITEMS

## THE DIAGNOSTIC AND THERAPEUTIC ASPECTS OF TUMORS OF THE FEMALE GENITAL TRACT

Second Annual Radiotherapy Symposium

The Second Annual Radiotherapy Symposium on "The Diagnostic and Therapeutic Aspects of Tumors of the Female Genital Tract," will be held at the Eden Roc Hotel, Miami Beach, Florida, January 26th through January 28th, 1968.

The University of Miami School of Medicine and the University of Florida College of Medicine are jointly sponsoring the symposium.

Basic and clinical information will be covered and presentations of unusual cases will be discussed in panels. An outstanding group of specialists is being assembled to discuss the various aspects of diagnosis, treatment and prognosis of tumors of the female genitalia.

For further information please write to Mario Vuksanovic, M.D., Director, Division of Radiation Therapy, Jackson Memorial Hospital, 1700 N.W. 10th Avenue, Miami, Florida 33136.

## CHILDREN'S HOSPITAL MEDICAL CENTER OF NORTHERN CALIFORNIA

Postgraduate Course on Advances in Pediatric Roentgenology

The Department of Radiology of Children's Hospital Medical Center of Northern California will present a Postgraduate Course on "Advances in Pediatric Roentgenology," April 16–18, 1968.

The Guest Faculty will include: G. B. Clifton Harris, M.D., Richard G. Lester, M.D., Thomas H. Newton, M.D., Frederic N. Silverman, M.D., Edward B. Singleton, M.D., and Howard L. Steinbach, M.D.

The Panel Moderators will be: Robert S. Arkoff, M.D., Peter Kane, M.D., and William H. Northway, Jr., M.D.

The Sixteenth Annual Postgraduate Seminar and Clifford D. Sweet Memorial Lecture will follow the course on April 19 and 20, 1968. The subject will be "Tumors in Childhood."

For further information please contact Hooshang Taybi, M.D., Department of Radiology, Children's Hospital Medical Center of Northern California, 51st and Grove Streets, Oakland, California 94609.

## COLLEGE OF PHYSICIANS AND SURGEONS OF COLUMBIA UNIVERSITY

Postgraduate Course in Radiology of the Gastrointestinal Tract

A Postgraduate Course in "Radiology of the Gastrointestinal Tract" will be given by the Department of Radiology under the direction of William B. Seaman, M.D., at the Columbia-Presbyterian Medical Center, Alumni Auditorium, 168th Street, New York, N. Y., May 1–4, 1968.

In addition to the faculty of the College of Physicians and Surgeons, the guest faculty will include: Prof. Solve Welin of Malmo, Sweden, Dr. Richard Fleming, Dr. Harold Jacobson, Dr. Richard Marshak, Dr. Harry Z. Mellins, and Dr. Bernard Wolf.

Inquiries should be addressed to Dr. Melvin D. Yahr, Assistant Dean for Graduate Medical Education, 630 West 168th Street, New York, N. Y. 10032.

## AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE

Symposium on Physics of Diagnostic Radiology

A symposium on "Physics of Diagnostic Radiology" will be held at the University of California Medical Center, San Francisco, California, June 13–15, 1968. The meeting is being sponsored by the American Association of Physicists in Medicine.

Specialists in the different aspects of the physics of diagnostic radiology have been invited to discuss: Imaging System, Instrumentation in Diagnostic Radiology, and Recent Developments in X-Ray Diagnosis. One session will be devoted to nonradiologic techniques such as Thermogra-

phy and Ultrasonics which are frequently used in Departments of Radiology.

Proffered papers will be accepted where they fit into the theme of the program. Abstracts should be submitted to Program Chairman, Mary Louise Meurk, Claire Zellerbach Saroni Memorial Tumor Institute of San Francisco, by February 23, 1968.

## ARMED FORCES INSTITUTE OF PATHOLOGY (AFIP)

### Annual AFIP Course in Oral Pathology

This course will be held March 4–8, 1968. It provides dentists, physicians and trainees in oral and general pathology a fundamental knowledge of various aspects of oral disease, and brings them abreast of recent developments in this field. Developmental disturbances of the head, neck and oral region, inflammatory diseases of the oral mucosa and jaws, the oral manifestations of certain systemic diseases and neoplasms of the oral cavity and related structures will be discussed in detail and their clinical, roentgenographic and microscopic characteristics illustrated. The lectures are correlated with case presentations, microscopic slide seminars and round table discussions.

Applicants must be members of the Medical or Dental Corps. Qualified civilians will be accepted on a space available basis.

To apply, contact the Director, Armed Forces Institute of Pathology, ATTN: MEDEM-PAD, Washington, D. C. 20305.

## THE DONNER LABORATORY

### Fellowship in Radiologic Science

The Donner Laboratory, University of California, announces an opportunity for advanced study and research in radiologic science. Activities include investigation of the biologic effects of high-energy particles and participation in the use of high-energy particles in therapy.

One or two courses in the graduate program in biophysics and medical physics may be taken concurrently.

Applicants must have completed residency training in radiology or comparable training. Stipend will depend on qualifications and experience.

Application forms are available from: John H. Lawrence, M.D., Director, Donner Laboratory, University of California, Berkeley, California 94720.

## UNIVERSITY OF MIAMI SCHOOL OF MEDICINE

### Postgraduate Seminar on Radiographic Diagnosis of Head and Neck

The University of Miami School of Medicine presents a Postgraduate Seminar on "Radiographic Diagnosis of Head and Neck," March 21–24, 1968, at the Fontainebleau Hotel, Miami Beach, Florida.

The Director is Raymond E. Parks, M.D., Chairman, Department of Radiology, University of Miami School of Medicine; the Chairman of the Scientific Program Committee is F. P. Gargano, M.D., and the Chairman of the Registration Committee is Albert Weinfeld, M.D.

In addition to the local faculty the Guest Faculty includes: Raymond A. Brinker, M.D., St. Louis, Missouri; Giovanni Di Chiro, M.D., Bethesda, Maryland; G. H. du Boulay, M.D., London, England; Lewis E. Etter, M.D., Pittsburgh, Pennsylvania; E. S. Gurdjian, M.D., Detroit, Michigan; Lee A. Hadley, M.D., Syracuse, New York; William N. Hanafee, M.D., Los Angeles, California; Galdino E. Valvassori, M.D., Chicago, Illinois; Alex A. MacMillan, Jr., M.D., Boston, Massachusetts; P. E. S. Palmer, F.F.R., Cape Town, South Africa; Gordon Potts, M.D., New York, New York; and William B. Seaman, M.D., New York, New York.

For further information, please write to Mrs. L. R. Kelley, Department of Radiology, Jackson Memorial Hospital, Miami, Florida 33136.

# BOOKS RECEIVED

*Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.*

## BOOKS RECEIVED

ESSENTIALS OF ROENTGEN DIAGNOSIS OF THE SKELETAL SYSTEM. By Lester W. Paul, M.D., Professor of Radiology, The University of Wisconsin Medical School; and John H. Juhl, M.D., Professor of Radiology and Chairman of the Department of Radiology, The University of Wisconsin Medical School. Cloth. Pp. 288, with 410 illustrations. Price, $12.50. Hoeber Medical Division of Harper & Row, Publishers, 49 East 33rd Street, New York, N. Y. 10016, 1967.

DIABETES MELLITUS: DIAGNOSIS AND TREATMENT. Volume II. Edited by George J. Hamwi, M.D., and T. S. Danowski, M.D. Prepared under the auspices of the Committee on Professional Education of the American Diabetes Association, Inc. Cloth. Pp. 250. Price, $2.50. American Diabetes Association, Inc., 18 East 48th Street, New York, N. Y. 10017, 1967.

MALIGNANT TUMORS OF THE THYROID GLAND. By Edith Beregi, R. Jankovics, and Z. Brasch. Cloth. Pp. 81, with many illustrations. Price, $6.00. Akadémiai Kiadó, Publishing House of the Hungarian Academy of Sciences, Budapest V. Alkotmány U. 21, Hungary, 1967.

CASE OF THE DAY. By Benjamin Felson, M.D., Professor and Director, Department of Radiology, University of Cincinnati College of Medicine; Director, Departments of Radiology, Cincinnati General, Children's, Daniel Drake, Dunham, Christian R. Holmes, and Longview Hospitals, Cincinnati, Ohio; Special Consultant, United States Public Health Service; Consultant to the Dayton and Cincinnati Veterans Administration Hospitals; Consultant, Walter Reed Hospital (U. S. Army); Consultant, Armed Forces Institute of Pathology; Consultant to Surgeon General of Air Force, and Wright Patterson AFB Hospital; and Jerome F. Wiot, M.D., Professor of Radiology, University of Cincinnati College of Medicine; Co-Director of Department of Radiology, Cincinnati General Hospital; Consultant to Cincinnati Veterans Administration Hospital, Cincinnati, Ohio. Cloth. Pp. 84, with many illustrations. Price, $8.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1967.

CARCINOGENESIS: A BROAD CRITIQUE. A collection of papers presented at the Twentieth Annual Symposium on Fundamental Cancer Research, 1966. Cloth. Pp. 775, with many illustrations. Price, $16.00. Published for The University of Texas, M. D. Anderson Hospital and Tumor Institute, Houston, Texas. The Williams & Wilkins Company, 428 East Preston Street, Baltimore, Md. 21202, 1967.

ROENTGENOLOGIC DIAGNOSIS. A Complement in Radiology to the Beeson and McDermott Textbook of Medicine. Volume I. By J. George Teplick, M.D., Clinical Associate Professor of Radiology, Hahnemann Medical College; Marvin E. Haskin, M.D., F.A.C.P., Clinical Assistant Professor of Radiology, Hahnemann Medical College; and Arad P. Schimert, M.D., Formerly Associate Professor of Radiology, Hahnemann Medical College. Cloth. Pp. 527, with many illustrations. Price, $38.00 for set of two Volumes. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1967.

THE TRUTH ABOUT CANCER. By Charles S. Cameron, M.D., President, The Hahnemann Medical College and Hospital of Philadelphia; formerly Medical and Scientific Director, American Cancer Society. Paper. Pp. 285, with some illustrations. Price, $1.50. Collier Books, 866 Third Avenue, New York, N. Y. 10020, 1967.

HYSTEROSALPINGOGRAPHY. By Alvin M. Siegler, M.D., D.Sc., Clinical Associate Professor in Obstetrics and Gynecology, State University of New York, Downstate Medical Center; Attending Physician in Obstetrics and Gynecology, Kings County Hospital Center and the Brookdale Hospital Center; Director of the Sterility Clinic, Kings County Hospital Center, Brooklyn, N. Y. Cloth. Pp. 418, with 543 illustrations. Price, $21.00. Hoeber Medical Division of Harper & Row, Publishers, 49 East 33rd Street, New York, N. Y. 10016, 1967.

DIAGNOSTICA PNEUMORADIOGRAFICA IN MEDICINA INTERNA. By Luigi Lacroix, Libero Docente e Aiuto della Clinica Medica, dell'Università di Torino, Italy. Cloth. Pp. 350, with many illustrations. Price, L 20,000. Il Pensiero Scientifico Editore, Rome, Italy, 1967.

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# ABSTRACTS OF RADIOLOGICAL LITERATURE

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## ROENTGEN DIAGNOSIS

### Head

Fischgold, H. Particularités et limites de la radio-neuro-ophthalmologie. (Possibilities and limitations of radioneuro-ophthalmology.) *J. de radiol., d'électrol. et de méd. nucléaire*, May, 1967, *48*, 243–252. (From: Service de Neuroradiologie et d'E.E.G. Groupe Hospitalier Pitié-Salpétrière, 83, boulevard de l'Hôpital, Paris, 13, France.)

The author presents a rather detailed historic-philosophic review of the developments in diagnostic radiology pertaining to ophthalmology and neuro-ophthalmology during the past 30 years.

This includes the development of several radiographic projections and techniques, their place and limitations, as well as a consideration of the problems which are still faced and which remain unsolved.

The ophthalmic radiologist is concerned mainly with a study of the orbital bone structure, the eyeballs, lacrimal ducts, and foreign body localization; the neuroradiologist is concerned with a general study of the skull and its contents, the optic canal and the orbital fissures—areas of common interest to the radiologist, ophthalmologist, neurologist and neurosurgeon.

Included is a discussion of the personnel, equipment and space requirements. The radiologic techniques and procedures used and their indication are considered in detail. They are conveniently presented in table form as follows:

1. Radiography of the skull and orbits—standard projections, special projection and tomography are basic examinations in the study of all neuro-ophthalmic conditions.

2. Radiography following intra-orbital injection of contrast media—in a study of unilateral exophthalmos and intra-orbital tumors.

3. Angiography of the internal carotid artery—in the presence of decreased visual acuity, papilledema, hemianopsia, exophthalmos and suspicion of angiomas and aneurysms.

4. Angiography of the external carotid artery—in a study of meningiomas, angiomas of the orbital wall, angiomas of the eyelid.

5. Angiography of the vertebral artery—in a study of lateral homonymous hemianopsia, homonymous scotomas.

6. Orbital phlebography—in a study of exophthalmos, venous malformations of the orbit and cavernous sinus syndrome.

7. Fractionated pneumoencephalography—in studies of the third ventricle, lesions of the occipital region, the optic chiasma, sellar and parasellar tumors, the olfactory region and cerebral peduncles.

8. Lipiodol opacification of the ventricles and cerebral aqueducts—in studies of lesions of the 3rd ventricle, the nuclei of the 3rd, 4th, 5th and 6th nerves.

9. Radioisotope studies of the orbits—in exophthalmos, tumor metastasis as well as evaluation of primary lesions.

Four figures and tables, and 5 reproductions of roentgenograms accompany this article.—*William H. Shehadi, M.D.*

Berger, Robert L., Sidd, James J., and Ramaswamy, K. Retrograde vertebral-artery flow produced by a correction of subclavian-steal syndrome. *New England J. Med.*, July 13, 1967, *277*, 64–69. (From: Departments of Medicine and Surgery, St. Elizabeth's Hospital and Tufts University School of Medicine, 136 Cambridge Street, Boston, Mass. 02135.)

The subclavian-steal syndrome is a well-known phenomenon associated with occlusion of the subclavian artery proximal to the origin of the vertebral artery. Blood is diverted from the opposite vertebra artery into the obstructed side in order to profuse the distal subclavian bed with blood that was intended for the cerebral circulation. The symptoms are similar to basiler artery insufficiency and may be precipitated by exertion of the involved extremity.

Two patients are reported who had complete obstruction on one side with partial obstruction on the other. The steal phenomenon presented on the side of the complete obstruction. Following surgical correction of the complete obstruction, there was produced retrograde vertebral flow on the side of the partial obstruction. This is an unrecognized feature of the syndrome which is well demonstrated by these 2 patients.

The first patient, a 49 year old man, presented with syncopal episodes for 3 years precipitated by a physical exertion, driving a bus. The aortic arch angiogram demonstrated a complete occlusion of the right and partial occlusion of the left subclavian artery. Both lesions were proximal to the vertebral arteries. Late roentgenograms showed retrograde filling of the right vertebral and distal subclavian artery. A right subclavian endarterectomy was performed. A repeat aortic arch angiogram on the tenth postoperative day showed excellent filling of the right subclavian and vertebral arteries. On late roentgenograms there is now seen to be retrograde flow in the left vertebral artery. Repair of the left subclavian artery was then done. The postoperative course was uneventful, and when last seen 6 months after operation the patient was free of cerebral symptoms.

The second case was a 63 year old woman who experienced dizziness and light headedness for 2 years. An aortic arch angiogram demonstrated total obstruction of the innominate artery and partial obstruction of the left subclavian artery near its origin.

The left vertebral artery was visualized. Late roentgenograms demonstrated retrograde filling of the right carotid, vertebral, and subclavian arteries. At operation there was found to be a totally occluded innominate artery. A graft was inserted and a good blood flow was obtained. An aortic arch angiogram performed on the tenth postoperative day demonstrated a functioning graft with adequate foreward flow through the subclavian, carotid, and vertebral arteries on the right side. Late roentgenograms now demonstrated retrograde flow into the left vertebral artery. It was decided to delay additional surgery, and the patient when last seen was much improved.

The vertebral basilar circulation is a closed hydraulic system and the direction of flow within it is governed by the pressure relation between different parts of the system. It is easy then to understand how the correction of a complete obstruction with restoration of a normal pressure gradient will be followed by reversal of flow on the partially occluded opposite side due to the decreased pressure. The subclavian-steal syndrome is reproducible in the laboratory. The syndrome described in this communication can similarly be duplicated in dogs. Obstruction of one subclavian artery and partial constriction of the contralateral subclavian vessels result in the retrograde vertebral flow on the totally occluded side and forward flow on the partially obstructed side. Release of the totally occlusive clamp, a maneuver simulating endarterectomy in patients, converts vertebral blood flow from the antegrade to the retrograde on the partially obstructed side.

It is important to recognize this feature of the subclavian-steal syndrome and to repeat aortic arch angiography after the initial surgical correction in patients with this particular vascular pattern.—*S. A. Kaufman, M.D.*

CHATTERJEE, S. K., and MAZUMDER, J. K. Massive fibro-osseous dysplasia of the jaws in two generations. *Brit. J. Surg.*, May, 1967, *54*, 335–340. (From: Nilratan Sircar Medical College Hospital, University of Calcutta, Calcutta, India.)

Nonmalignant tumors of the jaws have been classified into ectodermal and mesodermal, each having been subdivided into surface and diffuse tumors. These have been further subdivided with localized surface tumors resulting from fibroma, fibrous osteoma, osteofibroma and osteoma. The localized diffuse forms consist of fibrous dysplasia, fibro-osseous dysplasia and leontiasis ossea.

The authors report an unusual variant of fibro-osseous dysplasia occurring in a father and his 2 sons with the disorder strictly confined to the connective tissue of the oral mucoperiosteum. The initial case, a 25 year old Bengali Muslim male, first was seen with hideous swellings of the face and mouth and a history of progressive increase of the swelling of the upper jaw since childhood. Following a successful 5 stage surgical procedure, an excellent plastic result was obtained. Histologically, the tissue suggested a diagnosis of fibrous dysplasia and there was no recurrence of the disease process.

Six years later the authors examined 2 boys, children of the patient, aged 4 years and aged 8 months, and in both the disease was evident. This suggested strongly that the disease is a disorder of development rather than a neoplasm with transmission from one generation to the next as a dominant trait. The disorder is apparently a failure of the restraining influence of the oral mucoperiosteum resulting in uninhibited proliferation of the underlying connective tissue similar to metaphysial aclasis. There was no evidence of the condition in any member of the previous generations or in any other member of the same family.—*James R. Knapp, M.D.*

## NECK AND CHEST

AMBIAVAGAR, M., CHAN, O. L., PAIS, F. W., and D'BRAS, B. E. G. The acid aspiration syndrome. *Singapore M. J.*, March, 1967, *8*, 35–42. (From: Anaesthetic Unit, General Hospital, Singapore, Singapore.)

Seven patients with gastric contents aspirated into their lungs are reported.

All patients were either unconscious or somnolent due to disease or drugs at the time of aspiration. The disease was most frequently associated with obstetric anesthesia. Vomiting and aspiration were not clinically apparent in all patients. Progressive bronchospasm, cyanosis, tachycardia and right heart failure were the chief findings. If the episode of regurgitation was noticed, the diagnosis was clear, but if not, differentiation from pulmonary embolism, atelectasis, pneumothorax, bronchopneumonia, acute bronchial asthma, and cardiac failure can be difficult.

Chest roentgenograms showed irregular bilateral infiltrates without mediastinal shift or pleural fluid, and the pathologic changes disappeared completely in those patients who recovered.

Treatment consisted of endotracheal suction if the episode occured during anesthesia. Bronchospasm was treated with adrenaline, aminophyline and hydrocortisone, and prophylactic antibiotics were provided.

Four of the 7 patients died from cardiovascular collapse that did not respond to treatment with steroids, transfusions, vasopressors or digitalis. The postmortem findings in 2 patients are described; autopsy should be performed within 2 hours after death or the characteristic features of the disease can be obscured by postmortem changes. The lungs were hemorrhagic, edematous and hyperemic and the tracheobronchial tree contained exudative material. Microscopically, the alveoli were filled with red blood cells, fluid, desquamated epithelium and undigested food particles.

This disease often occurs in otherwise healthy persons with no cardiac or respiratory problems. Current methods of treatment are inadequate. When it occurs during induction of general anesthesia, it is truly an iatrogenic disease. Further study of the pathophysiology of acid aspiration into the lungs, and of a more effective therapeutic regimen is stressed.—*Mark D. Reiss, M.D.*

Wilson, J. A. S., and Munro, D. D. Surgical treatment of mycotic infections of the lung. *Canad. M.A.J.*, July 22, 1967, *97*, 166–173. (From: Department of Surgery, Royal Edward Chest Hospital, Royal Victoria Hospital and McGill University, Montreal, Quebec, Canada.)

A limited group of patients with pulmonary mycotic infections including nocardiosis, cryptococcosis, aspergillosis, coccidiomycosis, and histoplasmosis were treated by operation. The diagnosis was either proven preoperatively by culture or after operation by examination of the resected specimen.

The operation was for one of two reasons: (1) to establish a diagnosis in cases of presumed neoplasm; and (2) as definitive treatment of known chronic infections with harmful sequelae of the fungus.

The operation was considered indicated: (1) to remove caseous lymph nodes of histoplasmosis when causing bronchial obstruction or other phenomena such as compression of the recurrent laryngeal nerve; (2) to treat chronic cavitary infections; and (3) to treat complications.

In the authors' experience, amphotericin B was a highly toxic drug and was not felt to be necessary as a preoperative or postoperative measure in operation of a patient with histoplasmosis. They do, however, believe that it is indicated in patients with cryptococcosis as a deterrent to meningitis.

The authors had no postoperative mortality and all of their final results were gratifying with a cure in each case.

Multiple case reports are given in the paper.—*William Merrell, M.D.*

Weisser, K., Wyler, F., and Gloor, F. Pulmonary veno-occlusive disease. *Arch. Dis. Childhood*, June, 1967, *42*, 322–327. (From: Departments of Paediatrics and Pathology, University of Basle, Switzerland.)

Pulmonary veno-occlusive disease (also known as isolated pulmonary venous sclerosis and obstructive disease of the pulmonary veins) is an isolated primary obstructive disease of the small and medium-sized pulmonary veins.

The authors report a case in a 15 year old female who had had dyspnea on exertion for 8 months prior to admission; this was gradually progressive with weakness, fatigue, and weight loss. She developed jaundice, acholic stools and dark urine at 4 months prior to admission, which was diagnosed as infectious hepatitis apparently unrelated to her pulmonary disease. However, her general condition deteriorated and the parents finally consented to hospitalization.

On admission, a tachycardia of 140 was present with cardiac findings suggestive of increased pulmonary artery pressure and distented neck veins. The liver was palpable 8 cm. below the right costal margin and the extremities were cold. The child was orthopneic with increased respiratory rate and bilateral basilar rales were present.

Chest roentgenogram revealed moderately enlarged right ventricle and right atrium and distention of the pulmonary artery and its proximal branches. The left ventricle and atrium were not enlarged. Slight mottling was present in the lower lung fields with Kerley A and B lines.

Treatment consisted of digitalization, oxygen, diuretics and corticosteroids, but the deterioration continued and the child died 8 weeks after admission. Necropsy revealed a greatly enlarged heart with massive dilatation and hypertrophy of the right atrium and ventricle and normal left atrium and ventricle. The lungs were congested and edematous without effusion. The histologic examination demonstrated the medium-sized and small veins to be completely obliterated in most places. Their lumen was filled with an almost acellular fibrous tissue. The large pulmonary veins were normal. The small muscular pulmonary arteries showed medial hypertrophy and "onion skin" concentric thickening of the intima. No arterial thrombosis was present and the vascular changes were limited to the lungs.

The etiology is unknown and the disease has always been fatal. A striking analogy between pulmonary veno-occlusive disease and hepatic veno-occlusive disease (Budd-Chiari syndrome) is noted. —*John Truksa, M.D.*

Auger, Pierre, and Wigle, E. Douglas. Sudden, severe mitral insufficiency. *Canad. M. A. J.*, June 10, 1967, *96*, 1493–1503. (From: Department of Medicine, University of Toronto, and the Cardiovascular Unit, Toronto General Hospital, Toronto, Canada.)

The authors have distinguished between the mitral insufficiency resulting from rupture of the chordae tendineae or papillary muscle in previously normal valves and those in patients with rheumatic mitral valves. The former syndrome is characterized by a normal sinus rhythm and lack of gross left atrial enlargement. The latter is characterized by atrial fibrillation and gross left atrial enlargement.

Thirty-seven patients with pure severe mitral insufficiency were divided into 4 groups:

Group 1. Sudden severe mitral insufficiency in previously normal hearts (5 patients).

Group II. Severe chronic rheumatic mitral insufficiency (25 patients).

Group III. Same as Group II with superimposed ruptured chordae tendineae (5 patients).

Group IV. Severe, chronic nonrheumatic mitral insufficiency.

The left atrial-thoracic ratio was obtained by measuring the vertical and horizontal left atrial diameters on a posteroanterior chest roentgenogram, summed and divided by 2. This average was then divided by the transthoracic diameter.

In Group I, all were males with no history of rheumatic fever and soon developed right and left heart failure after the sudden onset of a harsh, widely propagated apical pansystolic murmur and thrill and faint or absent third heart sound. All were in sinus rhythm and showed no or only slight left atrial enlargement. The ruptures were spontaneous in 2, followed a myocardial infarction in 2 and followed a bout of bacterial endocarditis in 1. No patient in Group I had a left atrial-thoracic ratio of greater than 33 per cent and none in Groups II and III had a ratio less than 36 per cent.

In Groups II and III, most were females with a high incidence of rheumatic fever, long symptomatic histories and a high incidence of atrial fibrillation and large left atrium.

In Group IV, all were males without rheumatic fever, long symptomatic periods, 1 with atrial fibrillation and 1 without and only mild left atrial enlargement.

The authors point out that the time of investigation after the development of mitral insufficiency is also important in separating these two syndromes since the increase in compliance of the left atrium may be time dependant as well as the result of chronic left atrial fibrillation. Early surgical repair prior to the onset of complications offers the greatest benefit to these patients with sudden severe mitral insufficiency.—*Captain James R. Stevenson, MC USA.*

BARBACCIA, F., and DECHIARA, C. Anomalie d'Ebstein et malformations des valves tricuspidiennes siégeant à gauche dans la transposition corrigée des gros vaisseaux (étude radiologique). (Ebstein's anomaly and malformations of tricuspid valves lying to the left in corrected transposition of the great vessels [radiologic study].) *Ann. de radiol.*, 1966, *9*, 845–853. (From: Istituto di Radiologia e Medicina Nucleare, e Divisione Cardiodiagnostica dell'Ospedale Maggiore di Milano, Italy.)

In corrected transposition of the great vessels the following changes are observed: (a) abnormal position of the aorta and pulmonary artery; (b) morphologic alterations of the ventricular cavities; and (c) transposition of the atrio-ventricular valves, so that the mitral valve is located to the right and the tricuspid to the left.

Other cardiac malformations are often found, especially an interventricular communication. The co-existing malformations of the tricuspid valves can be summarized as follows:

1. The valves are inserted in the ventricular walls by short cords and by small papillary muscles.

2. The valves are fused to the ventricular walls to the point of being sometimes unrecognizable.

3. Abnormal insertion of the valves below the annulus fibrosus, associated or not with the above alterations; in these cases, the typical picture of Ebstein's anomaly is realized, as it involves the mitral valve of a normally orientated heart.

These anomalies rarely involve the tricuspid valves located to the right in normally orientated hearts, and are found with increasing frequency in the corrected transposition of the great vessels (CTGV). The complex embryologic development of the tricuspid valve might explain this fact: the mitral valve is rarely involved when located to the left or as in CTGV.

The authors then review the literature on the alterations of the tricuspid valves seated on the left in CTGV and the associated cardiac malformations. They summarize the characteristic findings of this syndrome as revealed by autopsy and they present the salient features of the case history, with regard to cardiac auscultation, electrocardiography and the diagnostic information provided by catheterization, plain chest roentgenography and angiocardiography.

The authors conclude that malformations of the left atrio-ventricular valve in CTGV can be diagnosed only by angiocardiography in the living; only one such case was reported by Ellis *et al.* They review 13 cases of CTGV on their own and present 2 cases of Ebstein's disease involving the left atrio-ventricular valves diagnosed by angiocardiography; they present reproductions of plain chest roentgenograms and anteroposterior and lateral roentgenograms of the opacified cardiac cavities.—*H. P. Lévesque, M.D.*

CARLSSON, ERIK, and MILNE, ERIC N. C. Permanent implantation of endocardial tantalum screws: a new technique for functional studies of the heart in the experimental animal. *J. Canad. A. Radiologists*, June, 1967, *19*, 304–309. (From: Department of Radiology and the Cardiovascular Research Institute, University of California Medical Center, San Francisco, California.)

A method of implanting minute tantalum screws in the dog endocardium is described. Readily available material such as Ödman catheters and flexible guide wires are employed. With the use of controll-

able flexible tips, all aspects of the endocardium can be reached. Cardiac chamber volumes are calculated by taking dimensions from roentgenograms in anteroposterior, lateral and oblique projections. Angiocardiography is used for comparison of the volumes outlined and the illustrations in the article show good correlation between the two methods. Ectopic beats are the only electrocardiographic abnormalities noted during the procedure.

The authors point out that the technique will allow long-term study of heart function, whereas conventional angiocardiography, while giving accurate volumetric measurements, allows only a brief functional study of 2–3 heartbeats. The introduction of a bolus of contrast medium is believed to initiate functional changes in itself. Their immediate application of this technique is to determine how much volumetric change the introduction of contrast medium for angiocardiography causes. Beyond this they describe an experimental model encompassing electromagnetic flowmeters at the aortic root and indwelling coronary artery catheters in addition to the tantalum screws, so that a wide variety of stimuli applied to the heart can be studied over an extended period of time.—*Everett H. Johnston, M.D.*

Ebert, Paul A., Gaertner, Robert A., and Zuidema, George D. Traumatic diaphragmatic hernia. *Surg., Gynec. & Obst.*, July, 1967, *125*, 59–65. (From: The Department of Surgery, The Johns Hopkins University School of Medicine, Baltimore, Md.)

The authors studied 53 patients who underwent operation at the Johns Hopkins Hospital for repair of a traumatic diaphragmatic hernia between the years 1926 and 1965. The patients were separated into two groups depending on whether the diaphragmatic hernia resulted from a penetrating injury of the abdomen or chest or whether it was a result of blunt trauma. There were 29 patients who underwent operation for repair of a diaphragmatic hernia because of the penetrating injury. In this group the chest roentgenogram was negative in 12 patients and positive in 16. In only 6 patients, however, was the examination diagnostic of a diaphragmatic hernia. The diaphragmatic defect was in general smaller in this group but had to exceed 3 cm. in length to be considered a hernia.

Twenty-four patients were operated upon for repair of hernia resulting from indirect blunt trauma. The trauma varied from a minimal bump to a massive crushing injury to the chest. In 22 of these patients the hernia occurred through the left hemidiaphragm, and in the majority was in the posterior central area extending medially to the pericardial attachment.

Traumatic diaphragmatic hernias resulting from penetrating injuries are usually diagnosed at the time of surgery, as a rule immediately performed because of the nature of the injury. It is the surgeon's responsibility to exclude the possibility of a ruptured diaphragm. Ruptures occurring as a result of blunt injury occur on the left side in approximately 98 per cent of the patients. It is not uncommon to find the stomach, spleen, left lobe of the liver, and the colon within the chest. Incarceration and strangulation may result as with any type of hernia. Respiratory symptoms from compressed lung and symptoms of chronic intestinal obstruction are more common when the hernia has been present for considerable period of time. Herniation of the abdominal viscera into the chest may occur at the time of rupture or may be delayed for some period of time and the diagnosis may be delayed for months or years following the initial injury. The injury itself may have been extremely minor, and one must suspect a traumatic diaphragmatic hernia if the classical physical findings of intestinal contents in the chest are present and if the roentgenogram of the chest shows a loss of the contour in the left diaphragm with air fluid levels in the left lower lung field. It must be born in mind that chest examination immediately following injury may be entirely normal, and the herniation may not occur until sometime later.—*S. A. Kaufman, M.D.*

## Abdomen

Reid-Smith, A. Tannic acid and the barium enema with reference to dosage. *J. Canad. A. Radiologists*, June, 1967, *19*, 335–337. (From: Woodstock General Hospital, Woodstock, Ontario, Canada.)

It became apparent in 1963 that tannic acid used as an additive in the barium enema was causing liver necrosis and subsequent death from liver failure in an occasional patient. In March 1964 the use of tannic acid in barium enemas was banned by the Food and Drug Administration of the United States. In Great Britian, the attention of all radiologists was called to the reports concerning liver necrosis. Most radiologists have discontinued using tannic acid and many agree that it has resulted in a considerable loss of accuracy in diagnosis during examination of the colon.

Prior to the publicity concerning the dangers of tannic acid, little was known about the actual amount of tannic acid being used per barium enema. Originally a level tablespoon of tannic acid in 2 quarts of fluid was recommended. Two heaping tablespoons in some areas was commonly used. Many radiologists probably did not know how much tannic acid they were using. Several recent papers have suggested that smaller doses of tannic acid might be safe and still effective in producing good evacuation and nice mucosal pattern studies.

The purpose of this paper and investigation was to attempt to discover if tannic acid in small amounts is of any value. The author uses 2 ounces of

castor oil the evening before the examination and if this is effective it is the only preparation that is given. The barium enema solution used consists of 20 ounces of barium mixed in 60 ounces of tap water. During the investigation 2.5 grams of tannic acid was added to the barium enema in half of the cases studied. This resulted in 0.15 per cent solution of tannic acid. The other half of the patients requiring enemas were given no additive. After evacuation all patients had post evacuation roentgenograms and the amount of mucosa that was clearly visualized was estimated from these roentgenograms. Two hundred and fifty barium enema examinations using 2.5 grams of tannic acid and 250 barium enema examinations without tannic acid were evaluated. The study indicated that there was some improvement in the percentage of mucosa demonstrated in those cases in which tannic acid had been used but the improvement was not very marked.

In view of the known toxicity, it does not appear justifiable to use small doses of tannic acid in the barium enema since there is only slight improvement in the demonstration of mucosal pattern on post evacuation roentgenograms.—*Richard E. Kinzer, M.D.*

NEELY, M. G., and GILLESPIE, G. Peutz-Jeghers syndrome: sporadic and familial. *Brit. J. Surg.*, May, 1967, *54*, 378–381. (Fom: The Royal Infirmary, Greenock, Scotland.)

Peutz-Jeghers syndrome is a condition in which intestinal polyposis is associated with melanotic macules around the mouth, palms, and soles. The condition is said to be inherited as a mendelian dominant trait. Occasional isolated cases have been described. The authors report 3 cases 1 of whom seems to be an isolated case while the other 3 are members of the same family. The article is profusely illustrated with very nice color reproductions. Two of the familial cases had been previously reported.

All 4 cases described were seen clinically with epigastric pains and all had polyps in the duodenum or jejunal area. At operation, Case I was found to have intussusception with a bilobed polypoid mass producing the intussusception. A further polyp was found 60 cm. distal to this. Case II was found to have intussusception of a jejunal polyp. Case III was operated on two different times. In January of 1964, resection of a segment of small bowel was required for an intussuscepted jejunal polyp. In August of 1964, two gastric polyps were removed but the patient continued to have intermittent episodes of abdominal pain. Barium meal study revealed multiple intestinal polyps. Case IV was the father of Cases II and III. He was originally suspected of having Addison's disease but on two separate occasions operation was carried out for the relief of intussusceptions due to jejunal polyps.

Diagnosis was made by observation of typical pigmentation on the lips, buccal mucosa, face and dorsum of the digits which appears early in childhood. It is noted that in about 90 per cent of the cases there are polyps present and these are usually multiple and occur in the small bowel. About 50 per cent of the cases have polyps in the large bowel. Intussusception is the commonest presenting feature. Bleeding from the polyps is extremely common. Malignant change is very exceptional and perhaps only 1 case of malignant change has been documented.—*Richard E. Kinzer, M.D.*

FRASER, G. C. and WILKINSON, A. W. Neonatal Hirschsprung's disease. *Brit. M. J.*, July 1, 1967, *3*, 7–10. (From the Hospital for Sick Children, Great Ormond Street, London W. C. 1, England.)

Swenson and Bill (1948) first showed that Hirschsprung's disease could be cured by resection of the abnormally innervated distal segment of rectum and colon and since that time, the clinical status of the disease has been transformed. Although the early onset of the clinical disturbance in Hirschsprung's disease has been emphasized repeatedly, the diagnosis was seldom made or the disease treated in the early days or weeks of life. Estimates of the mortality in the first year approximate 50 per cent and these children probably die of gastroenteritis superimposed on the intestinal obstruction caused by the unrecognized aganglionosis. The disease has been shown to affect the developing lower bowel at a very early age with some degree of intestinal obstruction being present weeks before birth. This is indicated by the hypertrophy of the intestinal muscle of the transitional segment and most distal part of the normally innervated segment which may readily be seen at operation just after birth.

With few exceptions, the clinical features by which a diagnosis of Hirschsprung's disease can be made are evident within a day or two of birth. The authors' report is based on a review of 54 babies, under the age of 1 month on admission, with confirmed diagnosis of Hirschsprung's disease. A mortality of 33 per cent was recorded for the whole series; however, those with a long segment proximal to the pelvic colon had a mortality of 66 per cent, whereas when only a short segment was involved, the mortality was 24 per cent. The earliest clinical features were vomiting, abnormal bowel habits and abdominal distention. This is associated with failure to progress, poor feeding, listlessness, irritability and failure to gain weight. The roentgenographic examination of the abdomen with the baby erect will usually show the degree and distribution of gaseous distention of the intestine and a lateral erect roentgenogram in a neonate with a distended abdomen and absence of gas from the pelvis suggests Hirschsprung's disease. A barium enema examination provided a correct diagnosis in 24 of 27 patients. However, in the 2 cases in which the diagnosis was incorrect, the rectum had been washed out before the barium exam-

ination. No complications were associated with the roentgenographic examinations.

Although the diagnosis may also be confirmed by histologic examination of a biopsy specimen, a rectal biopsy is difficult and dangerous in the neonate and should be done only when the result of roentgenologic examination is equivocal. The authors suggest that only in those babies whose general condition is too poor for roentgenologic examination and who urgently require decompression of the obstructed bowel, should a colostomy be made without establishing the diagnosis.—*James R. Knapp, M.D.*

AMAR, ARJAN D. Rectal perforation by electronic thermistor in an unconscious patient. *Canad. M. A. J.*, July 22, 1967, *97*, 186–187. (From: Department of Urology, The Permanente Medical Group, Kaiser Foundation Hospital, Walnut Creek, and the Presbyterian Medical Center, San Francisco, Calif.)

Perforation of the rectum by an electronic thermistor is reported. The patient was unconscious as a result of severe head injury. The thermistor was introduced following operative procedure on the head. Three days later hematuria was noted and excretory urography revealed perivesical air. Abdominal exploration failed to reveal injury to abdominal viscera.

The patient died 5 days after the accident and at autopsy perforation of the rectal wall was found below the peritoneal reflexion. This perforation was sealed off and no peritonitis occurred.—*John H. Harris, M.D.*

KAINBERGER F. Ein Beitrag zum sogenannten "Gallenblasenregenerat" und seiner Entstehung. (Contribution to so-called "gallbladder regeneration" and its development.) *Radiologia Austriaca*, 1967, *17*, 5–12. (Address: Prim. Dr. F. Kainberger, Krankenhaus der Barmherzigen Brüder, Röntgeninstitut, Salzburg, Austria.)

Regeneration of the gallbladder subsequent to cholecystectomy has been doubted in recent years but has never been quite disproved. Those who do not believe in the regeneration claim that a sac formation in the gallbladder area following administration of contrast material represents either the duodenal bulb or dilatation of a cystic duct stump. A confusion with the duodenal bulb can easily be clarified by oral barium studies. Dilatation of a residual stump of the cystic duct, however, is improbable when the latter has been completely resected at its junction with the common duct.

The author now attempts to clarify the question of the apparently complete resection of the cystic duct. While reviewing 158 intravenous cholangiograms, made during the past 3 years, he has found an abnormal course 8 times (5 per cent). In these instances, the cystic duct crossed the common duct posteriorly and coursed alongside the latter on the left, instead of the right, prior to its termination. When such variation exists, it is extremely difficult for the surgeon to trace and dissect the cystic duct completely especially when it extends downward almost to the papilla Vateri. Without the knowledge of such variation, the surgeon can easily gain the erroneous impression that he makes a complete resection to the right of the common duct. Thus, an unrecognized stump may remain inadvertently and at a later date may give rise to a cystic dilatation with or without the presence of stone formation. A true gallbladder regeneration, however, has been distinctly disproved by animal experiments.

In the differential diagnosis, one has to consider duplication of the gallbladder with incomplete removal, diverticulum of the duodenum or common duct, and possible paravertebral abscess.—*Ernest Kraft, M.D.*

COSTA, F., and COSMACINI, G. I segni radiologici della cosiddetta "miomatosi" colecistica. (Radiologic signs of the so-called cholecystic "myomatosis.") *Radiol. med.*, April, 1967, *53*, 346–355. (Address: Dr. F. Costa, Via Meravigli, 7-Milano, Italy.)

It is well known that the so-called adenomyomatosis of the glallbladder is a process of hyperplasia which involves in different degrees of severity the mucosa of the gallbladder.

We recognize: (1) a pseudoglandular hyperplasia of the epitheluim or adenomyomatosis; (2) a myomatosis of the muscular layer; (3) a fibromatosis involving the perimuscular and subserous connective tissues; and (4) a neuromatosis, as defined by Riopelle, involving the intramural plexuses. Cholecystic myomatosis should be considered one of the "hyperplastic cholecystoses" of Jutras.

In the opinion of the authors, the following roentgen signs are pathognomonic and should permit a correct diagnosis of the pathologic process: (1) increased thickness of the walls of the gallbladder demonstrable in the fundus as a double contour of minimal radiopacity surrounding the area involved; (2) notching of the fundus; (3) functional findings typical of the morpholgic changes: (a) distal pouch due to narrowing of the fundus and resembling a phrygian cap, but which in the cholecystokinetic stage contracts much more than the remaining segments of the viscus, (b) hour-glass deformity, and (c) intraluminal septa of the gallbladder which also are brought out or better outlined in the cholecystokinetic stage.—*A. F. Govoni, M.D.*

GRIFFIN, T. F. R., and WILD, A. A. The case for preoperative cholangiography. *British J. Surg.*, July, 1967, *54*, 609–615. (Dr. T. F. R. Griffin, Consultant Surgeon, Scarborough Hospital Group, Scarborough, England.)

The authors discuss preoperative cholangiography by stating that the frequent presence of impalpable

duct stones in an undilated duct system, the lack of correlation of duct stones with jaundice in nearly 50 per cent of cases, and the finding of duct stones in a fifth of all cases which have never shown jaundice, are strong arguments for routine pre-exploratory cholangiography in all cases of gall stones.

The technique permits routine pre-exploratory cholangiography within an additional operating time of 5 minutes by utilizing a cassette tunnel, rapid preoperative positioning of the patient, low power mobile roentgenographic machines, fast films, and direct needle puncture and rapid injection of contrast material in the choledochus. Pre-exploratory cholangiography by their technique usually eliminated unnecessary choledochotomy, and over and above cases in which minor operative advantage has resulted from the examination, major advantage has resulted in 10 out of 253 cases by the disclosure of clinically undetected duct stones (4 per cent).

The authors describe their method for postexploratory cholangiography in detail.—*Richard L. Bauman, M.D.*

CATALANO, D. La colegrafia potenziata: colecistocolangiografia con Bilivistan per infusione glucosata lenta. (Potentiated cholegraphy; cholecystocholangiography with bilivistan by slow glucosed infusion.) *Radiol. med.*, April, 1967, *53*, 335–345. (Address: Via Costantinopoli, 84—Napoli, Italy.)

Drip infusion cholangiography was adopted by the author to obtain a more complete demonstration of the extrabiliary system.

After several tests it was found that 20 to 40 cc. of bilivistan added to 250 cc. of an isotonic glucosed solution, introduced intravenously by drip infusion in a period of 60 minutes, produced the best demonstration of the extrabiliary system with minimal or no general side-effects.—*A. F. Govoni, M.D.*

GEORGI, M. Kontrastanfärbung der Leber—Eine seltene Komplikation während der Lymphographie. (Contrast staining of the liver—a rare complication of lymphography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, June, 1967, *106*, 853–858. (Address: Universitäts-Strahlenklinik, Vossstrasse 3, 69 Heidelberg, Germany.)

Contrast staining of the liver occasionally occurs in metastasizing tumors of the testicle and the cervix and following radical Wertheim operations of the pelvic organs. It is always associated with a lymphatic block of the external iliac, the common iliac, or the prelumbar lymph nodes. There also co-exist lymphovenous anastomoses and occasionally a thrombosis of the inferior vena cava. The prognosis in such cases is considered very poor.

The author encountered liver staining in 1 of 84 patients in whom lymphography was performed for possible metastatic spread of testicular tumors. The patient was a 30 year old man with a history of seminoma with semicastration and postoperative radiation therapy. Seventeen months after the operation, there was metastatic spread to retroperitoneal and supraclavicular lymph nodes. Lymphography disclosed a block of the para-aortic lymph with collateral circulation, anterior displacement of the prelumbar lymph nodes and miliary staining of the liver 24 hours after the injection. Thirty-four months postoperatively, bilateral lymphography revealed lacunar defects of the retroperitoneal lymph nodes and miliary staining of the liver in addition to metastatic lymph nodes.

Lipiodol injected into the portal circulation of albino rats demonstated a reticular and miliary liver pattern which faded after 11 days. The contrast material most likely entered the liver via collaterals, lymphovenous anastomoses and the portal circulation. Possibility of a direct lymphatic spread without anastomoses could not be excluded as the intrahepatic lymph channels run close to the branches of the portal vein.—*Ernest Kraft, M.D.*

HERNANDEZ, CL., ECARLAT, B., and BISMUTH, V. L'exploration radiologique du pancréas: l'artérioportographie des affections pancréatiques. (Radiologic exploration of the pancreas: arterio-portography in pancreatic affections.) *J. de radiol., d'électrol. et de méd. nucléaire*, June–July, 1967, *48*, 327–338. (From: Service du Docteur Guy Morin, Hôpital Bichat, Paris, France.)

Arteriography is the method of choice in radiologic exploration of the pancreas (although there are two other approaches, suprahepatic caval filling, which is used principally to find hepatic metastases, and splenoportography to inspect the venous circulation). Selective arteriography carries little risk, and when separate injections are made in the celiac axis and the superior mesenteric artery the accuracy of diagnosis is high.

*Pancreatic cancer.* The alterations are arterial, venous and of the pancreatic mass, and may concern neighboring organs, the vena cava, splenic vein, portal trunk and gastric blood supply. Vessels can be altered in caliber, direction and termini, and shunts can develop. There may be no visible alterations in the parenchyma, or there may be hyperopacity in the tumor itself.

*Pancreatitis.* Ten per cent of the 110 cases of pancreatitis had no arterial anomaly; on the other hand 8 per cent of the whole series studied directed attention to hitherto unsuspected pancreatitis. The vessels are characteristically affected; 20 per cent of the cases showed diaphragmatic narrowing (a segment less than 1 cm. long being involved), 25 per cent had coned down sections, and 65 per cent had sleeve-like

(cylindrical) narrowing. Some cases revealed no pancreatic vessels, so that radiologically there was avascularity. Directions of normal vessels can be modified, and the volume of the pancreas changed. Noncalcifying nodules of pancreatitis may be revealed if the parenchyma around them opacifies from the contrast medium. In 4 per cent of the cases of calcifying pancreatitis late roentgenograms showed dense halos around the calcifications. In pancreatitis the pancreatic veins are not seen, although there may be modifications of the splenic-mesenteric-portal axis.

*Cysts.* The parenchyma rarely opacifies enough to contrast with a cyst which, however, may be identified by the deflection of larger pancreatic vessels. Arteriography also will be useful in differential diagnosis, since from it one may conclude that a mass is extrapancreatic.—*Frank A. Riebel, M.D.*

HERNANDEZ, CL., and HÉLÉNON, CH. (Paris, France.) Les tumeurs pancréatiques langerhansiennes: exploration vasculaire. (Langerhansian pancreatic tumors: vascular exploration.) *J. de radiol., d'électrol. et de méd. nucléaire*, June–July, 1967, *48*, 339–346.

In reporting 6 cases of tumors of the islands of Langerhans which were found by selective angiography the authors point out that the marked vascularity of the tumors provides for their clear visualization. They vary in size from very small to a mass several centimeters in diameter. The opacity may be rounded, well circumscribed or without clear limits. When surrounded by a clear zone one may suspect that it is benign, but the only certain proof of malignancy is the presence of metastatic opacifications in the liver. The differential diagnosis, radiologically, consists in distinguishing from a partly opacified duodenal bulb, a small bowel loop seen on end, or an angioma of the pancreas (one of the latter is reported here). It is unlikely that the lesions of the Zollinger-Ellison syndrome would be confused, as the nodules of this disease are usually too small (2 to 5 mm.) to be visualized angiographically.

The technique consists in selective injection of the celiac trunk and the superior mesenteric artery, separately. Combined filling can be done, but is considered to be an inferior approach, its only advantage being that a better portography is obtained.—*Frank A. Riebel, M.D.*

MALLET-GUY, P., and JACQUEMET, P. (Lyon, France.) Résultats comparatifs de la duodénographie hypotonique et des autres techniques d'exploration radiologique du pancréas. (Comparative results of hypotonic duodenography and other techniques of radiologic exploration of the pancreas.) *J. de radiol., d'électrol. et de méd. nucléaire*, June-July, 1967, *48*, 352–361.

Hypotonic duodenography consists of administrating an antispasmodic drug, followed by insertion and passage of the duodenal sound, through which barium is injected to fill out the duodenal loop. This results in more accurate delineation of the morphologic features which may be present. The diagnosis of tumors around the ampulla, chronic pancreatitis and carcinoma of the head of the pancreas may thus be made more easily than with routine barium studies.

Other methods discussed include pneumostratigraphy, intravenous cholangiography, splenoportography, as well as selective celiac and superior mesenteric arteriography. All of these methods present some difficulties with technique or interpretation and it is suggested that the simplest and possibly most effective way of studying lesions in the region of the head of the pancreas and ampulla of Vater is by the use of hypotonic duodenography.—*Charles M. Nice, Jr., M.D., Ph.D.*

WARTER, P., FONTAINE, R., KIENY, R., JAPY, CL., and BRIDIER, J. J. Valeur respective du transit baryté et de l'artériographie sélective dans les affections du pancréas. (Respective value of barium transit and selective arteriography in affections of the pancreas.) *J. de radiol., d'électrol. et de méd. nucléaire*, June–July, 1967, *48*, 361–368. (From: Centre Hospitalo-Universitaire de Strasbourg, Clinique Chirurgicale A, Strasbourg, France.)

The usual method of hypotonic duodenography involves administration of antispasmodic drug, passage of a duodenal sound, instillation of a local anesthetic, followed by the instillation of barium to distend the duodenal loop. In this paper a method is described without the use of the duodenal sound, utilizing the antispasmodic drug followed about 30 minutes later by a barium study of the duodenal loop.

The various signs of tumors, cysts, and pancreatitis which are found in arteriography and in the barium study are described. In a series of 40 cases the comparative results were slightly in favor of arteriography. The barium study was positive in 29 patients and the arteriographic examination was positive in 33 patients.—*Charles M. Nice, Jr., M.D., Ph.D.*

DU BOIS, R., and DEBEUGNY, P. (Lille, France.) Constatations radiologiques dans le pancréas annulaire. (Radiologic findings in annular pancreas.) *J. de radiol., d'électrol. et de méd. nucléaire*, June–July, 1967, *48*, 369–372.

Eight of the 9 cases of annular pancreas reported were examined between the 1st and 8th day of life, the studies being occasioned by symptoms of vomiting, epigastric distention with a flat lower abdomen,

and often a state of respiratory distress. These were cases of complete or nearly complete duodenal obstruction; the 9th case was in an infant of 10 months.

Three plain roentgenograms of the infant—supine, upright and head down—may be adequate for the diagnosis, as they give a classic picture of double air bubbles and double fluid levels, often with enormous dilatation of the duodenal bulb. A barium meal is additionally useful, but barium enema examinations have not helped, in the authors' experience.

Roentgenologic analysis is fully as important in the postoperative state. Surgery usually does not include sectioning the obstacle, for this provokes hemorrhage, pancreatitis or pancreatic fistulas. More often a simple gastro-jejunostomy is done, which however is usually inadequate, since the bulb remains dilated and the infant dies by the second month of febrile icterus, choleostasis or hepatic abscess. Therefore, it is necessary to decompress the enormous bulb by a short shunt. Either duodeno-jejunostomy or duodeno-duodenostomy can be done, of which the authors favor the latter.—*Frank A. Riebel, M.D.*

LEDOUX-LEBARD, G., HEITZ, F., BÉHAR, A., ROSIER, J., LEGER, L., SIGUIER, F., and PRÉMONT, M. La pancréatographie par voie veineuse à l'érythrosine B en clinique humaine: études des 30 premières observations. (Intravenous pancreatography with erythrosine B in human patients; preliminary report of 30 observations.) *J. de radiol., d'électrol. et de méd. nucléaire*, June–July, 1967, *48*, 373–378. (From: Chaire de Radiologie médicale à la Faculté, Paris, France.)

Erythrosine B is a coloring substance containing a tetraiodide. After performing retropneumoperitoneum and a small pneumoperitoneum the patient is given 20 ml. of 20 per cent erythrosine B intravenously. The pancreas is then studied by means of tomography. If the patient is prepared for a period of about 3 days (to cleanse the intestinal tract?) the use of pneumoperitoneum and retroperitoneum may be dispensed with. The tomograms were taken 20, 40, and 70 minutes after injection, since the pancreas usually begins to opacify at 20 minutes, is usually more opaque at 40 minutes, and then the shadow becomes attenuated at 70 minutes. Contrast medium is seen entering the duodenal loop and 24 hours later the gallbladder is clearly opacified.

In chronic pancreatitis the maximum opacification occurs at 20 minutes. No experience has been secured in patients with carcinoma of the pancreas as yet. One patient with hydatid cyst and another with a pseudocyst have shown filling defects in the pancreas.

Some patients may exhibit photosensitivity after the injection of erythrosine B. There may be a slight rose color of the skin for about 24 hours. Some workers have shown that antihistamine will protect against photosensitization and antimalarial drugs and for this reason antihistaminics are given for 3 days before the examination.

Some minor inflammatory changes at the point of injection and allergic reactions have also been shown, the latter usually responding to the injection of corticoids.—*Charles M. Nice, Jr., M.D., Ph.D.*

## GYNECOLOGY AND OBSTETRICS

GORECKI, P. (Kinshasa.) Hysterosalpingographie en Afrique Centrale. (Hysterosalpingography in Central Africa.) *J. belge de radiol.*, 1967, *50*, 3–10.

Based upon 3,000 hysterosalpingographies done on African women, the author finds definite differences as to results when compared to Caucasian women. The same is true of gynecologic diseases and causes of sterility.

Thus, infantilism is relatively higher in African women, so are endometriosis, uterine stenosis with round cornua, uterine septa (noted even after parturition) and uterine adhesions. A very high incidence of terminal arachnoidal sacs of otherwise normal tubes was noted. These sacs would hinder totally ova to enter the tubes. Tuberculosis and various other chronic inflammatory changes of the tubes were also frequent.

Rare were cancer and retraction of the uterus to the right due to recurrent appendicitis.—*Jirair N. Sarian, M.D.*

ROSENTHALL, LEONARD. Radionuclide visualization of the placenta with the gamma-ray scintillation camera. *Canad. M.A.J.*, July 29, 1967, *97*, 212–217. (From: Division of Nuclear Medicine, Montreal General Hospital, Montreal, Quebec, Canada.)

To visualize and localize the placental blood pool relative to the symphysis pubis, a technique employing technetium 99m pertechnetate ($Tc^{99m}O_4$) or strontium 87m ($Sr^{87m}$) and the gamma-ray scintillation camera were used. Approximately 8 minutes is required for frontal and lateral views. The absorbed fetal radiation dose is similar for pertechnetate as technetium 99m-labelled albumin, and the time-consuming preparation needed with the latter is avoided. Slightly more exposure is received with strontium 87m, but it is still less than radiography by a factor of at least 100.

Fifty-two women were examined in the second and third trimesters of gestation. When the placentas were localized by scan into categories of remote, low-lying, marginal, partial or central placenta previa, an accuracy of 95 per cent was obtained. The results are 98 per cent accurate when the categories are limited to remote and low-lying placenta previa. The placental blood pool is clearly delineated and gross changes in perfusion may be detected.—*Eugene J. McDonald, M.D.*

Shapiro, B. J., and Shaul, D. Radioisotope localization of the placenta. *Canad. M.A.J.*, July 29, 1967, *97*, 218–221. (Address: Dr. B. J. Shapiro, Department of Radiology, The New Mount Sinai Hospital, Toronto, Ontario, Canada.)

In the past, various methods of placentography have been tried, including soft tissue placentography, amniography, femoral arteriography and most recently thermography. The first can only be used after the 34th week of gestation, and the second carries a great risk of intrauterine death; the third is a major procedure for use as a diagnostic test. Radioisotope placentography, however, has proved to be an accurate and simple method.

Suitable radionuclide such as $I^{131}$-labelled human serum albumin is injected intravenously and 10 minutes allowed to elapse. Counts are then taken over 12 uterine ports and also over the "heart pool." The counts over the uterus were expressed as percentages of the counts over the heart pool.

A total of 75 patients were studied: 60 because of antepartum hemorrhage, 12 before amniocentesis and 3 for miscellaneous reasons. The over-all accuracy of localization was 96 per cent. There were one false positive, one false negative and one failure of localization. The accuracy of this method compares favorably with a 78 per cent accuracy of radiographic localization. The radiation dosage to the fetus is much less than with standard radiologic techniques.—*Eugene J. McDonald, M.D.*

## RADIATION THERAPY

Caldwell, William L., Bagshaw, Malcolm A., and Kaplan, Henry S. Efficacy of linear accelerator x-ray therapy in cancer of the bladder. *J. Urol.*, Feb., 1967, *97*, 294–303. (From: Division of Radiotherapy, Department of Radiology, Stanford University Medical Center, Palo Alto, Calif.)

Two-hundred and twenty-nine patients with proven pathologic diagnosis of carcinoma of the urinary bladder were treated over an $8\frac{1}{2}$ year period with the 5 mev. or the 6 mev. linear accelerator. In this article, these patients are evaluated according to the clinical classification of Marshall. Those patients with Stage A and $B_1$ disease ordinarily received a maximum tumor dose of 7,000 rads through multiple small fixed fields or through small fields using rotational therapy. Stage $B_2$ and C tumors were treated by large 15×15 cm. anterior, posterior and lateral fields. Patients in the latter groups received 6,000 rads and about 50 per cent of them received an additional 1,000 rads to the bladder through small fields at the end of the series. Stage $D_1$ tumors were generally treated with 4 field whole pelvis technique to 6,000 rads. The patients were treated with equally fractionated doses 4 to 5 times a week and the irradiation series was completed in approximately 6 to 8 weeks.

The authors include isodose curves to show the pattern of irradiation dosage with the various techniques used.

The overall gross survival indicates that 34 per cent or 45 of 132 patients survived 3 years, and 19 of 73 patients survived 5 years.

In another interesting figure, the authors show the absolute survival curves, uncorrected, according to the stage of the disease and in contrast with the actuarial survival curves by stage when compared to the life expectancy of the general male population of 65 years.

The latter set of survival curves indicates that 55 per cent of patients with Stage A disease survive 3 years. Forty per cent of those with Stage $B_1$ survive 3 years and approximately 35 per cent of those with Stage $B_2$ disease survive 3 years.

Most patients tolerated their treatment satisfactorily, with slight to moderate increase in urinary symptoms. Six patients did not complete the planned course of treatment because of general deterioration or evidence of distant metastases during the treatment period. Cystitis occurred more frequently in those patients with Stage A disease than in those with Stage B or C. The overall incidence of significant cystitis was approximately 14 per cent. Diarrhea persisting for more than 3 months occurred in 5 patients and a colostomy was necessary in 1 of these. In 2 patients, small bowel perforations occurred; in both instances the patients had persistent tumor in the bladder. An evaluation of 95 patients from autopsy study showed that 52 of these had persistent tumor limited to the pelvis. Transurethral resection, segmental resection or cystectomy were required in 15 patients in this series. Five of the 9 patients who required cystectomy are now apparently free of disease, whereas 5 of 6 requiring transurethral or segmental resection are living without demonstrable cancer.

The suggested treatment policy as recommended by the authors is that those patients with Stage A disease who have one or a few low-grade bladder tumors can be managed by wide transurethral resections. With multiple tumors or high grade tumors, megavoltage radiotherapy or cystectomy is currently preferred. For those patients in Stage B and C, either supervoltage radiation therapy with a complete series of treatments or a total cystectomy is recommended. The authors point out that transurethral resection or segmental resection of disease in this stage is not an entirely rational approach since the network of lymphatics and vessels in the bladder wall favors early spread of invasive tumors. Furthermore, radiotherapy after such procedures is associated with a higher incidence of complications. The plea in this group of patients is for early recognition of invasive bladder carcinoma and the need for radical treatment initially in order to achieve the maximum cure rates.

Planned preoperative radiotherapy with subsequent cystectomy may increase survival rates over those of cystectomy alone. For those patients with Stage $D_1$ disease cystectomy, like irradiation, has yielded a very small survival percentage; and extended radical surgery has not increased this percentage. Palliation may be achieved by a 4-field whole pelvis irradiation technique delivering 6,000 or more rads to the bladder area.

This article includes 4 figures and 8 tables to illustrate the various data of importance.—*George W. Chamberlin, M.D.*

WEAVER, ROBERT G., CHAMBERLIN, E. CARL, and BROWN, BLAINE R. Localization and efficiency of interstitial chromic phosphate in the bladder. *J. Urol.*, April, 1967, *97*, 646–653. (From: Division of Urology and Department of Radiology, University of Utah College of Medicine, Salt Lake City, Utah.)

This work consists of animal experiments to determine the behavior of colloidal chromic phosphate when injected interstitially into the bladder wall of a series of dogs: its localization, spread, extravesical involvement, and quantitative irradiation of the area injected. In addition, a brief account is given of the clinical application of such treatment in 9 patients who have proven microscopic diagnosis of papillary carcinoma of the bladder, Grade II or III, with infiltration of the bladder wall.

It is demonstrated that colloidal chromic phosphate can be injected into the bladder wall in sufficient quantities to produce carcinocidal irradiation and that such injection and irradiation of the bladder wall are well tolerated. As much as 75 to 95 per cent of the chromic phosphate is retained at the site of the injection for a period of 40 days. When used in the proper dilution, the isotope can be evenly distributed throughout the area of injection and doses of from 6,000 to 10,000 rads can be given to a selected area.

Three tables and 5 figures illustrate the technical details and observations included in this study.—*George W. Chamberlin, M.D.*

## RADIOISOTOPES

DEREYMAEKER, A., DE ROO, M., and DARDENNE, G. Study of the real tissue concentration of radioactive tracers, used for the external detection of cerebral tumors. *J. belge de radiol.*, 1967, *50*, 69–77. (Address: Dr. Dereymaeker, Waversebaan, 290, Heverlee, Belgium.)

The true tumor concentration of two radioactive tracers was determined in 43 patients with verified cerebral neoplasms to find if there was a valid correlation between the information obtained with external measurement techniques and the type of tumor. $RI^{131}SA$ and neohydrin $Hg^{203}$ were used as the tracers. Brain scanning or gamma encephalography was done. No postmortem specimens were included. Blood samples, tumor tissue, and normal cerebral tissue adjacent to the lesion were taken at the time of the operation. Measurements were made, and the true amount of radioactivity in the tissues was obtained by subtracting the activity due to blood contamination from the total tissue activity. The accumulation ratios were established for normal brain and tumor tissues after correcting for blood activity.

Corrected accumulation ratio measurements gave similar results for some histologic types of tumors, regardless of the radioactive tracer used. The astrocytomata, however, showed a marked lower accumulation ratio for $RI^{131}SA$ and the glioblastomata and metastases showed a greater concentration ability for neohydrin $Hg^{203}$.

The authors found that there was no apparent specific tissue concentration in several types of cerebral tumors. The relation between external radiation measurements and the type of cerebral tumor was thought to be due to vascular phenomena, such as volume and density of blood vessels, and to blood flow in the tumor rather than to the specific concentration ability of the tumor for the radioactive isotope.—*Charles W. Cooley, M.D.*

ROSENTHALL, LEONARD. Thoracic radionuclide angiography with the gamma-ray scintillation camera. *J. Canad. A. Radiologists*, March, 1967, *18*, 270–273. (From: Department of Radiology, Division of Nuclear Medicine, Montreal General Hospital, Montreal, Quebec, Canada.)

The author points out that the gamma camera lends itself to continuous monitoring of a significant area over a period of time by multiple serial pictures. This attribute has been used to visualize the large vessels of the thorax and heart chambers with serial 5 second exposure scintiphotos. The isotope used was 10 millicuries of technetium 99m pertechnetate injected in the antecubital vein. Total body radiation exposure was about 130 millirads per 10 millicurie dose.

Excellent scintiphoto reproductions show vascular obstructions, aneurysms, and pericardial effusions.—*George A. Miller, M.D.*

EVANS, TITUS C., KRETZSCHMAR, ROBERT M., HODGES, ROBERT E., and SONG, CHANG W. Radioiodine uptake studies of the human fetal thyroid. *J. Nuclear Med.*, March, 1967, *8*, 157–165. (From: Radiation Research Laboratory, Departments of Obstetrics and

Gynecology, and Internal Medicine, University of Iowa, Iowa City, Iowa.)

$I^{131}$ uptakes, radioautographs and chromatograms of thyroid hydrolysates were obtained on 36 fetal thyroids. There was no detectable uptake under 12 weeks' gestation. The glands were quite undifferentiated with only a few acini. Detectable uptakes were found at 12 to 13 weeks. The radioautographs showed activity in the periphery of the glands. The ability to bind iodine organically was just beginning, as shown by the difference in the radioautographs in the glands treated with acid preservatives and those preserved in neutral formalin.

The $I^{131}$ uptake was more uniform in the 3 to 6 month group. The uptakes from the 3 to 4 month group averaged 0.025 per cent per gland, or approximately 1 per cent per gram of thyroid. The 4 to 5 month group showed an average uptake of 0.2 per cent per gland and about 2 per cent per gram of thyroid. The highest concentration was 5 per cent per gram for the 5 to 6 month group. The uptake per gland was about 1 per cent. The percentage per gland increased to 2 per cent near term, but the concentration was less than that at the sixth month. The maximum concentration was 5 times that of the adult thyroid, but less than that found only a few days after birth.

The chromatography studies showed that the thyroid did not bind $I^{131}$ to thyroglobulin at the time of the earliest uptake. $I^{131}$ was found in the organic fractions by the 20th week, and chiefly as moniodotyrosine. The relative amounts of the diiodotyrosine, triiodothyronine and tetraiodothyronine fractions increased with fetal age.—*Charles W. Cooley, M.D.*

Braverman, Lewis E., Foster, Angela E., and Arky, Ronald A. Oragrafin and the triiodothyronine uptake test of thyroid function. *J. Nuclear Med.*, March, 1967, *8*, 209–212. (From: The Endocrine Division, St. Elizabeth's Hospital, and the Department of Medicine, Tufts Medical School; the Thorndike Memorial Laboratory and the Second and Fourth [Harvard] Medical Services, Boston City Hospital; and the Department of Medicine, Harvard Medical School, Boston, Massachusetts.)

Resin and the resin sponge uptake of $I^{131}$-labeled $T_3$, thyrobinding index (TBI), and charcoal $T_3$ studies were done in 19 clinically euthyroid subjects before and after the ingestion of orgrafin (sodium ipodate, an oral cholecystographic agent).

The resin uptake of $T_3$ was increased following the ingestion of orgrafin, but the increase was small and was greater than 2 per cent in only 3 of the 19 subjects, and in only 1 subject was the increase above the normal range. The resin sponge uptake of $T_3$ was increased in 10 of the 15 subjects studied, but in only 4 was the increase greater than 2 per cent, and in only 2 was the value increased into the thyrotoxic range. There was no change in the TBI in 8 subjects studied, or in the charcoal $T_3$ ratio in the 10 subjects studied.

The ingestion of oragrafin occasionally produced a rise in the resin sponge and resin $T_3$ uptake into the thyrotoxic range. The TBI and the charcoal $T_3$ ratio were not altered by this contrast medium.—*Charles W. Cooley, M.D.*

Winter, Chester C. Applications of the scintillation camera in urology. *J. Urol.*, April, 1967, *97*, 766–768. (From: Division of Urology, Department of Surgery, Ohio State University Hospital, Columbus, Ohio.)

The scintillation camera consists of a large crystal backed by an array of many photomultiplier tubes with a network of fixed, signal-mixing capacitors. These, in turn, are connected through a computing circuit and pulse-height selector which reproduces momentary light flashes on an oscilloscope screen. The flashes are so adjusted as to indicate the relative intensity of the radiation signal emanating from the kidney. The light emissions are recorded on Polaroid film. This results in a dynamic or continuous record of renal blood flow, function and excretion.

Various radioisotope compounds may be used. Orthoiodohippurate labeled with iodine 131 is generally the most satisfactory test material. The test may be carried out with the patient in the erect or horizontal position, using various collimators. With the presently available sized crystal, both kidneys can be evaluated at the same time in children or small adults, but for the larger adults it is difficult to obtain a record of both kidneys simultaneously. Future developments of the scintillation camera might include a larger sized crystal.

The author reproduces the scintiphotograph of a normal and abnormal kidney in one patient along with the intravenous pyelogram and the radioisotope renogram. He also reproduces a scintiphotograph of a solitary kidney and the adjacent ideal segment into which the ureter empties.

This procedure appears to have limited usefulness in urologic diagnosis, but it may enhance some of our presently available methods. It does not offer a method for specific diagnosis and it does not replace conventional renal function studies.—*George W. Chamberlin, M.D.*

Burke, Gerald, and Halko, Arlene. Scintillation camera renography in the study of prolonged renal transit time. *Radiology*, April, 1967, *88*, 704–712. (From: Radioisotope Laboratory, Division of Metabolism and Endocrinology, Department of Medicine, Michael Reese Hospital and Medical Center, Chicago, Ill.)

The authors have previously noted that three distinct intrarenal mechanisms may prolong renal hippuran transit time while yielding entirely similar abnormalities of renogram contour. The three mechanisms include decreased urine flow rate, increased intrarenal urine volume, and alteration in kinetics of renal cellular hippuran transport. The purpose of the present report is to demonstrate that scintillation camera renography, by concomitant study of renal topography and intercompartmental transit of $I^{131}$ hippuran permits delineation of the basis for prolongation of the transit time in disparate diseases of the kidney.

The technique presented is designed to visualize each kidney separately. Patients are usually examined seated and the kidney is aligned with the center of the crystal after a localizing dose of $Hg^{203}$ neohydrin. Following an intravenous dose of 200 microcuries of $I^{131}$ hippuran, a combination of one-minute counts and two-minute scintiphotographs is obtained which permits the plotting of a renogram curve and assures accumulation of sufficient information on film to permit direct monitoring of the passage of labeled hippuran through the renal parenchyma and pelvis. Information is accumulated in this manner until the bulk of the hippuran leaves the renal pelvis—about 20 minutes in a well hydrated patient—and the entire process is repeated on the contralateral side.

In prolongation of renal hippuran transit time secondary to decreased urine flow rate the scintiphotographs demonstrate slow parenchymal clearing and central retention of radioactivity. These features are clearly demonstrated in a case report of hypertensive renal artery stenosis and even allowed a suspicion of similar disease in the opposite kidney, later confirmed by angiography, but not indicated by the conventional renogram, rapid-sequence urogram, and differential renal function studies.

The prolonged transit time secondary to increased intrarenal urine volume, which includes inadvertent ureteral ligation and other forms of obstructive uropathy, presents characteristic findings on the scintiphotographs. These findings are prolonged pelvic retention of radioactivity and, later in the series, demonstration of a dilated ureter.

Renal parenchymal disease results in a prolonged transit time secondary to alteration in kinetics of renal cellular transport. The authors have studied a group of 50 children with acute glomerulonephritis and in 46 of the 50 the transit time was prolonged in the conventional renogram. Scintillation camera renography was performed in 9 patients and the scintiphotographs revealed a characteristic prolongation of radioactivity in the parenchyma without central or pelvic retention.

Although many factors affect renal hippuran transit time, the hippuran normally leaving the kidney is directly dependent on a functioning renal parenchyma, a flow ratio of urine between the parenchyma and pelvis, and discharge of urine from the pelvis. Alteration of any one of these factors by diverse renal disease will produce the similar abnormality on conventional renograms of a prolonged renal transit time. Scintillation camera renography, by the use of serial scintiphotographs, will clarify these several causes of prolonged renal transit time and thus represents a significant advance over conventional renographic studies in the evaluation of renal function.—*E. R. Best, M.D.*

CHISHOLM, G. D., AYE, M. M., and EVANS, K. Evaluation of the $Hg^{197}$ chlormerodrin kidney scan. *Brit. J. Surg.*, Feb., 1967, *39*, 38–49. (From: Urological Unit, Department of Surgery, Hammersmith Hospital, and Postgraduate Medical School, Du Cane Road, London, W.12, England.)

The authors state that, despite the availability of mercury 197 chlormerodrin for kidney scanning, this procedure has not gained wide acceptance. Because of the simplicity and safety of the procedure, together with no patient preparation, they feel that additional use is warranted. They report their clinical observations based on kidney scannings on 120 patients following the administration of 100 microcuries of $Hg^{197}$ chlormerodrin.

The authors found good correlation in the determination of kidney size by the scan but encountered some difficulty in determining the position of the right upper pole because of uptake of the radioisotope by the liver, particularly in patients with depressed renal function. The kidney scan readily outlined "cold" areas, indicating the presence of a space occupying lesion but gave no indication of the underlying pathology. The smallest filling defect detected was 2.5 cm. in diameter.

The relative uptake of the radioisotope by the two kidneys gives an estimate of renal function. This proved to be of particular interest in the evaluation of unilateral renal disease.

The authors found the scan to be of little value in the presence of renal failure due to a limited uptake of the radioisotope by the kidneys and concluded that the scan did not yield more information than could be obtained from an excretory pyelogram or other roentgen study.—*Merle K. Loken, Ph.D., M.D.*

CHISHOLM, G. D., EVANS, K., and KULATILAKE, A. E. The quantitation of renal blood flow using $I^{125}$ hippuran: an experimental study in the perfusion of the isolated canine kidney. *Brit. J. Urol.*, Feb., 1967, *39*, 50–57. (From: Urological Unit, Department of Surgery, Hammersmith Hospital, and Postgraduate Medical School, Du Cane Road, London, W.12, England.)

For this study the authors analyzed renograms following the injection of $I^{125}$ hippuran into an isolated canine kidney in a perfusion circuit. This technique reduced to a minimum the number of variables which oftentimes affect the renogram and therefore enables accurate measurements to be made of different values required for calculations of blood flow.

The results of their study show that if it is possible to quantitate the amount of radioactive hippuran in the human intact kidney, it should be possible to quantitate renal blood flow. Quantitation is made difficult because dynamic equilibrium is never reached in the kidney with a single injection of hippuran and, furthermore, hippuran is autonomous in the blood and does not trace any normal blood constituent through the system. An additional difficulty in measurements of renal uptake in the intact individual relates to the presence of hippuran in the perivascular and extracellular fluid spaces.

The studies by the authors were made in two parts. They analyzed the renogram curves from isolated kidneys applying theoretic equations and showed that quantitative values could be obtained for effective renal blood flow. In a second part of experiments, they found that the rate of disappearance of hippuran from the blood was related to renal blood flow, but could not be used to quantitate this factor.—*Merle K. Loken, Ph.D., M.D.*

Simon, Norman, and Feitelberg, Sergei. Scanning bremsstrahlung of yttrium-90 microspheres injected intra-arterially. *Radiology*, April, 1967, *88*, 719–724. (From: Department of Radiotherapy and the Andre Meyer Department of Physics, The Mount Sinai Hospital, New York, N. Y.)

The authors report that scanning the bremsstrahlung due to the therapeutic doses of pure $\beta$ emitters within the body has useful clinical application. Plastic microspheres of 15 microns in diameter containing $Y^{90}$ and suspended in dextran are injected intra-arterially into brain, kidney, liver and lung.

Three cases of carcinoid tumor of the liver were treated with these microspheres of $Y^{90}$. The common hepatic artery was catheterized percutaneously through the brachial or femoral artery. The catheter position was checked by opacifying the vessel before injecting the microspheres and afterwards by repeat angiography. Scanning was done within a few hours after the procedure to observe the distribution of $Y^{90}$ within the liver and also any leakage of the $Y^{90}$ into the lungs or extremities. In one case 25 mc was administered to a 1,500 gram liver via the right hepatic artery. The scan was accomplished the following day with a spectrometer setting which accepted bremsstrahlung with energies from 35 to 200 kev.

In the brain, the $Y^{90}$ microspheres may be injected into the cerebrum via the internal carotid artery to irradiate the cerebrum homogeneously and selectively. Patients with tumors of grave prognosis, multiple metastases or glioblastoma multiforme, were selected for this intra-arterial treatment. Usually, external radiation therapy applied to a unilateral cerebral lesion does not spare the "normal" side. However, the $Y^{90}$ therapy irradiates only the side of the cerebrum in which the injection is made. The local injection of the $Y^{90}$ was demonstrated by bremsstrahlung scanning the brain. The treatment was well tolerated in the 3 reported patients.

In the lung, the pulmonary artery distribution may be scanned after injecting the $Y^{90}$ microspheres into a peripheral vein. These scans have helped to define the indications for $Y^{90}$ microspheres in the treatment of pulmonary tumors. It is unreasonable to try to treat a large tumor of the lung via the pulmonary artery and intravenous injection but the lymphangitic spread of miliary metastases is likely to be within the range of beta particles stuck in the pulmonary arteries.

The kidney can be injected intra-arterially by percutaneous catheterization of the renal artery via the femoral artery. Arteriovenous shunts through the kidney or through a hypernephroma can be demonstrated by the scan obtained after the injection of the $Y^{90}$ microspheres.

The practicality of bremsstrahlung scanning of pure $\beta$ emitting $P^{32}$ makes this isotope preferable to $Au^{198}$ in the treatment of effusions. The distribution of $P^{32}$ can be satisfactorily determined by the scan and the absence of the gamma rays from $Au^{198}$ reduces the hazard and difficulty of administration of the isotope.

In the treatment of bone metastases of carcinoma of the prostate, the bremsstrahlung scan may show differential uptake of $P^{32}$ and thus provide important clinical information.

Until clinical observations yield more information, the authors are limiting the dose to the brain to 3,000 rads, to the lung to 2,000 rads, and to the liver to 2,500 rads.

The interested reader should consult the original article for the authors' detailed case reports.—*L. H. Deitermen, Jr., Ph.D.*

Simon, Norman, Feitelberg, Sergei, and Glickman, Stanley I. Scanning radioactive phosphorus in bone metastases from cancer of the prostate. *J. Urol.*, March, 1967, *97*, 323–325. (From: The Radiotherapy, Physics and Urology Departments, Mount Sinai Hospital, New York, N. Y.)

The recent development of sensitive scanning equipment now makes it possible to detect the uptake of $P^{32}$ in bone by virtue of the bremsstrahlung.

Radioactive phosphorus has been shown to be of some value in the treatment of bone metastases from carcinoma of the prostate in those patients who have become resistent to treatment after orchiectomy and estrogen therapy. In this group of individuals, androgens are used prior to and with the administration of 10 to 15 millicuries of $P^{32}$.

The authors state that this type of scanning has become a routine procedure at their institution. They illustrate the procedure and technique by a single case report, with one roentgenogram showing the metastatic bone lesions and one photoscan showing the increased uptake of the isotope in the metastatic bone lesions.

Using this method, it is now possible to detect and map radioactive phosphorus in bone *in vivo* and by repeat examinations to appropriately guide the therapeutic administration of the radioactive material. Since the effect of an internally deposited isotope depends upon the size of the dose in the target tissue, this is an important adjunct to our therapeutic regime.—*George W. Chamberlin, M.D.*

FABER, D. D., WAHMAN, G. E., BAILEY, T. A., FLOCKS, R. H., CULP, D. A., and MORRISON, R. T. An evaluation of the strontium 85 scan for the detection and localization of bone metastases from prostatic carcinoma: a preliminary report of 93 cases. *J. Urol.*, March, 1967, *97*, 526–532. (From: Departments of Urology, Nuclear Medicine and Radiation Research, University Hospitals, Iowa City, Iowa.)

Early detection of skeletal involvement by carcinoma of the prostate is important in estimation of the prognosis and selection of therapy. The present commonly used methods for evaluation of this group of patients fail to detect skeletal metastatic spread in its incipiency.

The authors studied 93 consecutive patients with various clinical stages of prostatic carcinoma and report their results in this article. Histologic proof of the disease was obtained prior to $Sr^{85}$ scans. An intravenous dose of 75 to 100 microcuries of $Sr^{85}$ was given and the scans were obtained 4 to 14 days later. A roentgenogram of the lumbar spine and pelvis was routinely made and the scans covered the same areas. Serum acid phosphatase was determined in all patients and bone marrow aspirates were obtained from the iliac crest in 38 patients.

Fourteen patients of this group had roentgenographic evidence of bone metastasis of the osteoblastic type. In each instance the radioactive isotope scan showed increased concentration of $Sr^{85}$ in the corresponding areas. The extent of the tumor involvement as indicated by the scan was generally the same as that shown on the roentgenogram.

Thirty-two patients or 34 per cent of the entire group had an abnormal scan. Of these, 14 had positive roentgenograms; in 18 the roentgenograms did not show tumor. Fourteen patients had an abnormally elevated serum acid phosphatase, but these are not necessarily the same patients as those who had positive roentgenograms. One patient out of 38 who had bone marrow aspirations, showed microscopic evidence of tumor cells.

The period of observation of the 18 patients having abnormal scans with normal roentgenograms has not been sufficiently long to demonstrate whether metastatic lesions will appear at a later date. Sixty-one patients had a normal scan, and none of these had roentgenographic evidence of bone metastasis, but 33 per cent of this group had an elevated serum acid phosphatase as compared to 44 per cent of those in which the scan was abnormal.

The results of this study suggest that the $Sr^{85}$ bone scan is more sensitive than the skeletal roentgenogram in the detection of metastatic bone disease. However, the serum acid phosphatase is less specific since it is elevated in Paget's disease, eosinophilic granuloma, healing fractures, and other metastatic bone lesions.

The authors believe that patients with abnormal scans and normal roentgenograms should be examined periodically to determine whether the apparent bone disease becomes evident roentgenographically. Only after these patients have been followed and evaluated, can one be certain that increased radioactivity on a scan is definitive evidence of metastatic disease. At the present time, the procedure is considered to be a valuable diagnostic parameter in the management of patients with carcinoma of the prostate, but further investigation is necessary before its full significance can be completely evaluated.

Eight composite figures illustrate this article.—*George W. Chamberlin, M.D.*

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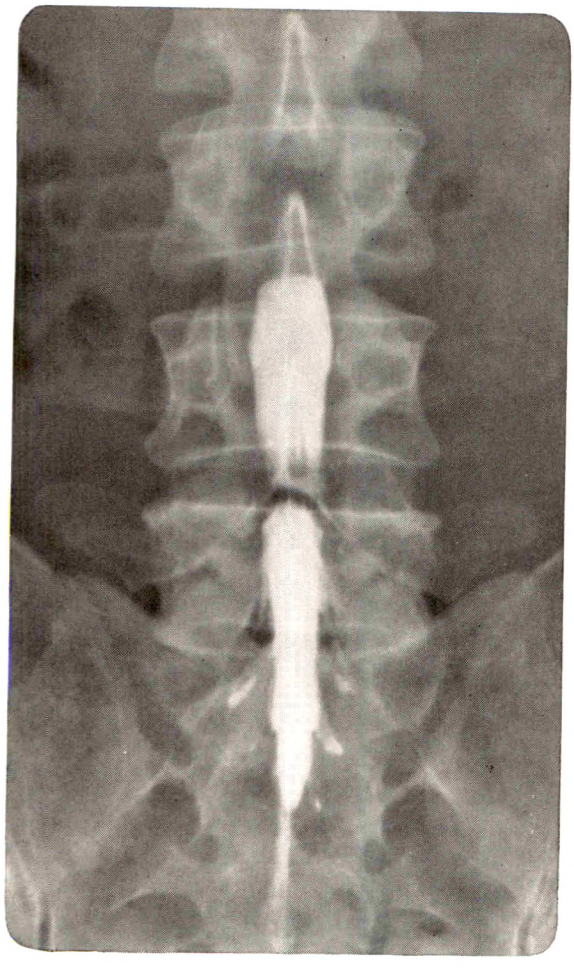
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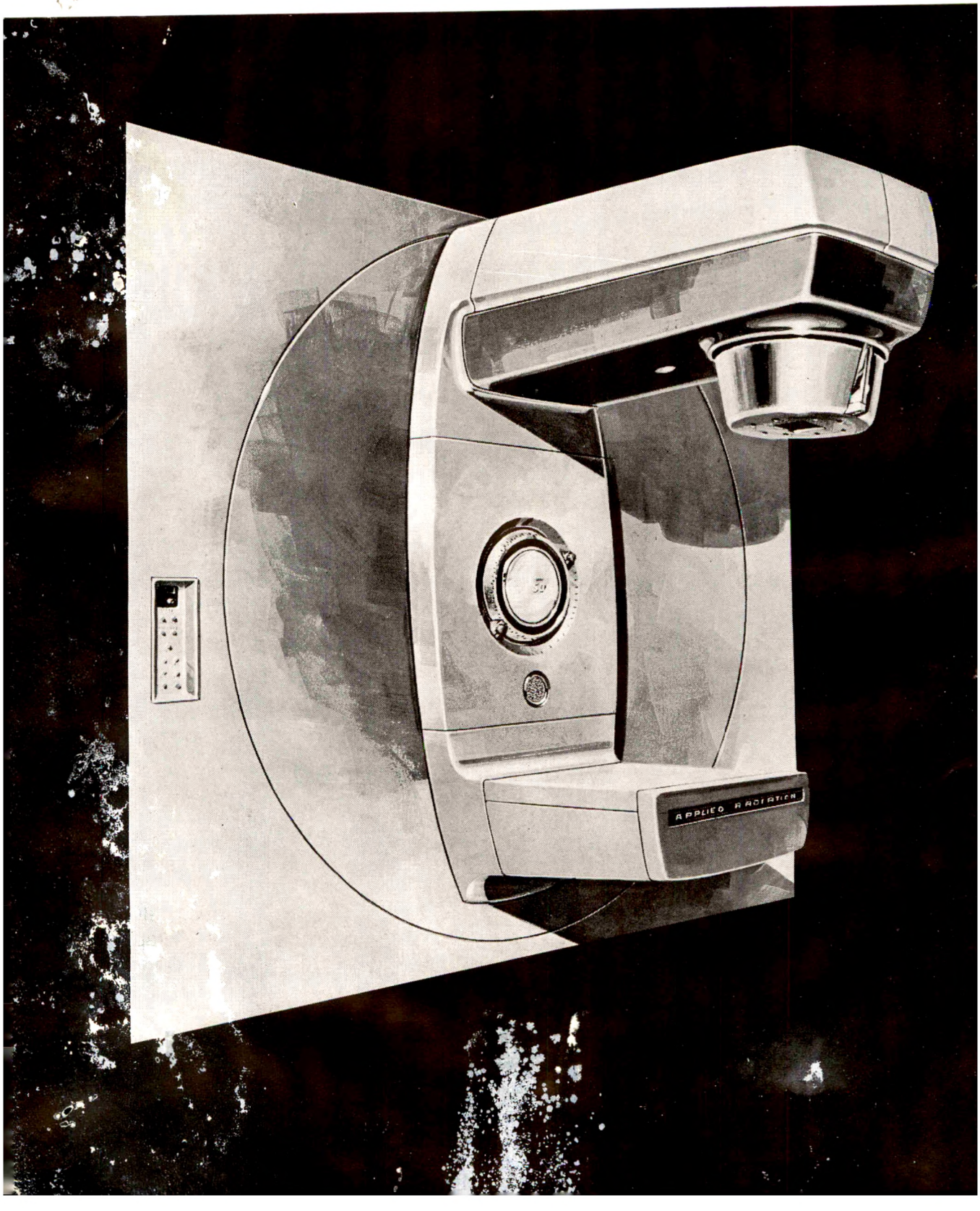


The patient, a 46-year-old male, had persistent low back pain radiating down both lower extremities posteriorly, and numbness and pain over both heels. X-ray examinations of back and hips, made 18 months before, following an automobile accident, were reported as normal.

Myelographic examination using PANTOPAQUE [Iophendylate Injection] showed a large defect at the fourth lumbar interspace, which was interpreted to be due to discal herniation in a narrow canal. At surgery, a large herniated intervertebral disc in a very narrow lumbar vertebral canal was found and removed.

"PANTOPAQUE" *is the registered trademark under which all leading x-ray dealers supply the compound ethyl iodophenylundecylate, which is synthesized in the laboratories of Eastman Kodak Company and prepared as the myelographic contrast medium Iophendylate Injection, U.S.P., by Lafayette Pharmacal Inc. The trademark serves to indicate to the radiologist continuity of experience in the manufacture of this medium.*

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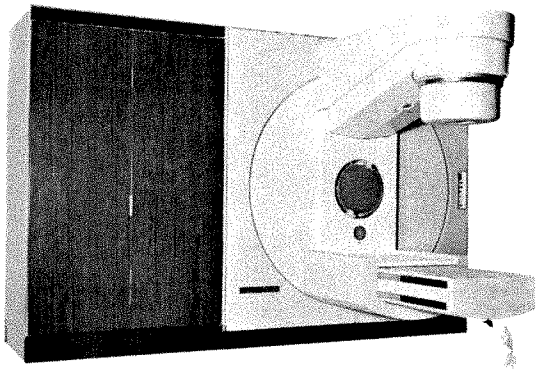
**400,000 r/min**—the experimental maximum dose rate. High dose rates facilitate research in hyperbaric oxygen therapy, whole body irradiation, and massive dose studies. Dose rates of 400 r/min X-rays and 500 r/min electrons are available for routine therapy.

**Short lived isotopes.** The neutron beam will produce isotopes as follows:

| Isotope | $F_{18}$ | $Na_{22}$ | $Co_{61}$ | $Cr_{48}$ | $Br_{80}$ | $Br_{80M}$ | $Br_{78}$ | $Sr_{85}$ | $I_{126}$ |
|---|---|---|---|---|---|---|---|---|---|
| Production rate per minute | 26mc | 6.7$\mu$c | 690$\mu$c | 10.3mc | 311mc | 19.5mc | 254mc | 29.0$\mu$c | 1.81mc |

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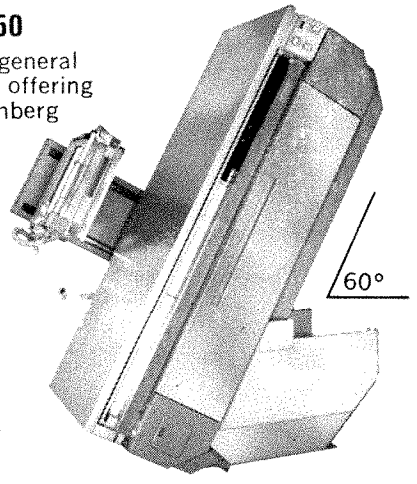
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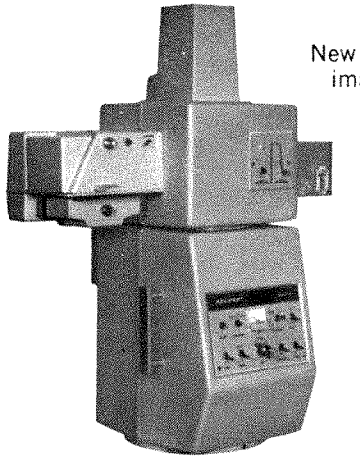
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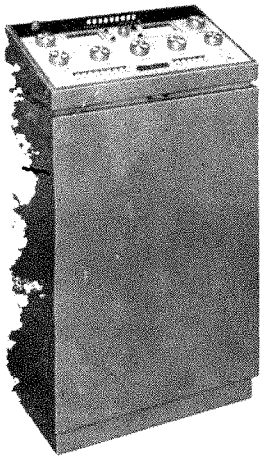
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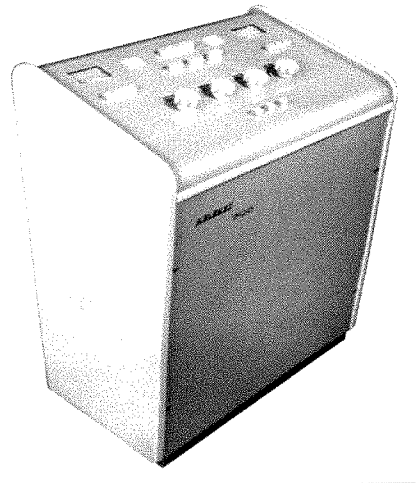

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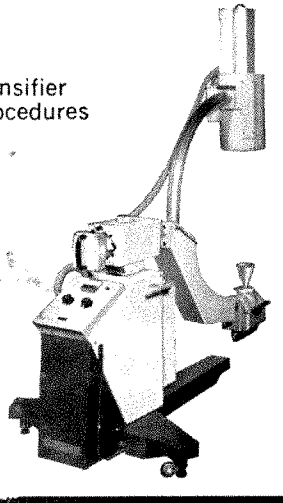


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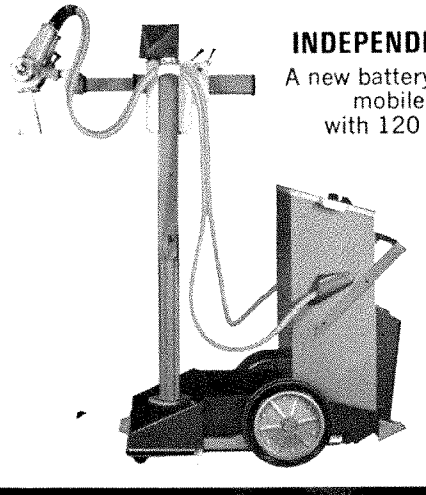
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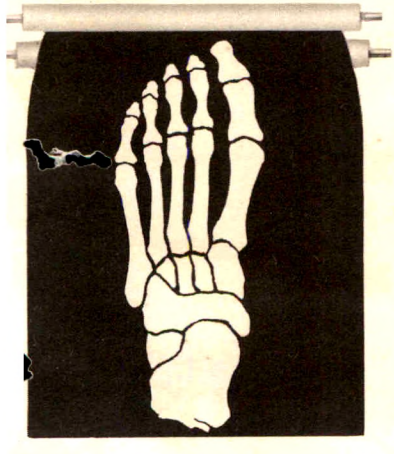
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Edited by William S. MacComb, M.D., Chief, Section of Head and Neck Surgery, and Gilbert H. Fletcher, M.D., Chairman, Department of Radiotherapy, The University of Texas, M.D. Anderson Hospital and Tumor Institute. With 36 Contributing Authors, most of whom are currently members of the Staff of the University of Texas M.D. Anderson Hospital and Tumor Institute.

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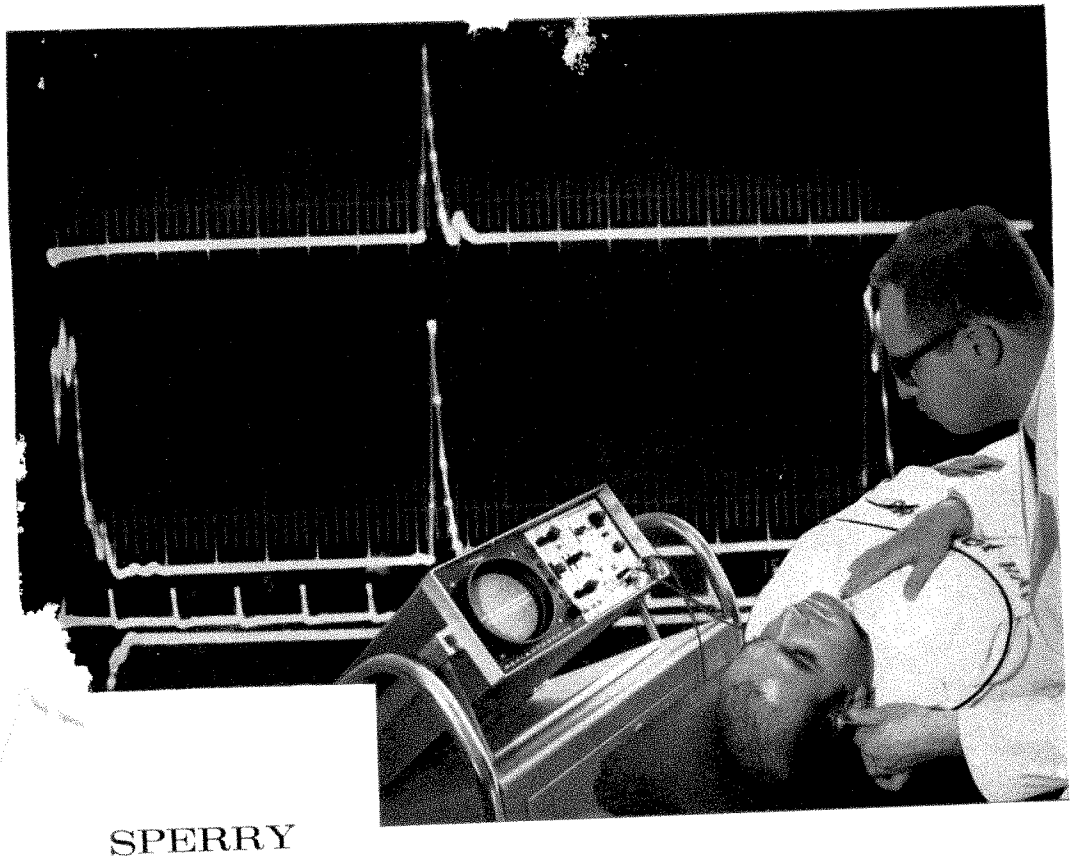
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1. S. Jeppsson, Acta, Chir. Scand 119 (1960) 445-462; Suppl 272(1961)
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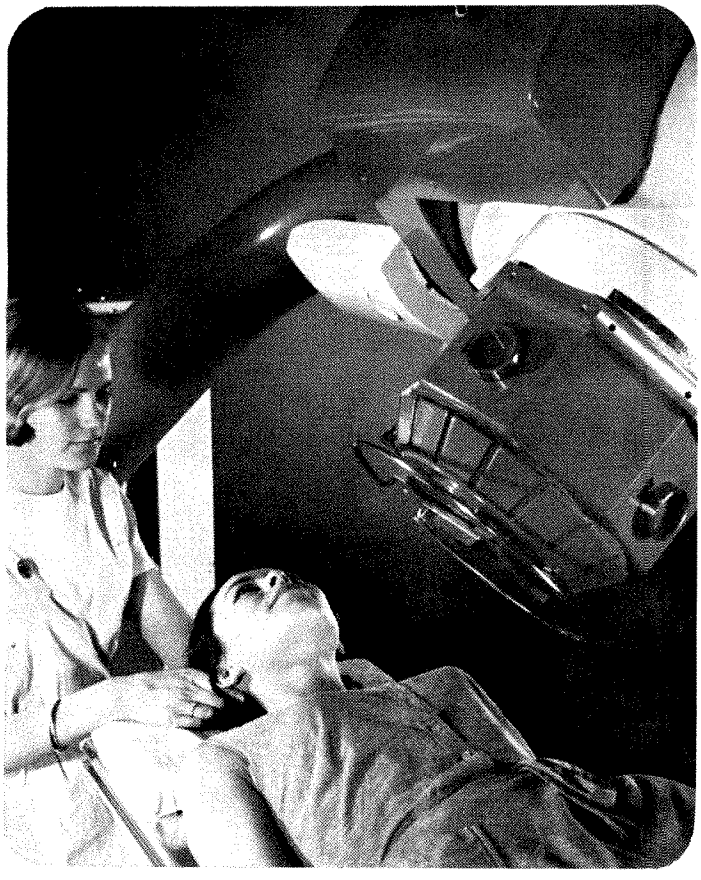
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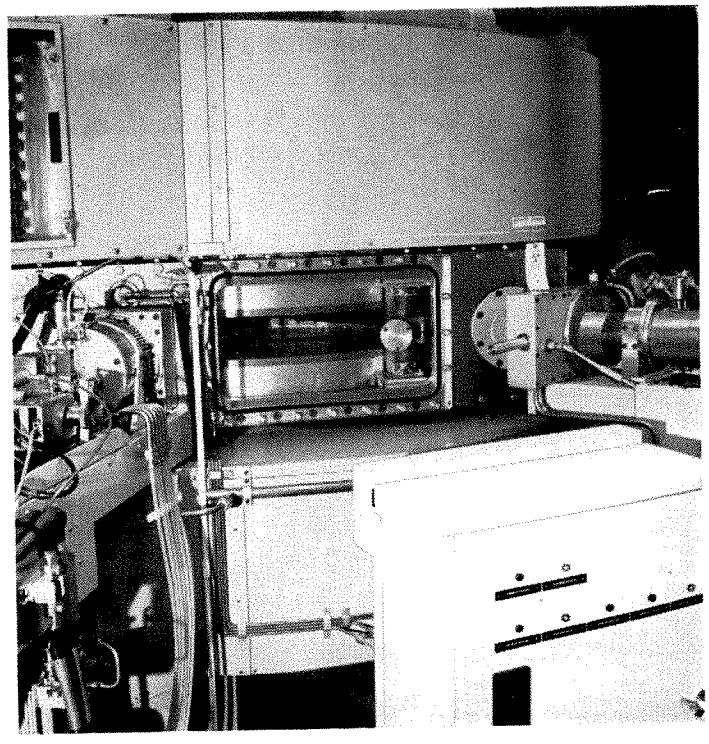
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**ROENTGENOLOGY OF THE SPLEEN AND PANCREAS** *by* **Josef Rösch,** *Charles University, Prague, Czechoslovakia.* Following detailed consideration of diagnosis and anatomy, the author describes various methods of roentgen diagnosis—*from the simplest and most commonly used to the most highly specialized.* While classical routine roentgen examination methods are described, major emphasis is on modern angiographic techniques. **The contribution of each examination to the diagnosis is stressed.** Various diseases of the spleen and pancreas are comprehensively analyzed with basic anatomical and clinical data. *Oct. '67, 376 pp. (6¾ × 9¾), 434 il., $29.75*

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**MEDICAL RADIOGRAPHIC TECHNIC (3rd Ed., 2nd Ptg.).** *Rewitten by* **William L. Bloom, Jr., John L. Hollenbach,** *and* **James A. Morgan,** *all of the Technical Service X-Ray Dept., General Electric Company, Milwaukee. Originally under the editorial supervision of* **Glenn W. Files** *(1897-1945).* Jan. '67, 368 pp. (7 × 10), 510 il., 7 tables, $11.00

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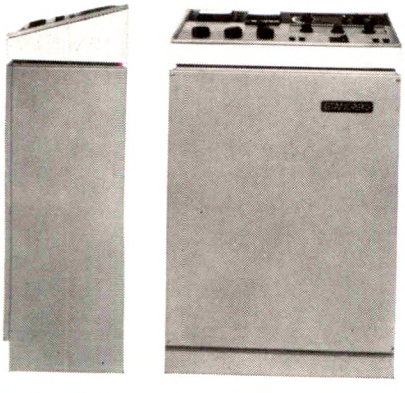
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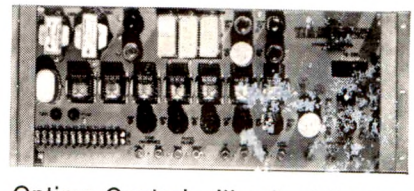


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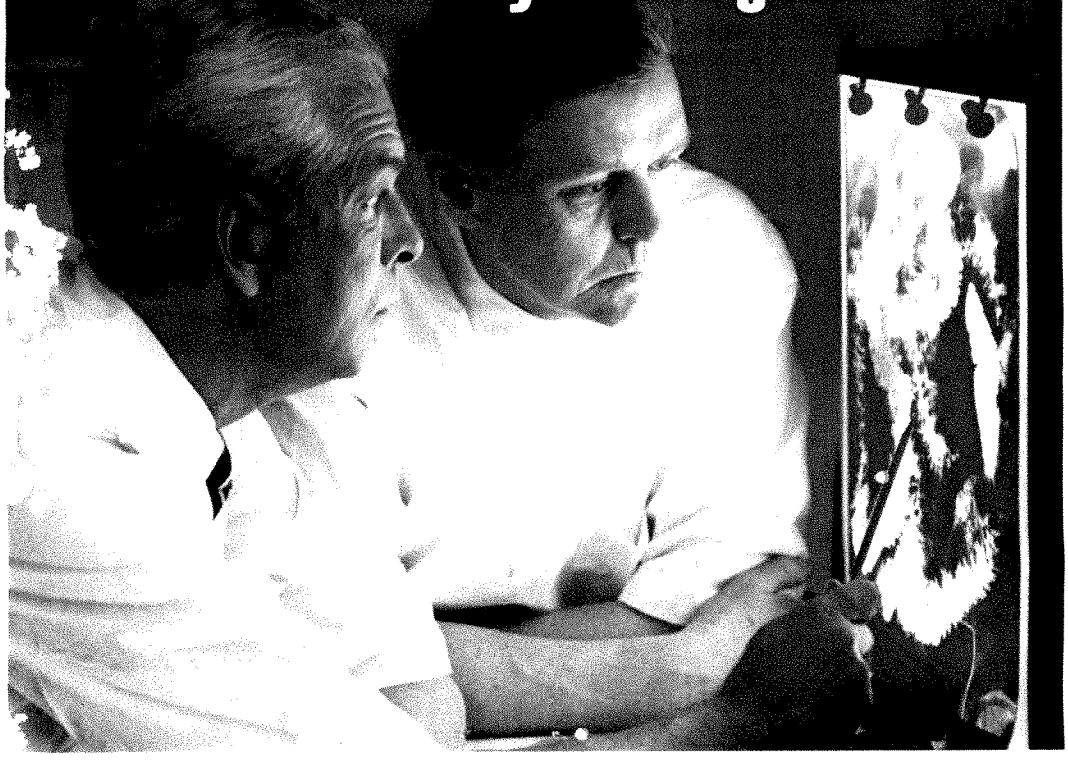
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In G.I.'s, small bowel or air-contrast colon studies, micro-[illegible]n BAROLOID aids the radiologist in diagnosis because it spreads evenly, clings to the mucosa, does not break up, crack, peel or flocculate. Provides sharp pictures of slight dilation of bowel loops or abnormal quantities of fluids with standard or high K. V. technics. Rigidly uniform, each batch exactly matches preceding batches. May be prepared as much as a day in advance.

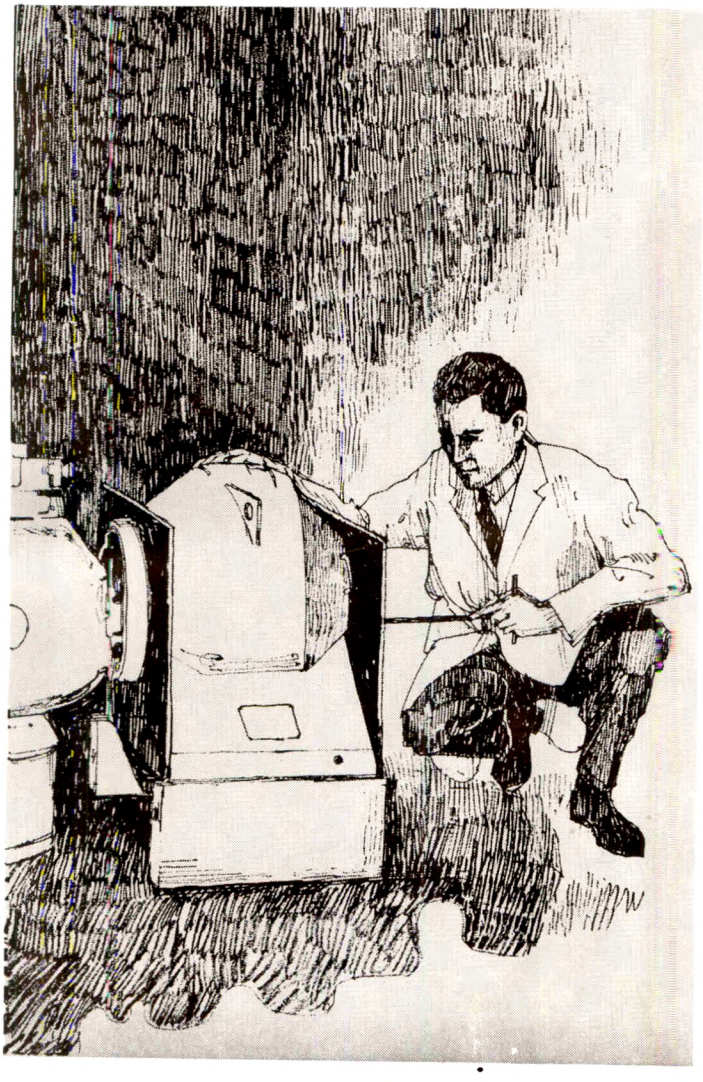
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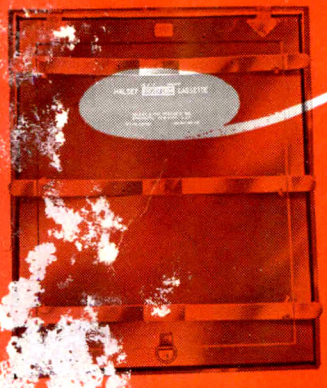
We guarantee source outputs (calibration traceable to the National Bureau of Standards), doubly encapsulated in stainless steel, heliarc-welded capsules. In addition we make a normal check-out of your teletherapy equipment.

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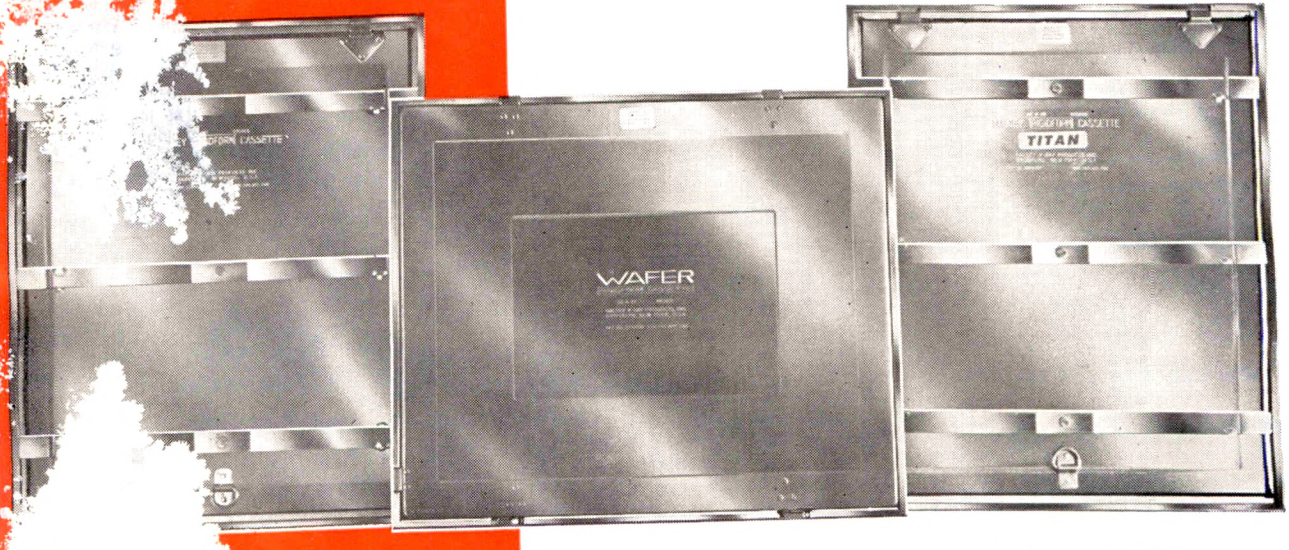
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## now comes premeasured prepackaged for single-use by either route







**MICROPAQUETTES** are now avail[illegible] 2 oz. or 4 oz. MICROPAQUE content. Si[illegible]em a packet into your mixer, add indic mix, and there's your correct dose premeasured by *weight*, not *volum[illegible]*, so to whip up the exact quantity you need curate control of density, making [illegible] thin as you want. Convenient, time a[illegible]




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# MICROPAQUE

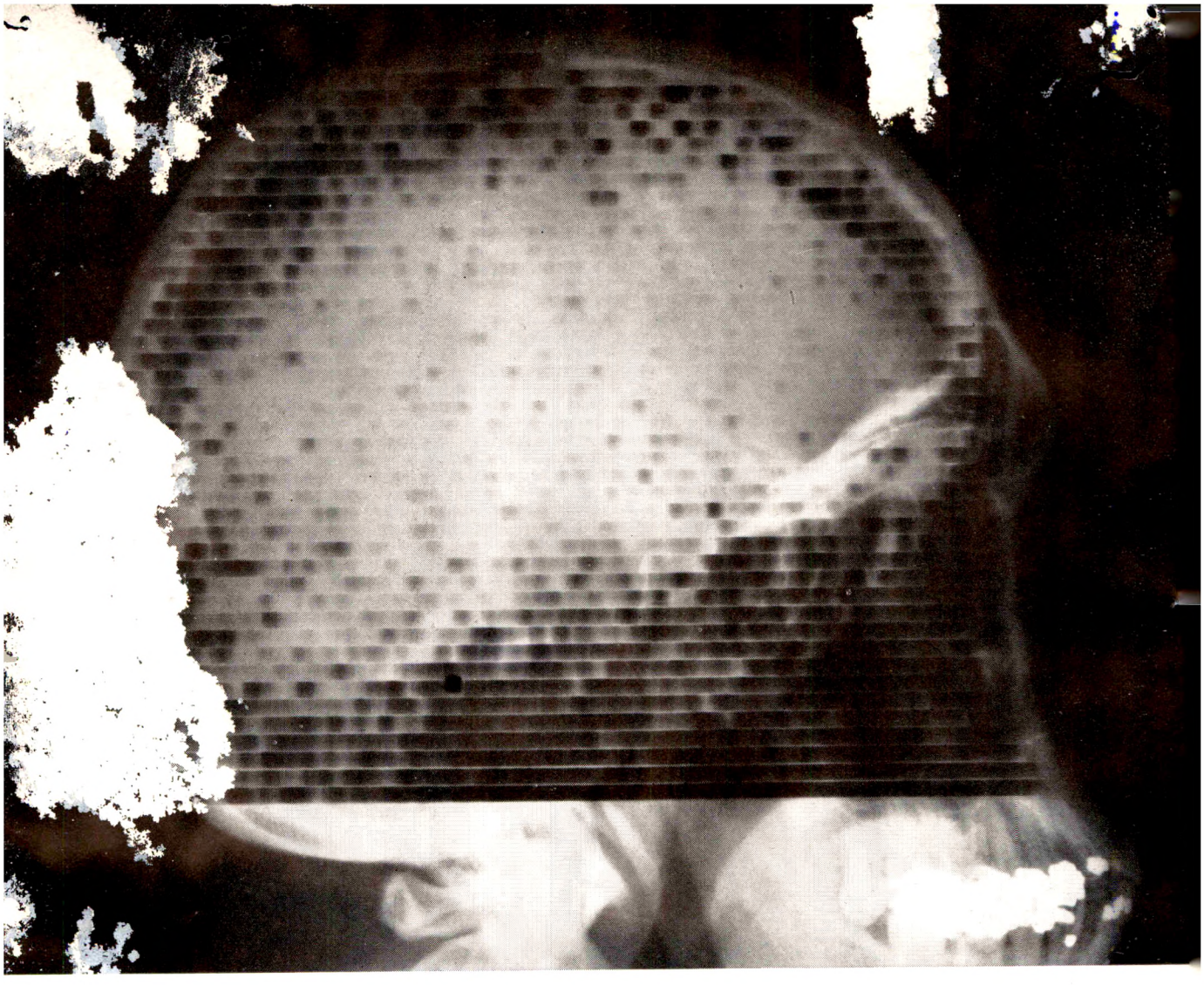
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## single-use economy

**either way,** there's no waste: you mix only as much as you need, one packet or bagful per patient. No unused surplus to squander down the drain. No possibility of contamination with dust or dirt.

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# announces

# Pertscan™-99m

## PERTECHNETATE Tc 99m

**For brain scanning, Pertscan-99m provides more information with less radiation to the patient than any other related cerebral test—whether other radioisotopes or x-rays.**

SPEED: Gives each projection fast—15 minutes or less with rectilinear scanners, 2 to 4 minutes with a camera.

CONVENIENCE: Supplied in a ready-to-use single dose vial.

SAFETY: Carrier-free, non-pyrogenic, sterile, and isotonic.

FLEXIBILITY: Oral or intravenous administration in two sizes: 10 millicuries in 4 ml. and 15 millicuries in 6 ml.

SHIPMENTS: Monday through Friday—and Sunday . . . allows scheduling of brain scans 6 days a week—Monday through Saturday.

INDICATIONS: Adjunctive diagnostic aid in detecting and localizing intracranial neoplastic (primary or metastatic) and non-neoplastic lesions.

CONTRAINDICATION: Radio-pharmaceutical agents should not be administered to pregnant women or to persons less than 18 years old unless the indications are very exceptional.

PRECAUTIONS: Care should be taken to ensure minimum radiation exposure to the patient as well as [illegible] personnel; to prevent extracranial contamination because this can lead to erroneous interpretation; and to differentiate areas of abnormal activity from areas of normal vascular activity. 712391

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